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The endogenous money hypothesis: empirical evidence from Türkiye (2008-2020)

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Abstract:
This paper examines the validity of the endogenous money supply hypothesis in Türkiye from 2008 to 2020. The endogenous money hypothesis underlines the fact that a demand for bank credit leads to the creation of credit and deposit. Deposits are created once credit application is approved by banks. Therefore, the money supply is endogenously determined by bank loans. However, there exist horizontalist, structuralist, and circuitist views, each proposing different causalities between monetary aggregates and the relationship between money and income. In this article, we put forth ten hypotheses to test the validity of the endogenous money hypothesis and three main perspectives over the period 2008-2020 in Türkiye. We aim to discern which of the three main views aligns best with the sample. Our findings provide new evidence on the validity of the endogenous money hypothesis in Türkiye from 2008 to 2020. Besides, the circuit theory of money fits precisely in the short run but partially in the long run. The findings also support the structuralist view partially according to the long-run results.

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Endogenous money hypothesis (EMH) is one of the building blocks of post-Keynesian economics. Early studies conducted by Kaldor (1970), Kaldor & Trevithick (1970), Moore (1979, 1983) emerged as a reaction to monetarism and its proponents, who heavily rely on the money multiplier theory. Thanks to the subsequent contributions of Basil Moore (1988a, 1988b), the EMH evolved into a comprehensive monetary theory. Especially with post 2000 studies, the empirical and theoretical literature on the EMH has significantly expanded. There are three main approaches on the EMH: Accommodationism or horizontalism, structuralism and circuitism. Although endogenous money creation is a common view, these approaches differ from each other on the issue of causality among monetary aggregates.

This paper contributes to the literature in two ways. Firstly, we aim to test the validity of EMH and determine which of the three main views on EMH best fits the Turkish dataset between 2008 and 2020. In doing so, we provide empirical evidence of monetary endogeneity in Türkiye.

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Secondly, the relevance of establishing the validity of EMH for Türkiye becomes evident due to the prevailing perspective on fiscal discipline among Turkish scholars. Many of them adhere to exogenous money theories and do not notice the implications of EMH. Consequently, their policy recommendations on fiscal policy and the role of the government are derived from economic models that depend on exogenous money theories. The prevalent neoclassical argument used by these scholars in suggesting fiscal policy for Türkiye revolves around the crowding out effect. However, if money is an endogenous phenomenon, the crowing out effect becomes untenable. For this reason, this paper contributes to the critique of the fiscal consolidation literature in Türkiye.

The following section comprises theoretical discussion in the EMH literature. Section 2 delves into the implications of the EMH, establishing a connection between the EMH and fiscal policy. Section 3 provides the empirical literature on the EMH. In section 4, we clarify the theoretical causalities utilized in this paper. We suggest ten testable hypotheses to assess the validity of the EMH and its three associated views. Section 5 introduces the data and methodology, while section 6 presents the empirical findings. Theoretical implications of empirical findings are discussed in section 7. Finally, we conclude our paper in section 8.

1. The theory of endogenous money

The endogenous money hypothesis (EMH) constitutes a foundational building block in post-Keynesian economics. It emphasizes the endogenous determination of money supply driven by credit demand, positing that bank loans and deposits coexist concurrently with the extension of credit by banks. This implies simultaneous relationship between bank loans and deposits. The money supply is contingent upon the level of credit. This stands in sharp contrast to the conventional mainstream theory, wherein banks are regarded as passive entities in the process of monetary creation, and the money supply is exogenously determined and controlled by the Central Bank. Within the extensive literature on the EMH, there are three different perspectives on the causality among monetary aggregates: Horizontalism, structuralism, and circuitism.

According to horizontalism, firms require business credit to roll over working capital such as wage payments and purchases of raw materials (Moore, 1983a, pp. 373). For this reason, money is demand-determined and is created when credit is granted by a bank. Horizontalism contends that banks are not constrained by the reserves when serving credit. Thus, the conventional money multiplier model—wherein causality emanates from monetary reserves to bank loans—is not valid (Moore, 1988a). Moore states that the ontological function of the Central Bank is to be the lender of last resort. Hence, the Central Bank must meet systemic liquidity needs; otherwise, the banking system is jeopardized. To avert the risk of a liquidity crisis, the Central Bank finances the banking system through its non-borrowed reserves. This is termed accommodationism in the literature of EMH. In a regime under free capital flows and flexible exchange rates, a bank's liquidity need can be met by alternative means, such as obtaining liquidity from another bank having excess reserves or applying for foreign suppliers at a level of interest rate (Moore, 1988a, p. 383). Rather than resorting to the Central Bank, banks may opt for these alternative channels owing to the potential for reduced costs. Nevertheless, the Central Bank must provide liquidity and accommodate monetary expansion while the banking system applies for liquidity. According to Moore (1988a, pp. 384), endogenous money theory is sometimes interpreted as the Central Bank being fully passive and having no ability to affect the money supply, but this is a misconception. Moore contends that the Central Bank can indeed influence the growth of the money supply through open market operation. Consequently, EMH means that money supply is

credit demand determined, with the short-term interest rate being exogenously set by the Central Bank (Moore, 1988a, pp. 384).

In this framework, banks assume a primary role as credit lenders, operating as quantity takers and price makers within the credit market. Contrary to conventional mainstream theory, the adjustment of interest rate is deemed unnecessary to address reserve requirements when banks seek liquidity. That is, there is no need to increase interest rates while banks look for reserves. Therefore, the money supply curve is posited to be horizontal rather than exhibiting an upward-sloping trajectory (pp. 381-382).

Structuralists diverge from the horizontalist perspective by rejecting the notion of a horizontal money supply curve. Although they accept that a firm's financial needs prompt credit demand, leading to an increase in the money supply, and align with horizontalists on the idea that deposits are created with credit, they dispute the horizontalist framework for monetary creation (Fontana, 2003). In contrast to horizontalists, structuralists strictly oppose the concept of a fully accommodative Central Bank. According to Palley (1996, pp. 592-593), Structuralists argue that the Central Bank has the capability to raise interest rates even as banks seek monetary reserves. Thus, the Central Bank's role extends beyond determining the price of money (via setting short-term interest rates) to also encompass influencing the quantity of money through quantity-based policy.

Structuralists envision the role of the Central Bank as partially accommodative, asserting that the credit supply curve should be upward sloping (Pollin, 1991, p. 398). This implies that Structuralists accept the influence of the money multiplier in the money creation process. Additionally, it is contended that horizontalists may not adequately incorporate the liquidity preference theory, a viewpoint that structuralists believe should play a crucial role in the understanding of the EMH (Fontana, 2003, p. 297). Therefore, the stance adopted by the Central Bank is central to the Structuralist interpretation of the EMH.

The discourse between horizontalists and structuralists revolves around the stance adopted by the Central Bank. Horizontalists contend that the Central Bank is fully accommodative, exclusively setting short-term interest rates. In contrast, structuralists argue that, while determining short-term interest rates, the central bank may also engage in a quantity constraint policy. This fundamental difference results in horizontalists positing a horizontal credit money supply curve, while structuralists propose an upward sloping one.

Pollin's empirical study supports horizontalism by asserting that the Central Bank does not pursue a quantity-constrained policy but rather exogenously sets short-term interest rates. However, market interest rates are not entirely determined by the Central Bank but by the complex interaction between the Central Bank and the financial markets (Pollin, 1991, p. 393). Additionally, the long-term interest rate is argued not to be within the direct control of the Central Bank but is shaped by market dynamics contingent upon future expectations (Moore, 1991, pp. 411-412). Palley (1994) contributes to this discourse by providing a comprehensive discussion of the differences between horizontalists and structuralists, summarizing their respective theoretical proposition. According to Palley, the empirical relevance of setting short-term interest rates outweighs that of a quantity-constrained policy (pp. 593-594).

Circuitists criticize the ongoing debate between horizontalists and structuralists, asserting that the essence of the disagreement lies in differing perspectives on the emerging point of money. Unlike horizontalists and structuralists, circuitists contend that money is an endogenous phenomenon not solely determined by the Central Bank's stance, but rather emerges in response to the demand from firms. They consider that endogeneity of money persists irrespective of the Central Bank's position (Lavoie, 1984, p. 778). Besides, circuitists argue that the degree of

accommodation, financial innovation, and liability management are not primary issues in understanding causalities within the monetary circulation. The monetary circulation begins with credits granted by a bank and concludes with the repayment of debt (Lavoie, 1996b, p. 553). As such, circuitists assert that the ongoing debate between horizontalists and structuralists does not contribute significantly to understanding the endogeneity of money within the monetary system.

Circuitists emphasize the significance of monetary circulation, viewing money as a necessary part of a circular flow within the system, encompassing its creation to eventual destruction. Graziani (2003, pp. 25-26) elucidates the process of monetary circulation, emphasizing that credit, particularly in the form of bank-supplied money, is the main form of money in modern monetary economies. Firms, in need of financing for working capital such as wage payments, raw materials, and capital goods expenditures (Eichner, 1979; Lavoie, 1984; Seccareccia, 1996), create credit demand, surpassing their planned expenditures driven by sales expectations. The initiation of monetary circulation occurs when banks grant credit, constituting a debt for firms. As this credit is repaid, money is destroyed, concluding the monetary circulation. This is termed a circuit.

According to circuitists, two primary household types exist in a society: capitalists and workers. Capitalists possess the privilege of accessing money. This easy access to bank money is not owned by workers. For this reason, money is not neutral due to the disparity of purchasing power across social classes. Workers tend to spend what they earn, and any attempt to save results in the closure of monetary circulation with a loss. Circuitists posit that money is endogenously created by banks through a negotiation process with firms. This negotiation, however, is not necessarily favorable to firms, as banks consider various factors such as the firm's profitability, future cash flow, prior relationship with banks, economic situation, sectoral circumstances, and future expectations. The creditworthiness of firms is a key favorable consideration for banks, and they may ease or tighten credit conditions accordingly (Rochon, 1999a, pp. 10-11). Therefore, the markup charged by banks varies due to these conditions (Palley, 2008, p. 7). Circuitists are close cousins of horizontalists, asserting that banks, within the circuit theory of money, do not face a reserve constraint while lending, as they lend money to firms and then seek reserves (Lavoie, 1996a). While circuitists maintain that banks operate without reserve constraints, certain monetary constraints are recognized within the system. Consequently, the Central Bank monitors the monetary system and its dynamics to preserve financial stability and prevent liquidity crises by facilitating sustainable conditions for bank transactions (Graziani, 2003). This role is fundamentally derived from the Central Bank's position as the lender of last resort. Hence, circuitists define a defensive role for the Central Bank (Eichner, 1987; Wray, 1998, p. 115; Rochon and Rossi, 2007). Besides, the interaction among economic and social agents is deemed crucial within circuitism.[1]

While financialization and financial innovations were initially considered less significant in the circuit theory of money (Lavoie, 1996b, p. 553), a growing literature is now exploring the nexus between financialization and endogenous money. In the traditional circuit theory of money, the endogeneity of money creation is addressed to firms' credit demand for continuing production. However, contemporary research suggests that financialization, particularly through households' demand for financial instruments, could constitute an additional source of endogeneity in money creation. Consequently, the interaction between the real and financial

[1] The circuit theory of money extends beyond a mere monetary theory; it encompasses a broader perspective as a theory of accumulation and distribution. Circuitists within this framework, not only scrutinize monetary circulation but also delve into the dynamics of wealth and debt formation, emphasizing the intricate interaction among economic and social agents, For the details, Graziani's *Monetary Theory of Production* serves as a comprehensive resource offering theoretical insights into this multidimensional approach.

sectors via the endogenous creation of money is gaining attention (Bhaduri, 2011, p. 15). Passarella (2014) introduces a monetary circuit model that emphasizes the interaction between household debt and increasing financial profit under the endogenous money creation of the banking sector. In this model, the initiation of the monetary circuit is triggered by households rather than the corporate sector. Thus, the expansion of household debt contributes to capital market inflation (p. 131). However, it is acknowledged that these new circuits, fueled by household credit, could potentially pose significant risks to the economy (Botta et al., 2015, p. 222). The rapid growth of household credit and debt in the era of financialization carries implications for financial cycles and instability (Sawyer, 2020, p. 361).

