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FINANCIAL DEEPENING AND ECONOMIC GROWTH: A CASE OF TURKEY

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Abstract

In this study, the relationship between economic growth (EG) and financial deepening in Turkey was explored by using the yearly data belongs to the series of GDP, gross fixed capital formation (PC), secondary education (HC) and portfolio investment (FD) for the period between 1994 – 2017. VECM employed to reveal the short-term and the long-term causality. The results of VECM did not indicate a long-term relationship. The short –term analysis revealed that there is (a) a bidirectional causality between economic growth and financial deepening, (b) a unidirectional causality from physical capital to economic growth (c) a bidirectional causality between financial deepening and physical capital (d) a unidirectional causality from human capital to financial deepening. There is not a causality (e) between human capital and economic growth (f) between human capital and physical capital in the short-term. The results of the analyses in this research support the Mutual Interaction Hypothesis that asserts a bidirectional relationship between financial deepening and economic growth, which is hypothesised by Lewis and Patric. In this context, financial deepening has an important place in terms of sustainable economic growth.

Keywords: *Economic Growth, Financial Development, Var Granger Causality Analysis, Johansen Cointegration Analysis.*

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FİNANSAL DERİNLEŞME VE EKONOMİK BÜYÜME: TÜRKİYE ÖRNEĞİ

Öz

Bu çalışmada, Türkiye'deki iktisadi büyüme ile finansal derinleşme arasındaki ilişkiler incelenmiştir. Veri seti, 1994 - 2017 yılları arasında gayrı safi yurtiçi hasıla (EG), gayrı safi sabit sermaye oluşumu (PC), orta öğretim mezunu sayısı (HC), ve portföy yatırımlarını (FD) kapsamaktadır. Seriler arasındaki kısa ve uzun dönemli ilişkilerin ortaya konmasında VECM kullanılmıştır. Buna göre seriler arasında uzun dönemli ilişki olmadığı sonucuna ulaşılmıştır. Kısa dönem sonuçlarına göre; (a) iktisadi büyüme ile finansal derinleşme arasında çift yönlü, (b) fiziki sermayeden finansal derinleşmeye doğru tek yönlü, (c) fiziki sermaye ile finansal derinleşe arasında iki yönlü, (d) beşeri sermayeden finansal derinleşmeye doğru tek yönlü ilişki olduğu sonucuna varılmıştır. Ancak, (e) beşeri sermaye ile fiziki sermaye arasında ve (f) beşeri sermaye ile iktisadi büyüme arasında kısa dönemli bir nedensellik ilişkisi olmadığı görülmüştür. Bu araştırmadaki analizlerin sonuçları, Lewis ve Patric tarafından öne sürülen finansal derinleşme ile iktisadi büyüme arasında iki yönlü ilişki olduğunu oraya koyan "Karşılıklı Etkileşim Hipotezini" desteklemektedir. Bu bağlamda, finansal derinleşmenin sürdürülebilir iktisadi büyüme açısından önemli bir yeri olduğu söylenebilir.

Anahtar kelimeler: İktisadi Büyüme, Finansal Gelişme, VAR Granger Nedensellik Analizi, Johansen Eşbütünleşme Analizi

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1. Introduction

In general, financial deepening encompasses all of the fiscal policies and arrangements made to stimulate domestic and foreign savings in the financial field. The increase in the efficiency of the financial intermediation sector is the main factor determining financial deepening. The financial intermediary acts as a bridge between the fund suppliers and the demanders and ensures the flow of these funds between the parties (Oktayer, 2007: 6-7).

Financial deepening (FD) creates a number of economic effects. Many researchers question whether FD has an impact on real GDP or how the interaction between these two variables is relevant. The link between FD and real GDP growth is presented in four different approaches in the theoretical and empirical literature.