2. Why does endogenous money matter?

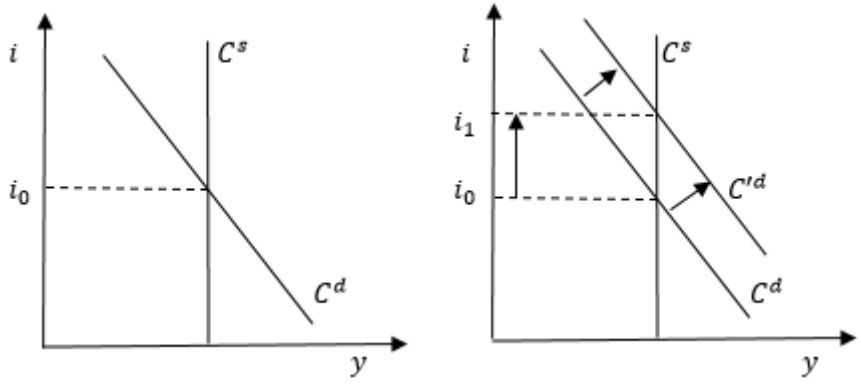
A significant implication of the endogenous money hypothesis concerns the role of government in an economy. Following the works of Fontana (2003) and Palley (2002, 2013), we assume a monetary economy where credit is an essential form of money. Banks function as suppliers of credit, catering to the credit demand of firms and households. Within the monetarist perspective, which adopts exogenous money, the credit market can be described by following equations:

$$C^d = f\left(\overset{-}{i}, \overset{+}{y}\right) \qquad \text{[credit demand]} \qquad (1)$$

$$C^s = \alpha y \quad \alpha > 0 \qquad \text{[credit supply]} \qquad (2)$$

where C^d represents credit demand, i is the interest rate, y denotes income, C^s stands for credit supply, α represents a coefficient, providing money form of income when multiplying with income. The signs above the terms in the parentheses indicate a sign of partial derivatives. Figure 1 illustrates a monetarist credit market.

Figure 1 – *Monetarist type of credit market*



Credit demand is a function of income (y) and the rate of interest (i). The central bank adheres to a strict quantity constraint policy, whereby the credit served by banks is fixed, necessitating a vertical credit supply. Any increase in credit demand meets no substitutable credit supply. For this reason, the level of economic activity is restricted by the money supply. Consequently, any shift in credit demand directly results in a sharp increase in interest rates as delineated in the graph on the right above.

When we replace credit demand equation (1) with the Hicksian representation (Hicks, 1937, pp. 153) of Keynes's general theory, the credit market can be analyzed using the following equations:

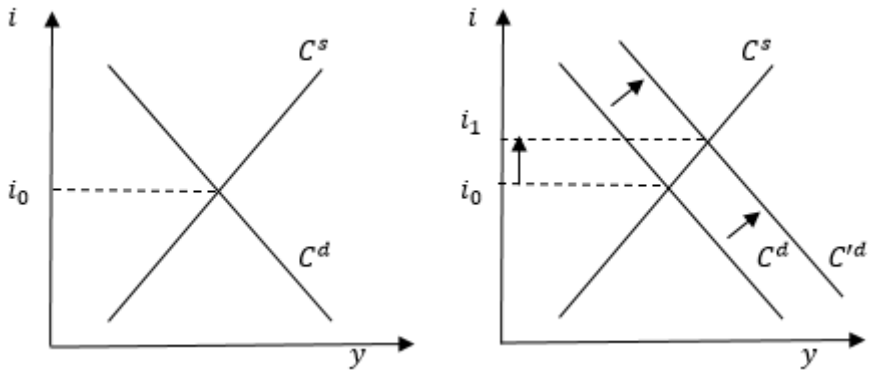
$$C^d = D\left(\overset{-}{\overbrace{\mathrm{i}}}, \overset{+}{\overbrace{\mathrm{Co}^d}}, \overset{+}{\overbrace{\mathrm{I}^d}}, \overset{+}{\overbrace{\mathrm{G}^d}}, \epsilon\right) \qquad \text{[credit demand]} \qquad (3)$$

$$C^s = f\left(\overset{-}{\overbrace{\mathrm{i}}}, \overset{+}{\overbrace{\mathrm{y}}}\right) \qquad \text{[credit supply]} \qquad (4)$$

where D is aggregate demand, Co^d represents consumption demand, $\mathrm{I}^d$is the investment demand, G^d indicates government investment demand and ϵ denotes all remaining factors that influence credit demand.

Now, credit demand and supply are interlinked through the level of economic activity and the rate of interest. This representation known as neo-Keynesian type of credit market is illustrated in figure 2. Essentially, the distinction between figures 1 and 2 is related to monetary creation. In this context, the money supply is not entirely constrained by the central bank.

Figure 2 – *Neo-Keynesian or Structuralist type of credit market*



As illustrated in the graph on the right, any increase in credit demand resulting from consumption or investment motives can be met with a substitutable credit supply through an increase in the rate of interest. For this reason, the rate of interest is sensitive to credit demand, as demonstrated by a positively sloped credit demand curve. This neo-Keynesian type of credit market aligns with the credit market envisioned by structuralist view of endogenous money

theory because banks raise the rate of interest while credit demand increases.[2] Additionally, government investment directly leads to crowd out the private sector. Therefore, the government's attempt to increase expenditure is not effective and depletes the funds in the credit market, resulting in an increase in the rate of interest. Consequently, the loanable funds hypothesis underlies the structuralist type of credit market.

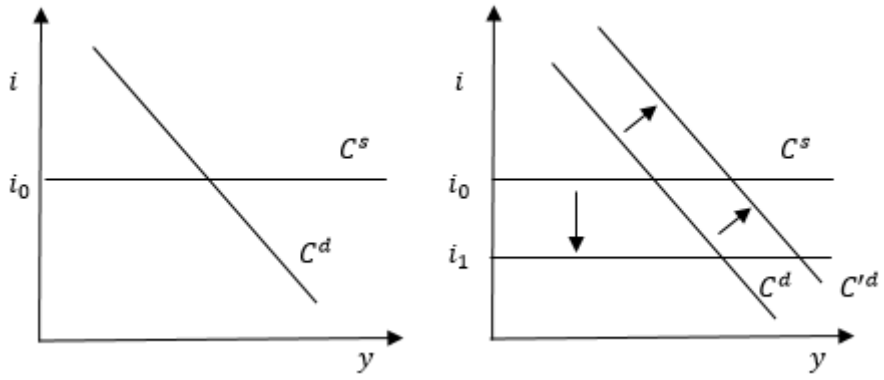
If money is endogenously created by banks and the role of the central bank is to maintain a healthier financial environment (Graziani, 2003), there is no necessity to increase the rate of interest in response to rising credit demand. Credit supply is now horizontal and is determined by a markup above the rate of interest charged by the Central Bank. The following equations can be suggested to represent the horizontalist type of the credit market:

$$C^d = f\left(\overset{-}{\hat{\imath}}, \overset{+}{\widehat{Co^d}}, \overset{+}{\hat{I}^d}, \overset{+}{\widehat{G^d}}, \epsilon\right) \qquad \text{[Credit demand]} \qquad (3)$$

$$C^s = \overline{C^s} \qquad \text{[Credit supply]} \qquad (5)$$

The accommodationist or horizontalist representation[3] of credit market consists of negatively sloped credit demand curve and horizontal credit supply curve, as depicted in figure 3.

Figure 3 – *Horizontalist type of credit market*



Horizontalist representation differs from the two previous illustrations because credit supply is horizontal. This exposes one of the most significant implications of the endogenous money theory, cutting off the link between credit demand and the rate of interest. Therefore, the government decision to invest becomes particularly meaningful and does not lead to an increase in interest rate or crowd out the private sector. This is obviously seen in the second graph above. Any shifts in credit demand stemming from the government decision to invest is accommodated

[2] There is no distinction between a fully exogenous money supply depicted in the figure 1 and money supply with an upward-sloping curve illustrated in figure 2. In the context of monetary creation, both representations offer nothing new to distinguish from neoclassical economics (Lavoie, 1996a, pp. 276)

[3] The horizontalist representation of credit market has consistently been linked to creditworthiness and credit restraints (Lavoie, 2022). Therefore, the horizontalist type of credit market depicted in figure 3 aligns with circuitism, as explained in the previous section.

by the financial system. Additionally, the central bank can influence and expand the money supply through the open market operation or an aggressive cut in the policy rate (Moore, 1998) as demonstrated in the second graph in figure 3. Consequently, this representation completely eliminates any trace of the loanable funds hypothesis.

The impact of government investment on the interest rate under the horizontal money supply is crucial because fiscal policy literature heavily relies on exogenous money supply and loanable funds theory. Notably, Turkish scholars such as Özatay (2008, p. 6; 2012, pp. 9-10), Kaya and Yilar (2011, pp. 61-62) and Akcay et al. (2018, p. 79) criticize the fiscal deficit of the Turkish economy and advocate for fiscal discipline based on economic models grounded in the loanable funds hypothesis. Despite a growing empirical literature on the validity of EMH for Turkish data, these scholars emphasize the crowding-out effect of government investment while ignoring the endogenous money theory and its implications. Consequently, their policy recommendations are derived from economic models that depend on exogenous money theories. However, if money is considered an endogenous phenomenon, the crowding-out effect does not hold. Therefore, this paper also contributes to the critique of fiscal consolidation literature in Türkiye.

3. Empirical studies

Empirical studies within endogenous money literature have experienced significant growth. Even if these studies vary in the sample, researchers have followed the same path in methodology. They employ the causality technique proposed by Granger (1988) and the cointegration test introduced by Johansen (1988). A few of them implement the Toda-Yamomoto (1995) causality techniques as well as the Granger causality and Johansen cointegration tests. For instance, Moore (1988b) conducted the first empirical research on the validity of the EMH by utilizing the Granger causality test. The findings revealed a causal relationship from bank loans to other monetary aggregates. This result supports the validity of monetary endogeneity. Pollin's (1991) empirical study also supports the EMH. Nevertheless, the difference in causality sparked a famous debate known as the 'accommodationist vs. structuralist debate in the determination of money supply'. Howells and Hussein (1998) investigated G7 economies through the application of the Granger causality and vector error correction model (VECM). The outcomes demonstrated that broad money is determined endogenously, but causality direction, specifically from bank loans to bank deposits, remained unclear. A few years later, Caporale and Howells (2001) applied the Toda and Yamamoto causality technique. Unlike the bivariate test carried out by Hussein and Howells in their 1998 paper, Caporale and Howells added total transaction as a third variable. The results still support the validity of the EMH. Notably, the introduction of a third variable and the utilization of the Toda and Yamamoto technique did not the refute earlier conclusions reached by Hussein and Howells. Therefore, the causality, specifically from bank loans to bank deposits, remains unclear.

In the early 2000s, empirical studies of the EMH expanded on a country-specific basis. For instance, Nell (2000) demonstrated that the money supply in South Africa was endogenously determined over the period 1966-1997. The empirical findings indicated a causal relationship where bank loans influenced bank deposits, and there existed a long-run cointegration between money income and the M3 money supply. Shanmugam et al. (2003) reached similar causality results as Nell (2000) but with a different sample. Their study focused on testing the EMH for Malaysian data between 1985-2000, revealing that bank loans lead to bank deposit and establishing a long run cointegration between money income and M3 in Malaysia. Vymyatnina

(2006) evaluated Russian data, concluding that EMH held true for Russia between 1995 and 2004. Additionally, Vymyatnina identified causality running from inflation to the growth of the money supply in this study. Ahmad and Ahmed (2006) showed the existence of monetary endogeneity in the short run for Pakistan data, while emphasizing that the Central Bank significantly influences the growth of the money supply in the long run. Panagopoulos and Spiliotis (2008) conducted a comprehensive examination by testing four orthodox and four heterodox money creation models for G7 economies. They explained the money-income relationship and money multiplier model, finding that results favored the validity of heterodox models. With the exception of France and possibly Japan, monetary creation in G7 economies could be better explained by heterodox theories, particularly the circuit theory of money, which demonstrated the best fit with the available data.

In the last decade, researchers shifted their focus to multi-country samples while continuing to analyze individual countries. For instance, Badarudin et al. (2012) implemented the VECM model to examine short and long run causality, utilizing quarterly data from Australia spanning from 1977 to 2007. Despite shifts in the Australian monetary regime, transitioning from exchange rate targeting (1970-1971) to monetary targeting (1973-1993) and eventually to inflation targeting (1973 to the present), the results consistently upheld the EMH throughout the entire period. Consequently, changes in the monetary regime did not appear to impact the validity of monetary endogeneity. Haghighat (2012) tested the EMH using the VECM model with Iranian data, revealing evidence of money supply endogeneity in the long run. Vera (2001) tested the EMH for Spain covering the period 1987-1998. The Granger test findings perfectly fits monetary endogeneity. Additionally, table 3 of the study shows that bank loans cause all the other variables. Mueller and Wojnilower (2016) evaluated the EMH using quarterly data from the U.S. spanning from 1959 to 2008. Their findings showed that changes in commercial bank loans led to changes in the monetary base and nominal income.

The scope of multicounty analysis expanded beyond the G7 to encompass additional regions such as the Eurozone, GCC and CEMAC. For instance, Lopreite (2012) employed VAR and VECM models for the Eurozone from 1999 to 2010. Despite the introduction of securitization providing supplementary liquidity for the banking sector, Lopreite's adjustment for securitization in the loan series supported the notion of monetary endogeneity in the Eurozone. Tas and Togay (2012) suggested a direct test for the validity of the EMH. In addition to the Granger causality test, they utilized instrumental variable (IV) and two-stage least square techniques for GCC countries: Bahrain, Kuwait, Qatar, Oman, Saudi Arabia and the United Arab Emirates. Across all these techniques, the findings supported the validity of the EMH. Badarudin et al. (2013) tested the EMH using quarterly data from G7 economies over a span of 26 years. The results supported Howells and Hussein's findings in their 1998 paper, with the distinction of employing a more extensive test period for G7 and emphasizing the influence of the controlling monetary regime in the causality model. Nayan et al. (2013) conducted research with the largest sample size, using a panel dataset of 177 countries from 1970 to 2011 to evaluate the money creation process. Instead of traditional causality techniques, they used the generalized method of moments (GMM) methodology, revealing that the money supply is indeed an endogenous phenomenon. Cyrille and Christophe (2022) evaluated the EMH for the CEMAC area: the Central African Republic, Equatorial Guinea, Cameroon, Gabon, and the Republic of Congo. Employing both traditional causality techniques and an alternative test proposed by Tas and Togay (2012), the results consistently supported the validity of in the CEMAC area.

Turkish scholars have shown a growing interest in the EMH, leading to an expanding empirical literature that incorporates Turkish datasets. Çavuşoğlu (2003) evaluated the determination of

money supply using quarterly data spanning from 1985 to 2001. By incorporating the consolidated budget cash balance, the study aimed to understand the impact of the need for public debt finance on money supply growth. The results not only supported the validity of the EMH but also revealed that the central bank could only increase money stock in the long run and was unable to reduce it. An important finding highlighted the endogenous determination of money, influenced not only by credit demand but also by public credit demand. Cifter and Oguz (2007) delved into the monetary transmission mechanism during the period 1997-2006. The results suggested that the monetary transmission mechanism in Türkiye appeared to align with the post-Keynesian policy proposition. Ozgur (2011) tested the EMH for Turkish data, including the quarterly period between 1987 and 2009. The findings supported the validity of EMH, but the relationship between bank loans and some monetary aggregates containing foreign exchange deposits was not statistically significant due to currency substitution. Ozgur addressed this by introducing a variable as a proxy for currency substitution and exchange rate into the model, revealing a statistically significant relationship between bank loans and monetary aggregates after this adjustment. Işık and Kahyaoğlu (2011) elucidated the EMH over the period between 1987 and 2007. They showed causality running from credit to the monetary base. The findings also demonstrated bidirectional causality between gross national product (GNP) and the monetary base. Incekara and Amanov (2017) employed Granger causality and Johansen cointegration tests spanning from 1990 to 2016. The results indicated that the central bank could not control the money supply in both the short and long runs, suggesting limitations in using monetary aggregates for managing monetary policy. Cepni and Guney (2017) investigated money supply theories between 2006 and 2015, finding that causality ran from bank loans to money supply in both the short and long terms. Baştav (2021) found almost similar results, establishing causality from bank loans to money supply using monthly data spanning from 2011 to 2018.

4. Testable hypotheses

Horizontalism, structuralism, and circuitism diverge in their conceptualizations of the causalities within monetary processes, despite a shared a common view that money is endogenously determined. The EMH implies that money is endogenously created by banks. A demand for bank loans causes bank deposits, thereby driving the money supply. Following the empirical literature, we formulate our first two hypotheses as follows:

H_1: *Bank loans cause deposits*.

H_2: *Bank loans cause money supply*.

Three main views on EMH converge on the acceptance of the first two hypotheses. In testing H1 and H2, we aim to determine whether money is endogenous. Subsequently, we proceed to test the hypotheses below in order to understand the validity of each of the three main views.

Horizontalists begin to accept H2 and H1. They also propose that monetary reserves are determined by bank loans (Holmes, 1969, pp. 73; Lavoie, 1984; Moore, 1998, pp.176). They refuse the money multiplier concept, contending that banks do not operate using such a money multiplier mechanism (Moore, 1988b, pp.70). This implies that the central bank accommodates credit expansion, and there is no need to increase interest rates when banks demand reserves (Moore, 1988a, pp. 382). Horizontalism anticipates bivariate causality between GDP and money

supply (Davidson, 1978; Moore, 1989, pp. 483; among other empirical studies). Accordingly, we define three more hypotheses, incorporating these propositions:

H_3: *Bank loans cause monetary reserves*.

H_4: *There is no causal relation between bank loans and the money multiplier*.

H_5: *There is a bivariate causality between GDP and money supply*.

Despite accepting H1 and H2, structuralists advocate for a partially accommodative central bank. Partial accommodation implies that the central bank conducts quantity-based policies (Palley, 1996, pp. 592-593). Structuralists accept the upward-sloping money supply while refusing the horizontal money supply (Fontana, 2003, p. 302). For this reason, this perspective implies bivariate causalities between bank loans, monetary reserves, and the money multiplier. Similar to horizontalists, structuralists propose bivariate causality between GDP and money supply, leading us to define the fifth hypothesis (Nell, 2000, pp. 314; among other empirical studies). Therefore, two more hypotheses are formulated based on structuralist propositions:

H_6: *There is bivariate causality between bank loans and monetary reserves*.

H_7: *There is bivariate causality between bank loans and the money multiplier*.

Circuitists not only embrace H1 and H2 but also accept H3. Despite their focus on countries without required reserve policies, circuitists argue that reserves in banks' possession stem from a prior credit created by banks (Graziani, 2003, pp. 84). This implies the presence of a causal relationship from bank loan to monetary reserves (H3). Circuitists define banks as entities that consider the creditworthiness of firms, emphasizing that credit demand is not automatically met but rather negotiated with banks (Rochon, 1999a, pp. 10-11; Graziani, 2003, pp. 27). Seeking creditworthiness is a kind of asset management conducted by banks. Therefore, the concept of a bank's creditworthiness implies causality running from bank loans to the money multiplier (Panagopoulos and Spiliotis, 2008). Regarding the connection between money and income formations, circuitists propose that bank loans cause GDP (Rochon, 1999b, pp. 63; Graziani, 2003, pp. 29). Subsequently, GDP is posited to cause changes in the money supply (Rochon, 1999b, pp. 63; Graziani, 2003, pp. 30-31). Therefore, these considerations lead to the formulation of three additional hypotheses:

H_8: *Bank loans cause money multiplier*.

H_9: *Bank loans cause GDP*.

H_{10}: *GDP causes money supply*.

To make these hypotheses tractable, we provide table 1, illustrating the association between each hypothesis and its corresponding view.

Table 1 – *Summary of hypotheses*

Hypothesis	EMH	Horizontalism	Structuralism	Circuitism
H_1: Bank loan → Bank deposit	•	•	•	•
H_2: Bank loan → Money supply	•	•	•	•
H_3: Bank loan → Monetary reserves		•		•
H_4: Bank loan ≠ Money multiplier		•		
H_5: GDP ↔ Money supply		•	•	
H_6: Bank loan ↔ Monetary reserve			•	
H_7: Bank loan ↔ Money multiplier			•	
H_8: Bank loan → Money multiplier				•
H_9: Bank loan → GDP				•
H_{10}: GDP → Money supply				•

• denotes that the view on the column requires a dotted hypothesis.

5. Data and methodology

We compile a monthly dataset comprising 156 observations spanning from 2008 to 2020. The dataset encompasses five primary monetary variables: bank loans (BL), bank deposits (DEP), monetary reserves (MRES), money supply (M2), and the money multiplier (MIER). In order to obtain these monetary series, we use the balance sheet of the banking sector served by the Turkish central bank statistics. Bank loans and bank deposits are derived from the liability and asset sides of banking sector's balance sheet. Monetary reserves are mandatory reserves held by banks in their accounts with the central bank. Banks must maintain these reserves for the new credits for a period of up to two weeks following the date of the liability calculation. Money supply (M2) contains banknotes and coins (currencies in circulation) and cash in the banking sector, along with bank deposit and time deposits. The money multiplier is calculated as the ratio of money supply (M2) to monetary reserves (MRES). We provide line graphs of each series in appendix B. According to the depicted graphs, it is evident that BL, DEP, and M2 exhibit similar patterns, suggesting a positive correlation among them. Conversely, MRES and MIER appear to be negatively correlated due to the calculation method of MIER (M2/MRES).

To explore the connection between money and income formation, we use the industrial production index (IPI)[4] as a proxy for GDP. The industrial production index is chosen due to its monthly reporting frequency, aligning with our monetary series, and is obtained from the Turkish Statistics Institute.

The abbreviations of the variables are given in table 2.

All variables are expressed in natural logarithms. With the exception of IPI, the other variables were not subjected to seasonal adjustment in the source from which we obtained the data. Therefore, seasonality was checked for all other variables using the STL Decomposition methodology (Seasonal and trend decomposition using LOESS), and no seasonality was identified.

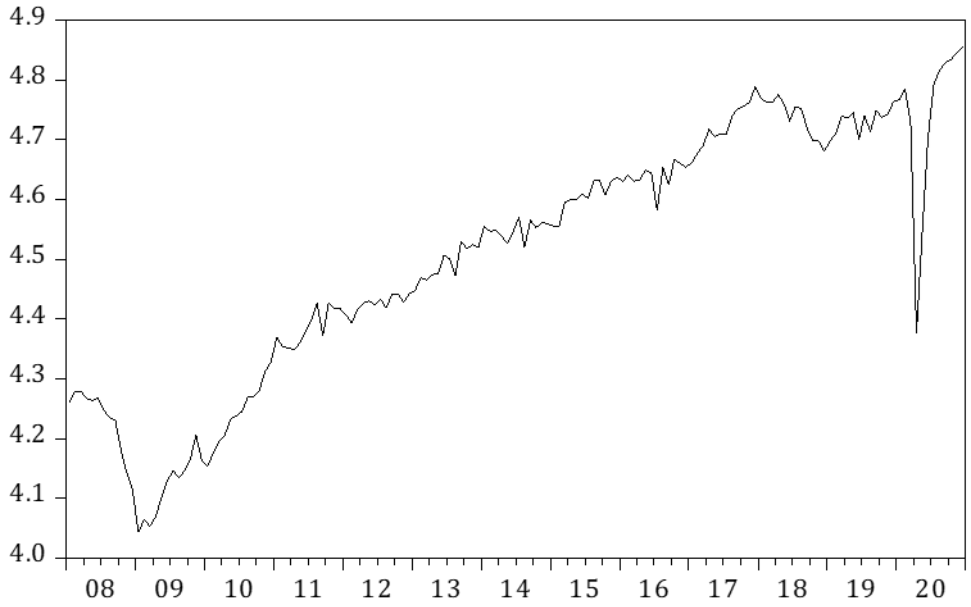
The analyses include IPI-required control for the structural breaks. Figure 4 represents this dataset.

[4] We use monthly industrial production series adjusted for seasonal and calendar effects.

Table 2 –*Variables and their abbreviations*

Bank loans	BL
Monetary reserves	$MRES$
Money supply	$M2$
Money multiplier	$MIER$
Bank deposits	DEP
Industrial production index	IPI

Figure 4 – *Industrial Production Index*



First, we tried to use dummy variables in order to control for the 2008 crisis (2008M08-2011M01) and the Covid-19 crisis (2020M02-2020M07). However, a technical problem regarding diagnostic tests (serial correlation and heteroskedasticity) emerged. Therefore, we went with restricted data (data that does not include either crisis, 2011-2019) when the industrial production index was used as one of the variables. However, apart from the diagnostic test results, the interpretation of estimation did not change.