The first two of these approaches are the "demand-following" and "supply-leading hypothesis" developed by Patrick (1966). "Demand-following hypothesis" suggests that demand for financial services depends on the increase in real output in the economy. Increasing the real national income level in the economy enables the financial system to operate by increasing the demand for financial intermediaries and services in order to meet the capital needed by investors and savings holders. Therefore, the "demand following hypothesis" argues that the growth in the demand for financial system deepening and sustainability will only be achieved through economic growth (EG), meaning that EG is a cause of FD. "Supply-leading hypothesis" arguing that FD is a driving force for EG; states that the financial system is extremely important for EG to convert savings into expenditure and inject them into the economy in countries with developed, reliable and stable financial markets. "Supply-leading Approach" emphasizes that the development of financial institutions, especially modern sectors, which are the driving force of growth, will increase the demand for financial services. These intermediary activities carried out in the financial system and the funds collected in return for deposits and other financial liabilities are transferred to the modern sectors from the traditional sectors in the form of loans and contributed to growth.

The "No Impact" opinion suggests that there is no relation between EG and FD. Lucas (1988) ve Stern (1989)'s studies support the view that there is no correlation between FD and EG. Finally, The "Mutual Interaction" viewpoints out that there is a mutual interaction between FD and EG (Patrick, 1966: 175-176; Manga et al., 2016: 814-815). Lewis (1955) argues that financial markets have developed as a result of EG, and then financial markets undertake a function to stimulate real EG.

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Markets and institutions that perform financial services lead to lower costs and increase efficiency in the financial sector. FD contributes to EG by encouraging savings and ensuring savings are directed to productive areas through efficient resource allocation. In addition, FD positively affects the long-term economic performance of developing countries as a result of human capital and developments in new production technologies (Yapraklı, 2007: 69-70).

The increase in the number of financial instruments and institutions contributes to the expansion of financial markets and financial depth. FD contributes to employment and growth by increasing the savings volume, capital accumulation and investment opportunities by leading to the development of money and capital markets. The diversity of financial innovations and the quality and functionality of financial services ensure that the funds created in the market are effectively transferred to the real sector. Investors' ability to create liquidity and directing their savings to efficient investment areas can be stated to strengthen the country's economy by reducing market risks. Accurate and systematic management of FD in countries where financial development takes place is important in maximizing national welfare.

2. Theoretical Framework

FD and EG can mutually affect one another. This interaction emerges as follows: Growth encourages FD by increasing demand for financial services; financial development encourages EG with its intermediary role in the provision of resources needed by the real sector.

While traditional growth theories, focusing on innovations in the real sector, suggest that the main factor determining the long-term growth rate is external technological change; contemporary growth theories emphasize the functions of financial intermediaries. Contemporary approaches suggest that financial markets and institutions that have emerged internally in order to reduce failures caused by information and transaction costs in the economy mobilize savings, divide the risk, ensure investments get funded and making efficient investment decisions by evaluating potential entrepreneurs, thus contributing to EG (Bozoklu and Yılancı, 2013: 163; Türedi and Berber, 2010: 302).

Solow's "Neoclassical Growth Model" suggests that sustainable growth in GDP per capita is technically the result of an exogenous-induced change. The Endogenous Growth Model, pioneered by Paul Romer and Robert Lucas, states that endogenous variables are the main determinant of the increase in output. According

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to this theory, endogenous factors such as knowledge and human capital continuously increase per capita income (Özcan and Arı, 2011: 123).

Greenwood and Jovanovic (1990), Levine (1991) and Saint-Paul (1992), in the light of similar theoretical models they have established; suggest that the effective functioning of the financial markets positively affects the EG by increasing the quality of the investments. Greenwood and Jovanovic (1990) state that EG will stimulate investment and that increased investment will accelerate growth. Financial intermediaries undertake an important function in this process. They ensure the most profitable use of funds and allow investors to achieve higher and reliable returns by providing the investors with the information they have collected and analysed. The increase in the return on capital investments increases the level of national income and EG (Bozoklu and Yılancı, 2013: 169).

Pagano (1993), arguing that FD would stimulate growth through productivity; states that the development in financial institutions increases the efficiency of investments by channelling the capital to projects with the highest marginal efficiency and thus contributes to growth. Lucas (1988) found that the impact of financial institutions on EG was very limited; it states that FD basically boosts EG through capital accumulation, total factor productivity growth and human capital accumulation channels. Roubini and Sala-i Martin (1992) say that the financial sector's repressive policies will slow down the growth rate by reducing the savings volume and reducing the efficiency of capital.

Levine (1991) states that the capital markets ensure good management of liquidity and productivity risk and protect individuals against company-specific productivity shocks and enables them to invest in sufficient amount of various companies. This positively affects the EG by increasing the human capital and technology used in the production process. Saint-Paul (1992) explains the impact of FD on EG with emphasis on the complementarity between financial markets and technology. Accordingly, when inefficient but flexible technologies in undeveloped financial markets are received, producers will not face high risk and thus there will be no pressure to develop financial markets. On the other hand, having a more risky and specialized technology in developed financial markets has a positive effect on productivity. In this context, it can be said that financial markets contribute to EG by increasing marginal productivity of labour and economies with developed financial markets reach higher growth rates than countries with undeveloped financial markets (Bozoklu and Yılancı, 2013: 169-171).

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According to La Porta et al. (1997), FD and its impact on EG depend on the institutional and legal structure of the countries. $Y = f(K, H)$ is a single-sector Cobb-Douglas production function where K and H are considered as production factors and the conditions of constant returns to scale are valid. In this model; K refers to physical capital and H is human (social) capital, and the relation between human (social) capital and growth is explained (Barro and Martin, 2004: 211). Barro (1991) type growth model states that basically there is a direct relationship between the share of government expenditures in national income and GDP per capita. Research and development activities will be able to increase EG provided that state-controlled activities such as technology transfer and innovation are subject to low private returns. Private sector investments increase the capital stock and output level. The legal institutional structure of the financial system is naturally affected by these variables, each of which represents an input to the production function. That is, government expenditures including such investments as social service, education, health, infrastructure services, etc. increase the growth potential of the economy. In this context, within the framework of the Barro-type growth model, we can say that the financial sector under state supervision promotes EG through the channels mentioned above (Sağlam and Sönmez, 2017: 122).

In fact, rapid growth in an economy is a result of the increased investments, the acceleration of FD and the increase in saving rates. FD also plays a significant role in EG by increasing the efficiency of investments. In countries with rapid growth rate, financial systems are adequately deep and the efficiency of investments is quite high (Bozoklu and Yılancı, 2013: 163). Joseph Schumpeter (1912) emphasizes the importance of a developing financial system and institutions in EG. It also states that a well-functioning banking system will support entrepreneurs who produce innovative products and technologies and thus accelerate EG with high value-added investments (Levine, 1997: 688). According to Schumpeter (2012), technological innovations are the engine of EG. These innovations are carried out by making technology-oriented investments and researches. Therefore, the elements that encourage technological innovation also accelerate EG (King and Levine, 1993a: 717). King and Levine (1993b) explain the relationship between FD and EG within the framework of endogenous growth model by focusing on the impact of FD on productivity rather than the formation of physical capital and the intermediation effect of financial markets between savings and investments. In the model developed by the authors, technological innovation is determined endogenously and the critical role of the financial sector is emphasized. This approach, which is in line with Schumpeter, concludes that the impact of FD on EG

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is positive. In other words, the mechanism implies that there is a unidirectional causality from FD to EG (Çeştepe and Yıldırım, 2016: 14).

To summarize, FD affects real GDP through many different channels. At this point, financial institutions and markets play a big role. Financial intermediaries contribute to the growth by effectively transferring funds in the market between fund suppliers and demanders, and by increasing productivity in investments. It must not be ignored the importance of government expenditures, capital accumulation and the labour force with high marginal returns in EG. FD also helps to reduce inequality in income distribution within the country by stimulating savings and by ensuring efficiency in resource allocation, thus leading to EG.

3. Literature Review

When the results obtained in the studies conducted to explain the causality relationship between FD and EG, it is seen that there is no common opinion. It can be argued that the reason for this is that the difference between the methods and data set used and the periods considered in each study is effective. The results of various empirical studies on this subject reveal the existence of bidirectional relations between FD and EG. But however, in some studies, there is evidence that the relationship between FD and EG is weak or there is no association between these variables. As can be seen from different studies, although there is no consensus on the direction of causality between the FD and EG, it has been concluded that mostly FD has a positive effect on EG.