As for empirical technique, we constructed vector auto regression models (VAR) with Granger causality tests for analyzing non-cointegrated series. We also built restricted VAR models called vector error correction models (VECM) to understand the causality relation between cointegrated series. VAR models treat each variable in the model endogenously and use their lag to correct possible endogeneity problems. Since all variables are endogenous, there is a correlation between the current values of the variables and the error term. Therefore, the VAR Model uses the lag(s) of the variables on the right-hand side in order to eliminate possible endogeneity problems. Correlation between an explanatory variable and an error term can also arise when there is an omitted variable problem because the omitted variable is a part of the error term and it is correlates with the explanatory variable. By adding lagged value(s), the VAR model solves this problem because lagged value(s) include other unobserved factors. A lagged regressor produces

collinearity; this is not a problem because there is no interest in inference on individual parameters in VAR models.

This feature of VAR models is very consistent with Granger causality methodology since the existence of Granger causality is determined by investigating the significant joint effect of all the lags of the independent variable(s) on the dependent variable (see appendix A for detailed information about VAR methodology.). Therefore, in the literature, they are generally used together.

In the first step, the integration order of the variables was tested by using ADF (augmented Dickey-Fuller) and PP (Phillips-Perron) unit root tests. According to empirical results, it was found that all the variables are integrated of order one. Optimal lags were selected according to what the majority of information criteria indicate (sequential modified LR test statistic, final prediction error, Akaike information criterion, Schwarz information criterion, and Hannan-Quinn information criterion). In order to test the long-run relationship between variables, the Johansen cointegration test was used. According to the result of the cointegration test, the VAR or VECM model was constructed. Lastly, diagnostic tests such as autocorrelation and heteroskedasticity were checked. The models have no serial correlation or a heteroskedasticity problem. All analyses were performed separately for the binary variable groups by changing the dependent variable to analyze the effect of each variable on the other. We have six different specifications that analyze the causal relations between variables. These analyze the causal relations between BL and five other variables (DEP, M2, MRES, MIER, IPI), respectively. Additionally, the causal relation between IPI and M2 is analyzed for all the hypotheses given in the testable hypothesis part. In order to comment on the signs of the effects, we utilized impulse-response functions (IRF). They were given according to the direction of causal relationships since we use Cholesky decomposition; and when this methodology is selected, there is a need to order the variables. In Cholesky's methodology, there is a lower triangle matrix and an upper triangle matrix. In the lower one, there is a need to order with an exogeneity criterion. In other words, the most exogenous variable comes first, then the second, until the variable on which all other variables have an effect. The concern about ordering in VAR is due to IRF specifications. We order variables according to the causal relationships. In other words, if there is a bi-directional causal relation, we order from the stronger to the weaker one, according to the significance level. Therefore, IRFs are also given according to causal relationships. To illustrate, if there is a uni-directional causality between X and Y, which runs from X to Y, only the accumulated response of Y to X is given. We use accumulated responses since differences are used in the models.

The reason for applying Cholesky decomposition is that we are investigating causal relationships. Cholesky decomposition imposes a recursive causal structure from the top variables to the bottom variables, but not the other way around (Lin, 2006, pp. 1).

Furthermore, we must explain why we prefer using the VAR methodology to local projection. Plagborg-Moller and Wolf (2021) prove that local projections and vector autoregressions estimate the same impulse responses. Brugnolini (2018) also compares the performance of the vector autoregressive model impulse response estimator with the local projection methodology and, using a Monte Carlo experiment, he finds that when the data-generating process is a well-specified VAR, the standard impulse response function is the best option. On the other hand, if the sample size is not large enough and the model lag length is misspecified, the local prediction estimator becomes a competitive alternative. Since we examine causality in pairs, our number of observations is sufficient in terms of degrees of freedom. Our lag length selection criterion also provided appropriately consistent results. Therefore, we used the VAR model impulse response function instead of the local projection method.

We use the VAR model rather than structural VAR because the potential problem of a VAR model is that it is usually impossible to measure the impact a sudden change in one variable will have on other variables in the model. However, there is no sudden change in the variables except IPI; and for that variable, we used restricted data where there is no break or sudden change. Figure A1, given in Appendix B, illustrates this.

6. Empirical findings

According to table 3 and table 4, all the variables are integrated of order one. Firstly, we analyzed the causal relationship between bank loans (BL) and bank deposits (DEP). According to Johansen cointegration test results, no cointegration/long-run relation between these variables was found.[5]

Therefore, the VAR model is used to investigate the short-run causal relationship between these variables. Table 5 and table 6 present the results. According to table 5, there is no short-run Granger causality running from DEP to BL. However, according to the results in table 6, there is a short-run Granger causality running from BL to DEP. Consequently, there is a unidirectional short-run Granger causality running from BL to DEP. Moreover, since the direction of the causal relation runs from BL to DEP, only the impulse response function that shows the response of DEP to BL is given in figure 5. It indicates that the effect of BL on DEP is positive.

Table 3 – *Unit root tests (2008-2022)*

Variable note: D_ stands for First Difference	**ADF test probability values**			**PP test probability values**			**Decision**
	Intercept	Trend and Intercept	None	Intercept	Trend and Intercept	None	
M2	0.9978	0.9506	1.0000	0.9983	0.9486	1.0000	I (1)
D_M2	0.0000***	0.0000***	0.0001***	0.0000***	0.0000***	0.0000***	
MIER	0.6857	0.9793	0.3394	0.6690	0.9662	0.3257	I (1)
D_MIER	0.0000***	0.0000***	0.0000***	0.0000***	0.0000***	0.0000***	
BL	0.9230	0.3021	0.9995	0.8866	0.6165	1.0000	I (1)
D_BL	0.0021***	0.0125***	0.0162**	0.0000***	0.0000***	0.0000***	
MRES	0.6888	0.9665	0.9995	0.7020	0.9488	0.9980	I (1)
D_MRES	0.0000***	0.0000***	0.0000***	0.0000***	0.0000***	0.0000***	
DEP	0.8845	0.3186	1.0000	0.8840	0.2931	1.0000	I (1)
D_DEP	0.0000***	0.0000***	0.0004***	0.0000***	0.0000***	0.0000***	
IPI	0.7703	0.0196**	0.9300	0.8921	0.0309**	0.9852	I (1)
D_IPI	0.0000***	0.0000***	0.0000***	0.0000***	0.0000***	0.0000***	

The lag length is automatically selected according to the Schwarz information criterion in the ADF unit root test. In the PP unit root test, the Newey-West bandwidth is automatically selected using the Barlett kernel method.

Notes: *** stationary at 1% significance level, ** stationary at 5% significance level, * stationary at 10% significance level.

[5] Cointegration results can be submitted upon request.

Table 4 – *Unit root tests (restricted data: 2011-2019)*

Variable Note: D_ stands for First Difference	**ADF Test Probability Values**			**PP Test Probability Values**			**Decision**
	Intercept	Trend and Intercept	None	Intercept	Trend and Intercept	None	
M2	0.9938	0.2633	1.0000	0.9989	0.2731	1.0000	I (1)
D_M2	0.0000***	0.0000***	0.0000***	0.0000***	0.0000***	0.0000***	
BL	0.0469**	0.7315	0.9990	0.0324**	0.8192	1.0000	I (1)
D_BL	0.0073***	0.0000***	0.0401**	0.0000***	0.0000***	0.0000***	
IPI	0.6602	0.5328	0.9987	0.7483	0.0248**	0.9987	I (1)
D_IPI	0.0000***	0.0000***	0.0000***	0.0000***	0.0000***	0.0000***	

In the ADF unit root test, the lag length is automatically selected according to the Schwarz Information criterion. In the PP unit root test, the Newey-West bandwidth is automatically selected using the Barlett kernel method.

Notes: *** Stationary at 1% significance level, ** Stationary at 5% significance level, * Stationary at 10% significance level.

Table 5 – *VAR estimates, equation 1 (dependent variable: D_BL)*

	Coefficient	Std. Error	t-Statistic	Prob.
C(1)	0.347238	0.097450	3.563255	0.0005
C(2)	0.215475	0.099753	2.160084	0.0327
C(3)	0.191900	0.099423	1.930142	0.0559
C(4)	–0.137512	0.100505	–1.368203	0.1738
C(5)	–0.026901	0.100918	–0.266560	0.7903
C(6)	0.052984	0.100796	0.525656	0.6001
C(7)	–0.067444	0.099871	–0.675309	0.5008
C(8)	0.017308	0.099367	0.174186	0.8620
C(9)	0.110336	0.104024	1.060683	0.2909
C(10)	–0.189069	0.104458	–1.810002	0.0728
C(11)	–0.148819	0.099662	–1.493234	0.1380
C(12)	–0.153520	0.062965	–2.438168	0.0162
C(13)	–0.091398	0.062141	–1.470819	0.1439
C(14)	0.055984	0.061139	0.915686	0.3617
C(15)	0.138352	0.060726	2.278312	0.0245
C(16)	0.103391	0.061848	1.671689	0.0972
C(17)	0.001920	0.062450	0.030737	0.9755
C(18)	0.051449	0.062807	0.819158	0.4143
C(19)	0.024484	0.062095	0.394298	0.6941
C(20)	–0.041892	0.063913	–0.655445	0.5134
C(21)	0.007269	0.064746	0.112274	0.9108
C(22)	–0.064698	0.064226	–1.007352	0.3158
C(23)	0.008957	0.002618	3.421728	0.0008
R–squared: 0.388126 Adjusted R–squared: 0.276876 Prob (F–statistic): 0.000005				

S.R. Granger Causality (Wald Test):
H0: C(12)=……..=C(22)=0
Chi–square: 16.76132
Prob. 0.1151
Criteria: Rejecting null (H0) indicates that there is S.R. causality running from the independent variable to dependent variable. 5% significance level was used in all decision making.
There is no S.R. Granger causality running from DEP to BL.

Diagnostic Tests:
*****Breusch–Godfrey Serial Correlation LM test:
Prob.Chi–Square(2): 0.6374
* Heteroskedasticity Test: Breusch–Pagan–Godfrey:

Prob.Chi–Square(22): 0.5930

Equation: D_BL = C(1)*D_BL(-1)+C(2)*D_BL(-2)+C(3)*D_BL(-3)+C(4)*D_BL(-4)+C(5)*D_BL(-5)+C(6)*D_BL(-6)+C(7)*D_BL(-7)+C(8)*D_BL(-8)+C(9)*D_BL(-9)+C(10)*D_BL(-10)+C(11)*D_BL(-11)+C(12)*D_DEP(-1)+C(13)*D_DEP(-2)+C(14)*D_DEP(-3)+C(15)*D_DEP(-4)+C(16)*D_DEP(-5)+C(17)*D_DEP(-6)+C(18)*D_DEP(-7)+C(19)*D_DEP (-8)+C(20)*D_DEP(-9)+C(21)*D_DEP(-10)+C(22)*D_DEP(-11)+C(23)
Notes: (1) C( ) stands for coefficient. (2) The lag level of the variables is given in the parentheses next to the variable names.

Table 6 – *VAR Estimates, equation 2 (dependent variable: D_DEP)*

	Coefficient	Std. Error	t-Statistic	Prob.
C(24)	0.350768	0.140349	2.499248	0.0138
C(25)	0.095071	0.143667	0.661743	0.5094
C(26)	–0.022873	0.143192	–0.159735	0.8734
C(27)	0.009344	0.144750	0.064555	0.9486
C(28)	0.068888	0.145345	0.473966	0.6364
C(29)	–0.008106	0.145169	–0.055835	0.9556
C(30)	0.125283	0.143836	0.871008	0.3855
C(31)	–0.143391	0.143111	–1.001951	0.3184
C(32)	0.001326	0.149817	0.008850	0.9930
C(33)	–0.365952	0.150443	–2.432490	0.0165
C(34)	–0.278603	0.143536	–1.940990	0.0546
C(35)	–0.335351	0.090684	–3.698019	0.0003
C(36)	–0.149393	0.089497	–1.669239	0.0977
C(37)	0.142979	0.088054	1.623762	0.1070
C(38)	–0.012154	0.087459	–0.138966	0.8897
C(39)	0.138778	0.089076	1.557974	0.1219
C(40)	0.083190	0.089942	0.924926	0.3568
C(41)	0.003702	0.090457	0.040926	0.9674
C(42)	–0.095757	0.089430	–1.070749	0.2864
C(43)	–0.088372	0.092049	–0.960046	0.3389
C(44)	–0.137829	0.093248	–1.478085	0.1420
C(45)	–0.281986	0.092500	–3.048492	0.0028
C(46)	0.022793	0.003770	6.046047	0.0000

R–squared: 0.375609
Adjusted R–squared: 0.262083
Prob (F–statistic): 0.000014

S.R. Granger Causality (Wald Test):
H0: C(24)=……..=C(34)=0
Chi–square: 34.57299
Prob. 0.0003
There is S.R. Granger causality running from BL to DEP.