King and Levine (1993a) use a sample of 80 countries for the period of 1960-1989. They propose that positive developments in the financial system will create alternative channels to reduce the risk by encouraging capital utilization coefficients and innovative activities, thus positively affecting EG.

Levine and Zervos (1998), using time series in 47 countries involving the period of 1976-1993, find that the developments in the banking sector and stock market increase long-term growth through capital accumulation and efficiency.

Kar and Pentecost (2000) investigate this relation for Turkey by Johansen Cointegration and Vector Error Correction Model. Authors use six different indicators for FD and state that the relationship between the series change according to the preferred variable for financial development. They find that there is a causal relation from FD to EG when M2Y/GNP series is used as an FD indicator, and from EG to FD for the series of bank deposits/GNP, private sector loans/GNP and domestic credit ratio/Total Local Loans, total local loans/GNP.

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Calderón and Liu (2003), using panel data analysis for 109 developed and developing countries for the period 1960 to 1994, find the evidence that the causality between the variables varies between countries, but generally, the direction of causality is from FD to EG.

Shan (2005) has used the Variance Decomposition and Impulse-Response Analysis for China and 10 OECD countries, and reports that the causality relationship between FD and EG is bidirectional in the economy of Denmark, the United States, the UK, Australia and Japan; unidirectional from EG to FD in South Korea, Italy, Canada and China; and unidirectional from FD to EG in Finland and Portugal.

Yılmaz and Kaya (2006) apply Johansen Cointegration and Granger Causality Tests for Turkey within the period of 1986-2004. Their research results show that there is the existence of unidirectional causality from EG to FD in the short run, but no relation between variables in the long run.

Abu-Bader and Abu-Qarn (2006) probe the association in 6 countries of Middle East and North Africa (Israel, Morocco, Algeria, Egypt, Tunisia, Syria) using Vector Error Correction Model, Johansen Cointegration and Granger Causality Test and put forward that in Israel, the direction of causality between two variables is from EG to FD (demand-following hypothesis) and that in other countries outside Israel, from FD to EG (supply-leading hypothesis).

Artan (2007) uses Panel Data Analysis to make a study of 79 Low, Middle and High-Income countries over the period 1980-2002. He asserts that FD affects EG positively in middle and high-income countries, but negatively in low-income countries. The results of the study show that a 10% increase in the level of FD reduces the GDP by approximately 0.5% in low-income countries, but increase the GDP 0.6% and 0.5% in middle and high-income countries respectively.

Oktayer (2007), by using Stock Exchange Capitalization Rate/GNP, M2Y/GNP, Government Debt Securities/Nominal GDP, Domestic Credit to Private Sector/Domestic Total Credit Volume and real GDP per capita with prices of 1987 in her study for Turkey, has not found any long-term relationship between FD and real GDP per capita.

Coşkun et al. (2009) examine the relation between FD and EG for Turkey within the frame of Johansen Cointegration and Granger Causality Analysis. Their empirical results deduce the existence of a unidirectional relationship between

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variables in the long run, but positive unidirectional relationship from EG to the banking sector in the short run.

Türedi and Berber (2010), in their study for Turkey, submit that there is a unidirectional causality between two variables. It is concluded in the study that the direction of the link is from FD to EG.

Güneş (2013) applies the Bound Test Approach to examine the FD-EG nexus for Turkey. She cannot find any evidence that FD causes EG. On the other hand, in the same period in Turkey, it is suggested that EG increases the share of financial market employees in the total labour force, but doesn't affect the ratio of the money supply to gross national product (M2Y/GNP). Moreover, the increase in financial employment and money supply (M2Y) is not the cause of EG.

Dudian and Popa (2013) investigate the case of 8 Central and Eastern European countries (excepting Slovenia and Slovakia) by panel data analysis and find that poor credits and spread of interest rates have a negative impact on EG. On the other hand, in contrast to the increase in private domestic credit share, an increase in the private sector credit growth rate and money supply (M2) have a positive effect on GDP.