Diagnostic Tests:
*Breusch–Godfrey Serial Correlation LM test:
Prob.Chi–Square(2): 0.2640
* Heteroskedasticity Test: Breusch–Pagan–Godfrey:

Prob.Chi–Square(22): 0.4446

Equation: D_DEP = C(24)*D_BL(-1)+C(25)*D_BL(-2)+C(26)*D_BL(-3)+C(27)*D_BL(-4)+C(28)*D_BL(-5)+C(29)*D_BL(-6)+C(30)*D_BL(-7)+C(31)*D_BL(-8)+C(32)*D_BL(-9)+C(33)*D_BL(-10)+C(34)*D_BL(-11)+C(35)*D_DEP(-1)+C(36)*D_DEP(-2)+C(37)*D_DEP(-3)+C(38)*D_DEP(-4)+C(39)*D_DEP(-5)+C(40)*D_DEP(-6)+C(41)*D_DEP(-7)+C(42)*D_DEP(-8)+C(43)*D_DEP(-9)+C(44)*D_DEP(-10)+C(45)*D_DEP(-11)+C(46)
Notes: (1) C( ) stands for coefficient. (2) The lag level of the variables is given in the parentheses next to the variable names.

As mentioned earlier, the optimal lag length was determined based on information criteria. However, to examine simultaneity between bank loans and bank deposits for theoretical reasons discussed at the beginning of section 3, the minimum lag option (one lag) for the variables was employed. This choice was necessitated by the nature of the VAR methodology, wherein only lagged versions of variables can be included as independent variables in the model. The results are given in appendix C. According to the results that do not contain serial correlation and heteroscedasticity, this time the Granger causality relationship emerges bidirectionally between BL and DEP.

Secondly, the causal relation between bank loans (BL) and money supply (M2) was analyzed. Since no cointegration relation was found, by using the VAR model, the short-run causal relationship between these variables was investigated. According to table 7, no short-run Granger causality running from M2 to BL was found. On the other hand, table 8 indicates that there is a short-run Granger causality running from BL to M2. Since there is a one-way causal relation that runs from BL to M2, only the response of M2 to BL is given in figure 6 in order to investigate the sign of the effect of BL on M2. According to figure 6, the effect is positive.

Figure 5 – *Impulse response function*

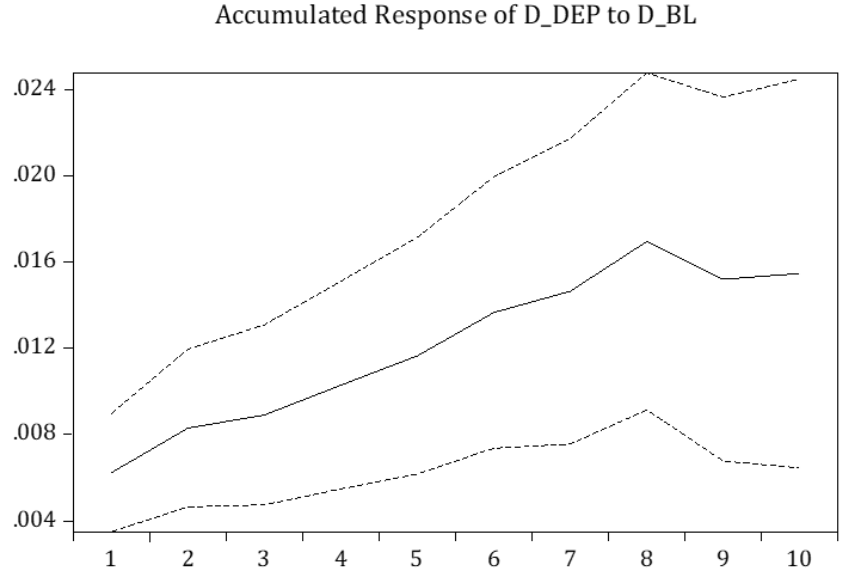


Table 7 – *VAR Estimates, equation 1 (dependent variable: D_BL)*

	Coefficient	Std. Error	t-Statistic	Prob.
C(1)	0.455636	0.075399	6.042993	0.0000
C(2)	–0.074890	0.057265	–1.307771	0.1929
C(3)	0.009311	0.001621	5.744241	0.0000

R–squared: 0.195396 Adjusted R–squared: 0.184739
Prob (F–statistic): 0.000000

Diagnostic Tests:
–Breusch–Godfrey Serial Correlation LM test: Prob.Chi–Square(2): 0.0169
(There is Serial correlation problem*)
–Heteroskedasticity Test: Breusch–Pagan–Godfrey:

Prob.Chi–Square(2): 0.1246

S.R. Granger Causality (Wald Test):
H0: C(2)=0
Chi–square: 1.710265
Prob. 0.1910
There is no S.R. Granger causality running from M2 to BL.

Equation: D_BL = C(1)*D_BL(-1)+C(2)*D_M2(-1)+C(3)
Notes: (1) C( ) stands for coefficient. (2) The lag level of the variables is given in the parentheses next to the variable names.
Notes: * This problem was eliminated by increasing the lag to 3 (Breusch-Godfrey Serial Correlation LM test: Prob.Chi-Square(2): 0.6613). Also there is no problem with respect to heteroskedasticity when lag length is 3 (Heteroskedasticity Test: Breusch-Pagan-Godfrey: Prob.Chi-Square(6): 0.4063). Also, S.R. Granger Causality Results did not change (Prob. 0.2880). In other words, still there is no causality running from M2 to BL.

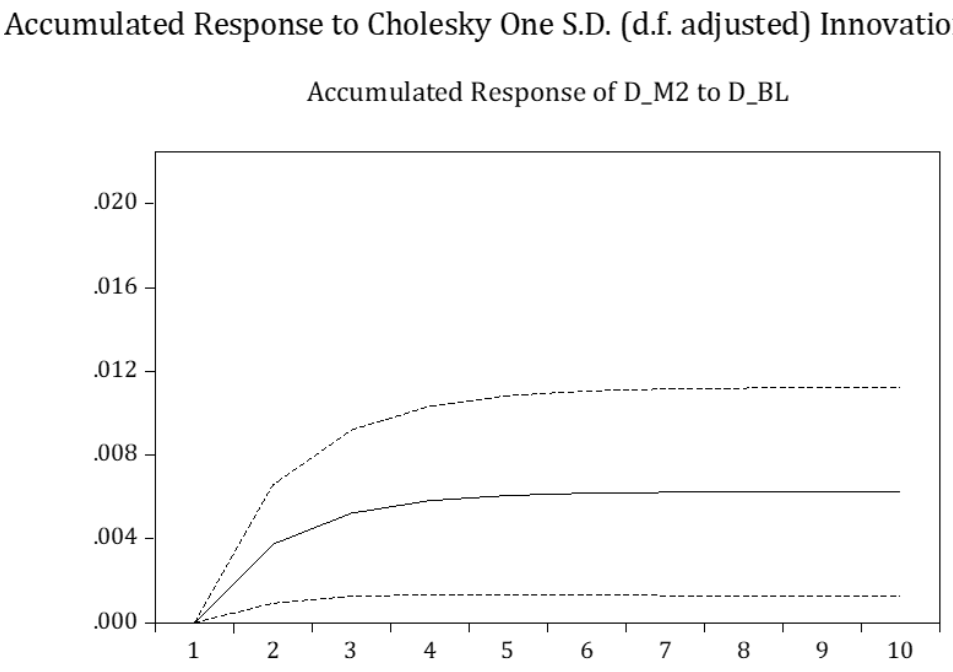
Table 8 – *VAR Estimates, Equation 2 (Dependent variable: D_M2)*

	Coefficient	Std. Error	t-Statistic	Prob.
C(4)	0.294473	0.109500	2.689258	0.0080
C(5)	–0.061031	0.083164	–0.733863	0.4642
C(6)	0.010931	0.002354	4.643566	0.0000
R–squared: 0.045707			Adjusted R–squared: 0.033068	
Prob (F–statistic): 0.029239				
Diagnostic Tests:				
*Breusch–Godfrey Serial Correlation LM test: Prob.Chi–Square(2): 0.1761				
* Heteroskedasticity Test: Breusch–Pagan–Godfrey:				
Prob.Chi–Square(2): 0.2689				

S.R. Granger Causality (Wald Test):
H0: C(4)=0
Chi–square: 7.232109
Prob. 0.0072
There is S.R. Granger Causality running from BL to M2.

Equation: D_M2 = C(4)*D_BL(-1)+C(5)*D_M2(-1)+C(6)
Notes: (1) C( ) stands for coefficient. (2) The lag level of the variables is given in the parentheses next to the variable names.

Figure 6 – *Impulse-response function*



We also investigated the causal relation between bank loans (BL) and monetary reserves (MRES). Johansen cointegration test indicated a long-run relationship between these variables in the linear model with constant and trend indicated by the information criteria. Therefore, VECM estimates were used to analyze the causal relation between these variables. According to table 9, there is a long-run Granger causality running from MRES to BL. However, there is no short-run causality running from MRES to BL. On the other hand, table 10 indicates that there is no long-run Granger causality running from BL to MRES, but there is a short-run Granger causality running from BL to MRES. Consequently, the results differ in terms of the short-run and the long-run. In the short run, the direction of causality runs from BL to MRES. However, in the long run, it runs from MRES to BL. Therefore, it is better to give impulse-response functions in both directions. According to the results given in figure 7, the effect is positive in both directions.

Table 9 – *VECM estimates, equation 1 (dependent variable: D(BL))*

	Coefficient	Std. Error	t-Statistic	Prob.
C(1)	–0.121280	0.034168	–3.549551	0.0005
C(2)	0.377271	0.082413	4.577829	0.0000
C(3)	0.148848	0.087341	1.704204	0.0906
C(4)	0.310124	0.086569	3.582386	0.0005
C(5)	–0.005690	0.090460	–0.062897	0.9499
C(6)	0.088967	0.092598	0.960794	0.3384
C(7)	0.089491	0.092177	0.970862	0.3334
C(8)	0.000731	0.014910	0.049027	0.9610
C(9)	0.015814	0.014277	1.107660	0.2700
C(10)	–0.004755	0.014274	–0.333096	0.7396
C(11)	0.005137	0.015005	0.342321	0.7326
C(12)	–0.017308	0.015238	–1.135873	0.2580
C(13)	–0.005142	0.015521	–0.331302	0.7409
C(14)	–0.000224	0.002668	–0.084155	0.9331
R–squared: 0.345070 Adjusted R–squared: 0.282002 Prob (F–statistic): 0.000000				
Diagnostic Tests: *****Breusch–Godfrey Serial Correlation LM test: Prob.Chi–Square(2): 0.7772 * Heteroskedasticity Test: Breusch–Pagan–Godfrey: Prob.Chi–Square(14): 0.3204				

L.R Granger Causality:
Criteria: Coefficient of cointegrated equation (C(1)) needs to be statistically significant and negative in sign. When this condition holds, there is S.R. adjustment to L.R. equilibrium by the percentage of the coefficient of cointegrated equation. Also, it means that there is L.R. Granger causality running from independent variable to dependent variable.
Note: 5% significance level was used in all decision making.
Result: There is L.R. Granger Causality running from MRES to BL.
S.R. Granger Causality (Wald Test):
H0: C(8)=……..=C(13)=0
Chi–square: 3.453440
Prob. 0.7502
There is no S.R. Granger Causality running from MRES to BL.

Equation: D(BL) = C(1)*(BL(-1) - 0.144941347433*MRES(-1) - 0.0117696235521*@TREND(08M01) - 16.8543498696)+C(2)*D(BL(-1))+C(3)*D(BL(-2))+C(4)*D(BL(-3))+C(5)*D(BL(-4))+C(6)*D(BL(-5))+C(7)*D(BL(-6))+C(8)*D(MRES(-1))+C(9)*D(MRES(-2))+C(10)*D(MRES(-3))+C(11)*D(MRES(-4))+C(12)*D(MRES(-5))+C(13)*D(MRES(-6))+C(14)
Notes: (1) C( ) stands for coefficient. (2) The lag level of the variables is given in the parentheses next to the variable names (3) D( ) means first difference of that variable.

Table 10 – *VECM Estimates, Equation 2 (Dependent variable: D(MRES))*

	Coefficient	Std. Error	t-Statistic	Prob.
C(15)	0.600140	0.190118	3.156665	0.0020
C(16)	0.437271	0.458568	0.953556	0.3420
C(17)	0.169677	0.485993	0.349135	0.7275
C(18)	0.228049	0.481695	0.473430	0.6367
C(19)	–1.646732	0.503344	–3.271584	0.0014
C(20)	1.548564	0.515241	3.005512	0.0032
C(21)	–1.465518	0.512901	–2.857313	0.0049
C(22)	0.236001	0.082961	2.844721	0.0051
C(23)	0.061277	0.079442	0.771338	0.4419
C(24)	–0.077098	0.079425	–0.970701	0.3334
C(25)	–0.049901	0.083495	–0.597652	0.5511
C(26)	0.129264	0.084786	1.524591	0.1297
C(27)	0.084042	0.086361	0.973144	0.3322
C(28)	0.023253	0.014844	1.566514	0.1196

R-squared: 0.281191 Adjusted R-squared: 0.211973
Prob (F-statistic): 0.000013

Diagnostic Tests:
*Breusch-Godfrey Serial Correlation LM test:
Prob.Chi-Square(2): 0.3828
* Heteroskedasticity Test: Breusch-Pagan-Godfrey:
Prob.Chi-Square(14): 0.1920

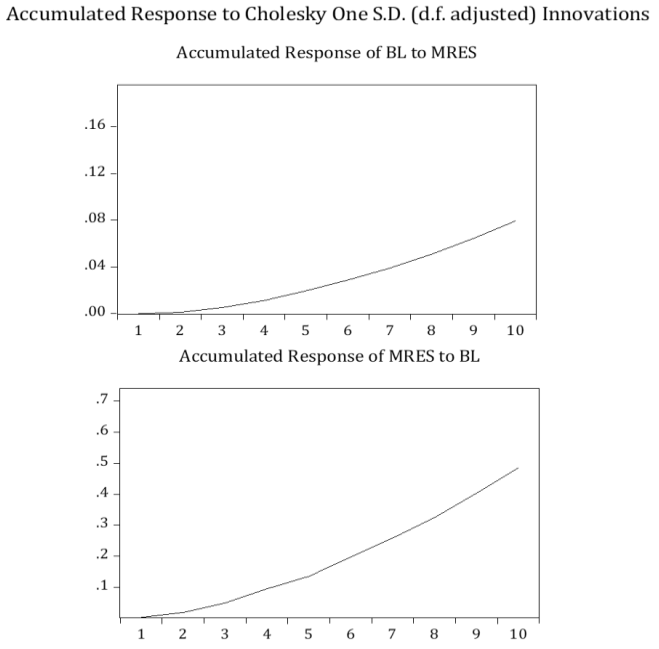
L.R Granger Causality:
There is no L.R. Granger Causality running from BL to MRES.

S.R. Granger Causality (Wald Test):
H0: C(16)=……..=C(21)=0
Chi-square: 25.28889
Prob. 0.0003
There is S.R. Granger Causality running from BL to MRES.

Equation: D(MRES) = C(15)*(BL(-1) - 0.144941347433*MRES(-1) - 0.0117696235521*@TREND(08M01) - 16.8543498696)+C(16)*D(BL(-1))+C(17)*D(BL(-2))+C(18)*D(BL(-3))+C(19)*D(BL(-4))+C(20)*D(BL(-5))+C(21)* D(BL(-6))+C(22)*D(MRES(-1))+C(23)*D(MRES(-2))+C(24)*D(MRES(-3))+C(25)*D(MRES(-4))+C(26)*D(MRES(-5)) +C(27)*D(MRES(-6))+C(28)

Notes: (1) C( ) stands for coefficient. (2) The lag level of the variables is given in the parentheses next to the variable names (3) D( ) means first difference of that variable.

Figure 7 – *Impulse response functions*



We analyzed the causal relation between bank loans (BL) and the money multiplier (MIER) in the next specification. The Johansen cointegration test indicated that there is a long-run relationship between these variables in the linear model with constant and trend indicated by the information criteria. Therefore, VECM estimates were investigated in order to capture the long-run and the short-run causality between these variables. According to table 11, although there is a long-run Granger causality running from MIER to BL, there is no Granger causality in the same way as in the short-run. Table 12 indicates that there is both a short-run and a long-run Granger causality running from BL to MIER. Consequently, in the long run, Granger causality between these variables is bidirectional. However, in the short run, it is unidirectional from BL to MIER. Therefore, in order to investigate the sign of the effect of each variable on other variables, impulse-response functions were given in figure 8. According to figure 8, the effects are negative in both directions.

Table 11 – *VECM Estimates, equation 1 (dependent variable: D(BL))*

	Coefficient	Std. Error	t-Statistic	Prob.
C(1)	–0.114862	0.031779	–3.614370	0.0004
C(2)	0.372596	0.082343	4.524910	0.0000
C(3)	0.153462	0.087506	1.753731	0.0817
C(4)	0.306351	0.086508	3.541295	0.0005
C(5)	0.006539	0.090025	0.072632	0.9422
C(6)	0.095800	0.092931	1.030870	0.3044
C(7)	0.079350	0.092231	0.860333	0.3911
C(8)	–0.007520	0.014333	–0.524646	0.6007
C(9)	–0.016192	0.013888	–1.165883	0.2457
C(10)	0.004777	0.013801	0.346106	0.7298
C(11)	–0.008782	0.014411	–0.609360	0.5433
C(12)	0.016861	0.014649	1.150984	0.2518
C(13)	0.009282	0.014830	0.625910	0.5324
C(14)	–0.000409	0.002746	–0.148798	0.8819

R–squared: 0.353497 Adjusted R–squared: 0.291241
Prob (F–statistic): 0.000000

Diagnostic Tests:
*****Breusch–Godfrey Serial Correlation LM test:
Prob.Chi–Square(2): 0.8163
* Heteroskedasticity Test: Breusch–Pagan–Godfrey:

Prob.Chi–Square(14): 0.2032

L.R Granger Causality:
There is L.R. Granger Causality running from MIER to BL.

S.R. Granger Causality (Wald Test):
H0: C(8)=……..=C(13)=0
Chi–square: 4.726828
Prob. 0.5793
There is no S.R. Granger Causality running from MIER to BL.

Notes: (1) C( ) stands for coefficient. (2) The lag level of the variables is given in the parentheses next to the variable names (3) D( ) means first difference of that variable.
Equation: D(BL) = C(1)*(BL(-1)+0.136329291938*MIER(-1) - 0.013787990838*@TREND(08M01) - 19.6757197931)+C(2)*D(BL(-1))+C(3)*D(BL(-2))+C(4)*D(BL(-3))+C(5)*D(BL(-4))+C(6)*D(BL(-5))+C(7)*D(BL(-6))+C(8)*D(MIER(-1))+C(9)*D(MIER(-2))+C(10)*D(MIER(-3))+C(11)*D(MIER(-4))+C(12)*D(MIER(-5))+C(13)*D(MIER(-6))+C(14)

Table 12 – *VECM Estimates, equation 2 (Dependent variable: D(MIER))*

	Coefficient	Std. Error	t-Statistic	Prob.
C(15)	–0.639695	0.182465	–3.505847	0.0006
C(16)	–0.315572	0.472787	–0.667472	0.5056
C(17)	–0.007262	0.502429	–0.014455	0.9885
C(18)	–0.123013	0.496700	–0.247660	0.8048
C(19)	1.831823	0.516894	3.543904	0.0005
C(20)	–1.540312	0.533580	–2.886750	0.0045
C(21)	1.553356	0.529560	2.933292	0.0039
C(22)	0.265218	0.082297	3.222684	0.0016
C(23)	0.061221	0.079741	0.767746	0.4440
C(24)	–0.055435	0.079243	–0.699556	0.4854
C(25)	–0.038465	0.082746	–0.464857	0.6428
C(26)	0.136972	0.084110	1.628483	0.1058
C(27)	0.117455	0.085148	1.379425	0.1700
C(28)	–0.024279	0.015764	–1.540166	0.1259

R–squared: 0.285748 Adjusted R–squared: 0.216968
Prob (F–statistic): 0.000009

Diagnostic Tests:
*****Breusch–Godfrey Serial Correlation LM test:
Prob.Chi–Square(2): 0.2678
* Heteroskedasticity Test: Breusch–Pagan–Godfrey:

Prob.Chi–Square(14): 0.4716

L.R Granger Causality:
There is L.R. Granger Causality running from BL to MIER.

S.R. Granger Causality (Wald Test):
H0: C(16)=……..=C(21)=0
Chi–square: 25.76853
Prob. 0.0002
There is S.R. Granger Causality running from BL to MRES.

Equation: D(MIER) = C(15)*(BL(-1)+0.136329291938*MIER(-1) - 0.013787990838*@TREND(08M01) - 19.6757197931)+C(16)*D(BL(-1))+C(17)*D(BL(-2))+C(18)*D(BL(-3))+C(19)*D(BL(-4))+C(20)*D(BL(-5))+C(21)*D(BL(-6))+C(22)*D(MIER(-1))+C(23)*D(MIER(-2))+C(24)*D(MIER(-3))+C(25)*D(MIER(-4))+C(26)*D(MIER(-5))+C(27)*D(MIER(-6))+C(28)
Notes: (1) C( ) stands for coefficient. (2) The lag level of the variables is given in the parentheses next to the variable names (3) D( ) means first difference of that variable.

The causality between bank loans (BL) and the industrial production index (IPI) was also analyzed. Since no cointegration relation between these variables was found, VAR estimates were constructed in order to investigate the short-run causal relationship between these variables. According to the results given in table 13 and table 14, there is a unidirectional short-run Granger causality running from BL to IPI. In order to investigate the sign of the effect of BL on IPI, impulse response function was given in figure 9. According to figure 9, the response of IPI to BL is positive.

Figure 8 – *Impulse response functions*

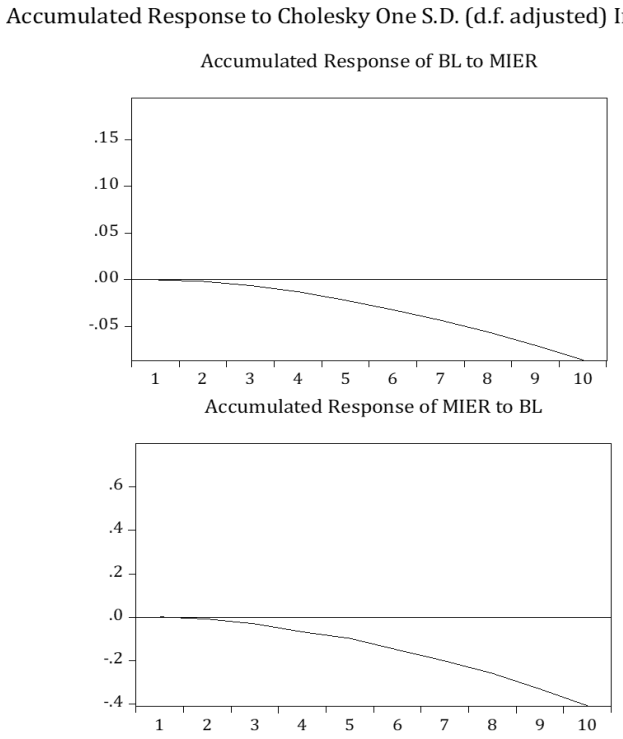


Table 13 – *VAR Estimates, equation 1 (dependent variable: D_BL)*

	Coefficient	Std. Error	t-Statistic	Prob.	
C(1)	0.247952	0.097163	2.551918	0.0123	**S.R. Granger Causality (Wald Test):**
C(2)	0.005656	0.103197	0.054803	0.9564	H0: C(4)=C(5)=C(6)=0
C(3)	0.235328	0.100041	2.352311	0.0207	Chi-square: 6.275601
C(4)	0.024313	0.056564	0.429836	0.6683	Prob. 0.0980
C(5)	0.133148	0.065365	2.036982	0.0444	There is no S.R. Granger causality running from IPI to BL.
C(6)	0.007820	0.056982	0.137240	0.8911	
C(7)	0.005998	0.001922	3.121330	0.0024	
R-squared: 0.239326			Adjusted R-squared: 0.192274		
Prob (F-statistic): 0.000141					
Diagnostic Tests:					
*Breusch-Godfrey Serial Correlation LM test: Prob.Chi-Square(2): 0.9493					
*Heteroskedasticity Test: Breusch-Pagan-Godfrey:					
Prob.Chi-Square(6): 0.8643					

Equation: D_BL = C(1)*D_BL(-1)+C(2)*D_BL(-2)+C(3)*D_BL(-3)+C(4)*D_IPI(-1)+C(5)*D_IPI(-2)+C(6)*D_IPI(-3)+C(7)
Notes: (1) C( ) stands for coefficient. (2) The lag level of the variables is given in the parentheses next to the variable names