Petkovski and Kjosevski (2014) study the effect of FD on EG in a sample of 16 Central and South-eastern European countries with GMM method and indicate that the difference between private sector credits and interest rates affects EG negatively, but the impact of near money on EG is positive.

Obradović and Grbić (2015), putting to use the Toda-Yamamoto Causality Test for Serbia, state that there is a unidirectional causality from the share of private entrepreneurs and household credits in GDP to EG. In addition, the authors state that there is a bidirectional causality between the share of the non-financial private sector in total domestic credits and EG.

Samargandia et al. (2015), for a panel of 52 middle-income countries, find no statistically significant relationship between FD and EG in the short run, but in the long run, they observe an inverted-U relationship between two variables.

Ak et al. (2016) review the connection between FD and EG in Turkey for the period 1989-2011. They show that in the post-1989 period, there is a unidirectional causality relationship that mainly proceeds from EG to FD. In other words, a demand-following causality between FD and EG exists.

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Çeştepe and Yıldırım (2016) apply Toda-Yamamoto and Granger Causality Test to Turkey and provide evidence of a bidirectional causality between FD and EG both in the short and long run.

Luintel et al. (2016) use Bayesian Method to examine 69 countries from 1989 to 2011. They find a strong relationship between FD and EG in high-income countries.

Ono (2017) uses VAR Toda-Yamamoto Causality Test to view Russia for 1999-2016 time period and determines that there is a causal link between two variables, from GDP to the money supply and banking sector.

Sönmez and Sağlam (2018) investigate the relationship in 10 European Transition Economies over the period 2001-2014. It is observed the existence of a unidirectional causality from FD to EG in the period they consider for selected countries.

Altıner and Bozkurt (2018) examine the N11 (Next Eleven) countries' FD and EG causal relation from 1980 to 2016. They suggest that there is a unidirectional causality relationship between finance and growth. In the study, the direction of causality is determined from EG to FD. In other words, their empirical evidence provides support for the demand-following relation between variables.

Ouyang and Li (2018) employ GMM Panel VAR Method on the correlation between FD and EG for 30 Chinese provinces covering the period of 1996-2015. The results of their study reveal that FD has a negative impact on EG.

Asteriou and Spanos (2018) analyzed the effects of FD on EG for a sample of 26 EU countries with panel data. Authors suggest that while before the crisis FD encourages growth, after the crisis, it adversely affects economic activity.

4. Econometric Analysis

4.1. Variables, Data Set, Model and Methodology

The dataset covers 24 observations between 1994-2017 that belong to the series of economic growth (EG), gross fixed capital formation (PC), secondary education (HC) and portfolio investment (FD)

The functional form of the model, which is defined economic growth (EG) as a predicated variable, and physical capital, human capital and financial deepening as predictor variables, can be described as in Eq.(1)

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$$Economic\ \ Growth = f\ (Physical\ Capital, Human\ Capital, Financial\ Deepining\ ) \quad (1)$$

$$EG\ = f(PC, HC, FD)$$

EG	: Economic growth (GDP constant 2010 US$)
PC	: Gross fixed capital formation (current US$)
HC	: Secondary education, general pupils
FD	: Portfolio investment, bonds (PPG + PNG) (NFL, current US$)

To employ into the empirical analysis, the functional model seen in Eq. (1) is expressed statistically as in Eq. (2).

$$EG_t = a + \beta_1 PC_t + \beta_2 HC_t + \beta_3 FD_t + \ u_t \quad (2)$$

$\beta_1, \beta_2\ and\ \beta_3$ are the coefficients of the regression that reflect the change in EG corresponding with per unit change in PC, HC and FD respectively. t represents a time trend. "a" symbolized the constant, while u_t represents "the error term that explain the deviations from the trend in each year."