Table 14 – *VAR Estimates, equation 2 (dependent variable: D_IPI)*

	Coefficient	Std. Error	t-Statistic	Prob.
C(8)	0.784710	0.171090	4.586526	0.0000
C(9)	0.060372	0.181715	0.332232	0.7404
C(10)	–0.018682	0.176158	–0.106053	0.9158
C(11)	–0.674532	0.099602	–6.772286	0.0000
C(12)	–0.277224	0.115099	–2.408573	0.0179
C(13)	–0.210795	0.100337	–2.100869	0.0382
C(14)	–0.002712	0.003384	–0.801450	0.4248

R–squared: 0.391440 Adjusted R–squared: 0.353797
Prob (F–statistic): 0.000000

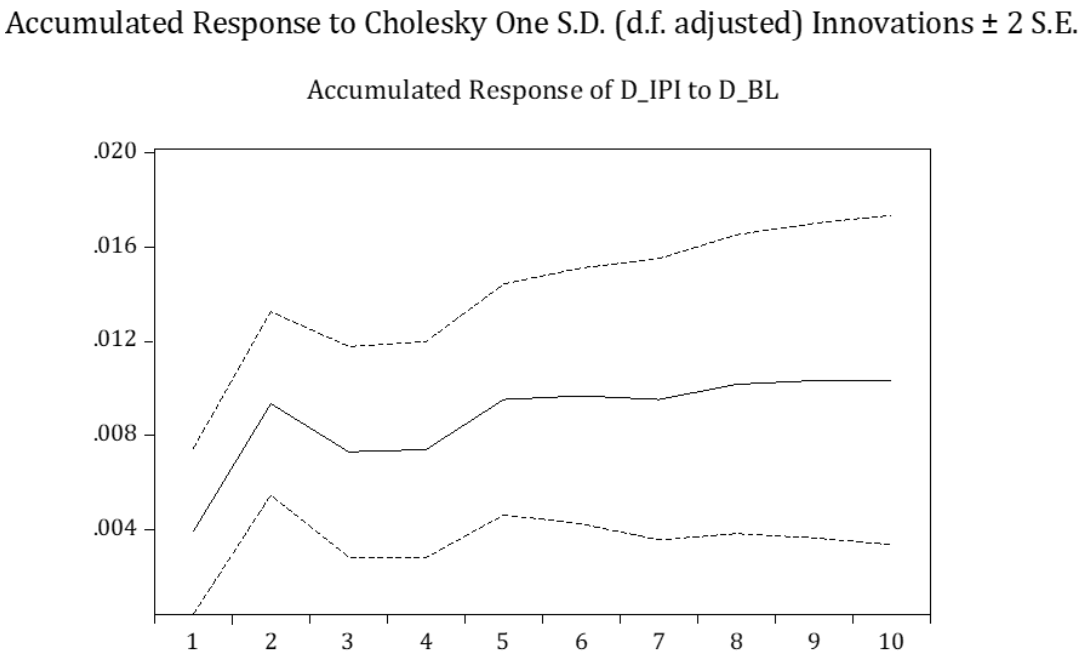
Diagnostic Tests:
*Breusch–Godfrey Serial Correlation LM test: Prob.Chi–Square(2): 0.2048
*Heteroskedasticity Test: Breusch–Pagan–Godfrey:

Prob.Chi–Square(6): 0.3359

S.R. Granger Causality (Wald Test):
H0: C(8)=C(9)=C(10)=0
Chi–square: 23.64312
Prob. 0.0000
There is S.R. Granger causality running from BL to IPI

Equation: D_IPI = C(8)*D_BL(-1)+C(9)*D_BL(-2)+C(10)*D_BL(-3)+C(11)*D_IPI(-1)+C(12)*D_IPI(-2)+C(13)*D_IPI(-3)+C(14)
Notes: (1) C( ) stands for coefficient. (2) The lag level of the variables is given in the parentheses next to the variable names.

Figure 9 – *Impulse response function*



We investigated the causality between the industrial production index (IPI) and money supply (M2). Since no cointegration between these variables was found, VAR estimates were constructed to analyze the short-run causal relationship between these variables. According to table 15 and table 16, unidirectional short-run Granger causality runs from IPI to M2. Therefore, in order to investigate the sign of the effect of IPI on M2, the impulse response function that shows the response of M2 to IPI is given in figure 10. According to that figure, the effect is positive.

Table 15 – *VAR Estimates, equation 1 (dependent variable: D_IPI)*

	Coefficient	Std. Error	t-Statistic	Prob.	**S.R. Granger Causality (Wald Test):**
C(1)	–0.468294	0.087571	–5.347599	0.0000	H0: C(2)=0
C(2)	–0.028683	0.103061	–0.278312	0.7813	Chi–square: 0.077457
C(3)	0.005893	0.002358	2.499766	0.0140	Prob. 0.7808
R–squared: 0.222929			Adjusted R–squared: 0.207840		There is no S.R. Granger causality running from M2 to IPI.
Prob (F–statistic): 0.000002					
Diagnostic Tests:					
*****Breusch–Godfrey Serial Correlation LM test: Prob.Chi–Square(2): 0.3549					
*Heteroskedasticity Test: Breusch–Pagan–Godfrey:					
Prob.Chi–Square(2): 0.1317					

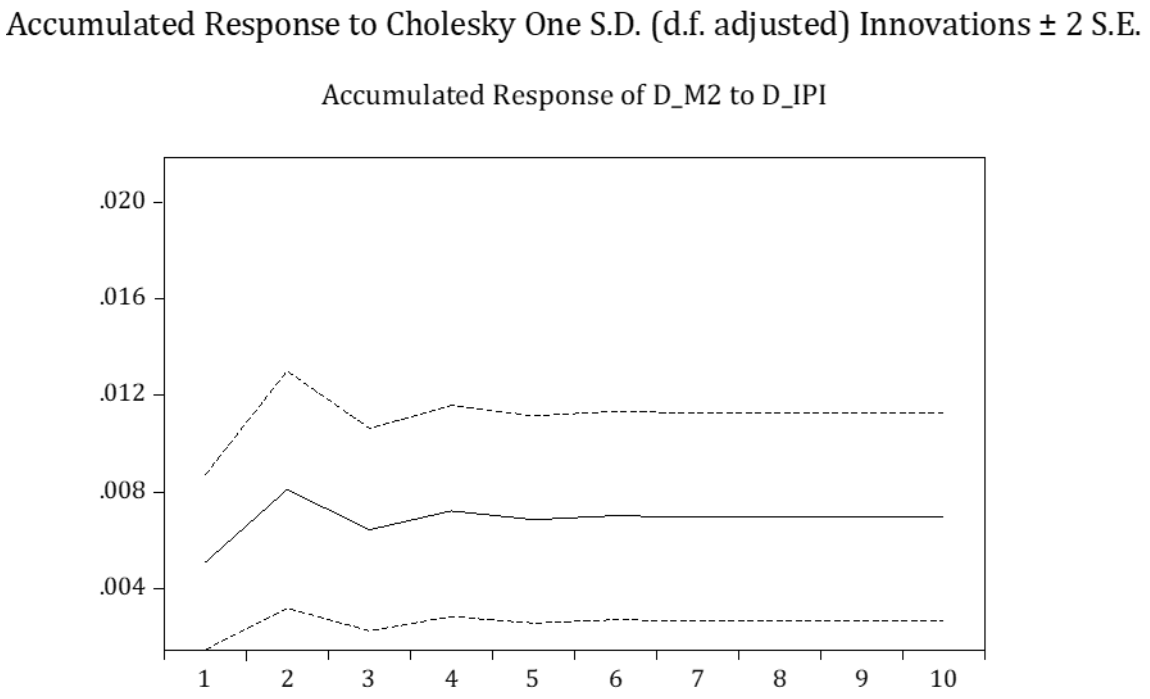
Equation: D_IPI = C(1)*D_IPI(-1)+C(2)*D_M2(-1)+C(3)
Notes: (1) C( ) stands for coefficient. (2) The lag level of the variables is given in the parentheses next to the variable names.

Table 16 – *VAR Estimates, equation 2 (Dependent variable: D_M2)*

	Coefficient	Std. Error	t-Statistic	Prob.	**S.R. Granger Causality (Wald Test):**
C(4)	0.161934	0.083494	1.939471	0.0552	H0: C(4)=0
C(5)	–0.041666	0.098263	–0.424021	0.6724	Chi–square: 3.761548
C(6)	0.013238	0.002248	5.889471	0.0000	Prob. 0.0524
R–squared: 0.035511			Adjusted R–squared: 0.016783		There is S.R. Granger causality running from IPI to M2.
Prob (F–statistic): 0.155347					
Diagnostic Tests:					
*****Breusch–Godfrey Serial Correlation LM test: Prob.Chi–Square(2): 0.3051					
*Heteroskedasticity Test: Breusch–Pagan–Godfrey:					
Prob.Chi–Square(2): 0.7911					

Equation: D_M2 = C(4)*D_IPI(-1)+C(5)*D_M2(-1)+C(6)
Notes: (1) C( ) stands for coefficient. (2) The lag level of the variables is given in the parentheses next to the variable names.

Figure 10 – *Impulse response function*



Lastly, we need to mention that some of the models have low R-squared since our purpose is to investigate bilateral relations. In other words, in every model, we have two variables. This is reasonable since we put only a little stress on predictive purposes. Rather, we focus on the explanatory purpose for the models. R-squared explains the predictive fit of the model, but our main aim is to explain the relationships. Even so, we report R-squared as an effect size.

7. Hypotheses and theoretical implications

We ran ten hypotheses separately to evaluate causality among monetary aggregates. We summarize the empirical results in table 17, including short-term and long-term findings. The first and second hypotheses represent simple propositions of the EMH. The first one is that loans make deposits. Table 17 demonstrates no long-run relationship between bank loans (BL) and bank deposits (DEP). However, there is a short-term relationship between bank loans and deposits. Besides, we examine the simultaneity between bank loans and bank deposits, albeit deviating from the consistency of our methodology by disregarding the lag selection criteria. Theoretically, we need to test this simultaneity, even in the face of violating our model's lag selection criteria. We run the estimation with a single lag option, the minimum and most appropriate choice for evaluating simultaneity. Table A1 and table A2 in appendix C show the results: that there is a bidirectional causality between bank loan and deposit with the single lag option. This outcome is consistent with theoretical foundation, indicating the simultaneous occurrence of deposits and loans following a positive response by banks to a demand for credit. However, it is not consistent

with the empirical methodology of lag selection criteria. Upon examining the results of the single lag option and of the lagged version in general, bidirectional causality between two series evolves into unidirectional causality from bank loans to bank deposits. This unidirectional causality may be a consequence of banks' credit supporting and influencing future borrowing as well as deposits. Additionally, unidirectional causality from bank loan to deposit implies no trace of the exogenous money theory, which conceptualizes banks as passive agents collecting deposit subsequently lending them. The second hypothesis is that the money supply is driven by bank loans. According to the findings, there is no long-run relationship between bank loans (BL) and money supply (M2), but there is a short-term relationship between these two series. According to the short-term findings, the first two hypotheses cannot be rejected. Therefore, the findings support the validity of EMH in Türkiye and implies that banks are the leading actors in the creation of money, and they are not financial intermediaries that collect deposit and lend money out of deposit. They can create money themselves.

The third hypothesis represents the causality of bank loans with monetary reserves. The findings show a long-run relationship between bank loans and monetary reserves (MRES). The direction of causality is from monetary reserves to bank loans. Causality in the other direction is not valid. On the other hand, there is a short-run relationship in the opposite direction, where monetary reserves do not lead to bank loans, but loans influence monetary reserves. Consequently, we reject the third hypothesis, depending on the long-run results. However, it cannot be rejected according to the short-term results. This is one of the interesting findings. It implies that banks lend money and then maintain reserves according to short-run findings.[6] In contrast, long-run findings indicate that monetary reserves influence bank loans, implying that the Central Bank may have a stance on the loan policy of banks and influence the level of bank loans.

The fourth hypothesis represents no causal relationship between bank loans and the money multiplier. However, the findings show a long-run relationship between bank loans and the money multiplier (MIER). The direction of causality is bidirectional. There is no bidirectional relationship in the short run, but bank loans influence the money multiplier. According to these results, we suggest rejecting the fourth hypothesis. Moreover, these findings are consistent with the inference we draw from the findings in the previous hypothesis. Therefore, the central bank's stance is active in money creation in the long run.

The fifth hypothesis represents bidirectional causality between GDP and money supply. The findings demonstrate no long-run relationship between GDP (IPI) and money supply. However, in the short-term, GDP causes money supply, while money supply does not lead to GDP. This implies that production may cause money demand, and thus its creation.

The sixth hypothesis represents bidirectional causality between bank loans and monetary reserves. Such a relationship does not exist in the short-run or long-run. So, we suggest rejecting the sixth hypothesis.

The seventh hypothesis represents bidirectional causality between bank loans and the money multiplier. The findings show a long-run bidirectional relationship among these series, but no bidirectional relationship in the short-run because the direction of causality in the short-run is from bank loans to the money multiplier. According to these results, the seventh hypothesis

[6] This result is consistent with the Turkish Central Bank's statement about reserve requirement ratios. It says that banks must maintain the required reserve for newly created credit up to two weeks after the date of the liability calculation. Please visit the following website for details: *Reserve requirement ratios*. TCMB. (n.d). https://www.tcmb.gov.tr/wps/wcm/connect/EN/TCMB+EN/Main+Menu/Core+Functions/Monetary+Policy/Reserve+Requirement+Ratios/

cannot be rejected due to the long-run results, but it can be rejected with regard to the short-term results.

The eighth hypothesis indicates causality from bank loans to the money multiplier. The findings demonstrate a long-run bidirectional relationship between bank loans and the money multiplier. In the short run, the empirical findings show causality from bank loans to the money multiplier. So, these results suggest that the eighth hypothesis can be rejected due to the long-run results, but it cannot be rejected with regard to the short-term results.

The ninth hypothesis represents a causality running from bank loans to GDP. The findings demonstrate no long-run relationship, but bank loans lead to GDP in the short-run. So, the ninth hypothesis cannot be rejected due to the short-term findings. It supports the circuitist idea that production requires bank loans.

The tenth hypothesis is that GDP causes money supply. The empirical findings suggest no long-run relationship, but GDP leads to money supply in the short run. So, the findings suggest that the tenth hypothesis cannot be rejected due to the short-term findings. The empirical findings suggest that circuitism fits best compared with horizontalism and structuralism, even if there is some partial evidence for horizontalism and structuralism. Besides, this partial evidence stems from the short-term findings of the third hypothesis for horizontalism and the long-term findings of the seventh hypothesis for Structuralism. However, some hypotheses of horizontalism and structuralism are rejected, while all circuitist hypotheses fail to be rejected.

Table 17 – *Causality test result*

		Short Term		**Long Term**			
DV	**INDV**	**Prob. val**	**Causality Result**	**ECT**	**Prob. val**	**Causality Result**	**IMPULSE–RESPONSE (sign)**
DEP	BL	0.000	BL causes DEP	–	–	–	Positive
BL	DEP	0.115	DEP doesn't cause BL	–	–	–	–
M2	BL	0.007	BL causes M2	–	–	–	Positive
BL	M2	0.191	M2 doesn't cause BL	–	–	–	–
BL	MRES	0.750	MRES doesn't cause BL	–0.121	0.000	MRES causes BL	Positive
MRES	BL	0.000	BL causes MRES	0.600	0.000	MRES doesn't cause BL	Positive
BL	MIER	0.579	MIER doesn't cause BL	–0.114	0.000	MIER causes BL	Negative
MIER	BL	0.000	BL causes MIER	–0.639	0.001	BL causes MIER	Negative
BL	IPI	0.098	IPI doesn't cause BL	–	–	–	–
IPI	BL	0.000	BL causes IPI	–	–	–	Positive
M2	IPI	0.052	IPI causes M2	–	–	–	Positive
IPI	M2	0.780	M2 doesn't cause IPI	–	–	–	–

Note: The Impulse-Response column shows the response of the dependent variable and it is given when there is a causal relationship.

8. Conclusion

The EMH holds a pivotal position in post-Keynesian economics. This article makes a dual contribution to the existing literature. Firstly, our primary objective is to assess the validity of the EMH and ascertain which of the three main perspectives on the EMH aligns most closely with the Turkish dataset from 2008 to 2020. In the process, we provide empirical evidence supporting the EMH. Secondly, we emphasize the significance of establishing the validity of the EMH for Türkiye, since the prevalent view on government investment and fiscal discipline, particularly among Turkish scholars, heavily relies on exogenous money theories. Many scholars adhere to these theories, ignoring the implications of the EMH. This is critical because their policy recommendations regarding fiscal policy and the government's role are derived from economic models grounded in exogenous money theories. The predominant neoclassical argument put forth by these scholars in proposing fiscal policy for Türkiye revolves around the crowding-out effect. However, if money is viewed as an endogenous phenomenon, the crowing-out effect does not exist. We also provide theoretical reasons explaining this phenomenon. Therefore, this paper contributes to the critique of the fiscal consolidation literature in Türkiye.

In this paper, we formulate ten testable hypotheses by following the literature. Initially, we establish the first two hypotheses, seeking to determine the validity of the EMH. Subsequently, we evaluate horizontalism through the third, fourth, and fifth hypotheses. Following that, structuralism is scrutinized with the fifth, sixth, and seventh hypotheses. Lastly, we define the third, eight, ninth, and tenth hypotheses to assess the circuitist view on the EMH. This study aligns with the empirical literature by adopting an econometric methodology. Specifically, we employ Granger causality methodology to explore the causal relationships among the variables of interest. The empirical results indicate no long run relationship between the series proposed by first two hypotheses. Nevertheless, the first two hypotheses have not been rejected based on the short-term findings. For this reason, there exists empirical support for the validity of the EMH for Türkiye. In the empirical findings section, we assess the validity of three main perspectives. The results in this section reveal a perfect fit for circuitism in our sample, with partial evidence also emerging for horizontalism and structuralism. Notably, this partial evidence is derived from the short-term findings of the third hypothesis for horizontalism and the long-term finding of the seventh hypothesis for Structuralism.

Appendices

Appendix A

To illustrate the basic concepts, let us start with the following autoregressive distributed lag model that has an optimal lag of q. At this point, it is required to state that the VAR model is a system of equations rather than a single equation. In other words, if there are two variables, there are two equations with different dependent variables in the system of VAR. For simplicity's sake, let us focus on one equation, given below.

$$Y_t = \alpha + \beta_1 Y_{t-1} + \ldots + \beta_q Y_{t-q} + \delta_1 X_{t-1} + \ldots + \delta_q X_{t-q} + \epsilon_t \quad \text{(A1)}$$

We say X Granger causes Y if all $\delta_i \neq 0$. In other words, if the null hypothesis, which states that all $\delta_i = 0$ is rejected, X Granger causes Y.

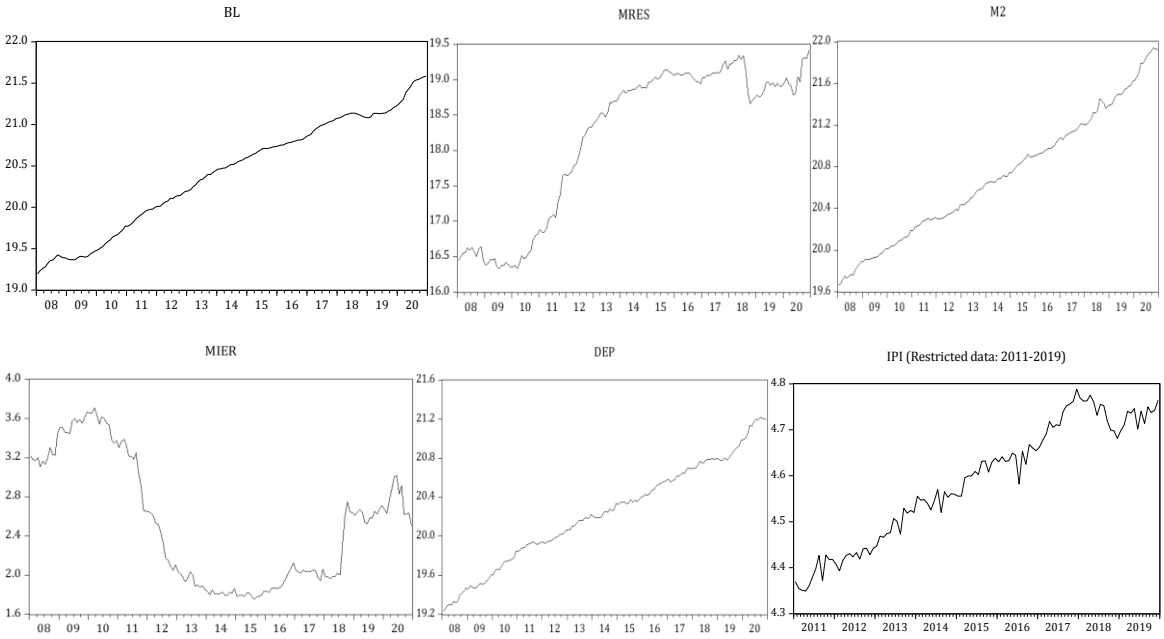
When there is a cointegration/long-run relation between Y and X, the error correction term is added to the model. Again, we assume that the optimal lag is q.

$$\Delta Y_t = \alpha + \lambda e_{t-1} + \beta_1 \Delta Y_{t-1} + \ldots + \beta_q \Delta Y_{t-q} + \delta_1 \Delta X_{t-1} + \ldots + \delta_q \Delta X_{t-q} + \epsilon_t \qquad \text{(A2)}$$

Since the variables are cointegrated, which means that they are non-stationary or first-difference stationary, but their combination is stationary, we use Δ to stand for the first difference of the variables. Error correction term is λe_{t-1}, where e_{t-1}=$Y_{t-1} - \alpha + \beta X_{t-1}$ are obtained from $Y_{t-1} = \beta X_{t-1} + e_{t-1}$. While for S.R. causality, the significance of the lag values of the X variable needs to be analyzed, like standard VAR model, for L.R. causality, the sign and significance of error correction term need to be analyzed. In other words, the coefficient of cointegrated equation (error correction term) needs to be statistically significant and negative in sign. When this condition holds, there is S.R. adjustment to L.R. equilibrium by the percentage of the coefficient of cointegrated equation because error correction term measures any movement away from long-run equilibrium. Also, it means that there is L.R. Granger causality running from the independent variable to dependent variable. It is possible to have evidence of long-run causality while there is no short-run causality and vice versa.

Appendix B

Figure A1 – *Trends of the variables*



Appendix C

Table A1 – *VAR estimates - equation A1 (dependent variable: D_BL)*

	Coefficient	Std. Error	t-Statistic	Prob.
C(1)	0.525376	0.077017	6.821595	0.0000
C(2)	–0.175603	0.055810	–3.146469	0.0020
C(3)	0.009374	0.001515	6.189145	0.0000

R–squared: 0.236351 Adjusted R–squared: 0.226237
Prob (F–statistic): 0.000000

S.R. Granger Causality (Wald Test):
H0: C(2)=0
Chi–square: 9.900264
Prob. 0.0017
There is S.R. Granger causality running from DEP to BL.

Diagnostic Tests:
*****Breusch–Godfrey Serial Correlation LM test: Prob.Chi–Square(2): 0.1761
*Heteroskedasticity Test: Breusch–Pagan–Godfrey:
Prob.Chi–Square(2): 0.1732

Notes: (*a*) C( ) stands for coefficient. (*b*) The lag level of the variables is given in the parentheses next to the variable names.
Equation: D_BL = C(1)*D_BL(-1)+C(2)*D_DEP(-1)+C(3)

Table A2 – *VAR Estimates - equation A2 (dependent variable: D_DEP)*

	Coefficient	Std. Error	t-Statistic	Prob.
C(4)	0.343739	0.118605	2.898191	0.0043
C(5)	–0.212789	0.085946	–2.475840	0.0144
C(6)	0.010056	0.002333	4.311266	0.0000

R–squared: 0.064757 Adjusted R–squared: 0.052369
Prob (F–statistic): 0.006380

S.R. Granger Causality (Wald Test):
H0: C(4)=0
Chi–square: 8.399511
Prob. 0.0038
There is S.R. Granger causality running from BL to DEP.

Diagnostic Tests:
*****Breusch–Godfrey Serial Correlation LM test: Prob.Chi–Square(2): 0.9773
*Heteroskedasticity Test: Breusch–Pagan–Godfrey:
Prob.Chi–Square(2): 0.0988

Notes: (*a*) C( ) stands for coefficient. (*b*) The lag level of the variables is given in the parentheses next to the variable names.
Equation: D_DEP = C(4)*D_BL(-1)+C(5)*D_DEP(-1)+C(6)

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