So as to convert the "static model" in Eq. (2) to "dynamic model", the lagged values of the series are taken and can be described in VAR System as in Eq.(3), Eq.(4), Eq.(5) and Eq.(6) below.

$$dEG_t = a_{11} + \sum_{i=1}^{n} \beta_{1i} dEG_{t-i} + \sum_{i=1}^{n} \beta_{2i} dPC_{t-i} + \sum_{i=1}^{n} \beta_{3i} dHC_{t-i} + \sum_{i=1}^{n} \beta_{4i} dFD_{t-i} + u_{1t} \quad (3)$$

$$dPC_t = a_{21} + \sum_{i=1}^{n} \beta_{5i} dPC_{t-i} + \sum_{i=1}^{n} \beta_{6i} dEG_{t-i} + \sum_{i=1}^{n} \beta_{7i} dHC_{t-i} + \sum_{i=1}^{n} \beta_{8i} dFD_{t-i} + u_{2t} \quad (4)$$

$$dHC_t = a_{31} + \sum_{i=1}^{n} \beta_{9i} dHC_{t-i} + \sum_{i=1}^{n} \beta_{10i} dPC_{t-i} + \sum_{i=1}^{n} \beta_{11i} dEG_{t-i} + \sum_{i=1}^{n} \beta_{12i} dFD_{t-i} + u_{3t} \quad (5)$$

$$dFD_t = a_{41} + \sum_{i=1}^{n} \beta_{13i} dFD_{t-i} + \sum_{i=1}^{n} \beta_{14i} dPC_{t-i} + \sum_{i=1}^{n} \beta_{15i} dHC_{t-i} + \quad (6)$$

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$\sum_{i=1}^{n} \beta_{16i} dEG_{t-i} + u_{4t}$

Where d represents "the first difference", u_1, u_2, u_3 and u_4 show the "error terms" and "n is the number of lag-lengths".

Under the next subjects, primarily the stationary of the series will be tested and the proper lag-lengths will be defined. Cointegration analysis and VECM analysis will be performed to reveal the long-term and short-term causalities.

4.2. Findings

4.2.2. Stationary of the Series

The degree of the integration of the series is a critical issue for causality analysis. Therefore, stationary of the series was examined with "Augmented Dickey-Fuller Unit Root Test (ADF-Test)" and the outcomes were reported in Table 1.

Table 1. ADF Test Outcomes

	I(0)				I(1)			
	Intercept		Trend & Intercept		Intercept		Trend & Intercept	
	t-Stat.	Prob.(1)	t-Stat	Prob.(1)	t-Stat	Prob.(1)	t-Stat	Prob.(1)
EG	2.24610	0.9999	-0.34976	0.9834	-3.19801**	0.0338	-4.06111**	0.0217
PC	-0.57636	0.8577	-2.16892	0.4833	-4.55670*	0.0017	-4.44004*	0.0100
HC	-0.46701	0.8809	-2.36729	0.3851	-5.05798*	0.0006	-4.94729*	0.0035
FD	-2.42340	0.1466	-3.46791	0.0670	-4.33651*	0.0041	-7.57475*	0.0001

"Note: * and ** show that coefficients are statistically significant at the 1% and 5% level of significance".
"(1) MacKinnon (1996) one-sided p-values"

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Table 1 includes the probability values and ADF t-statistics of the series at the level and at the first difference. All the series are non-stationary at the level because of the probability values of trend and trend & intercept are higher than 0.05. However, the outcomes of the first differences of the series show that all the series are stationary at the first difference so are I(1). Therefore, the VAR system will be established by taking the first differences of the series.

Defining the proper lag-length is also critical to producing the correct result. For this purpose, "VAR Lag Order Selection Criteria Test was used and the results are as in Table 2.

Table 2: VAR Lag Order Selection Criteria

Lag	LogL	LR	FPE	AIC	SC	HQ
0	-1506.131	NA	6.97e+61	150.9131	151.0625	150.9423
1	-1442.455	101.8824*	2.99e+59*	145.4455*	146.0429*	145.5621*
2	-1435.447	9.110669	3.94e+59	145.6447	146.6902	145.8488
3	-1427.419	8.027492	5.32e+59	145.7419	147.2355	146.0335
4	-1420.805	4.630040	1.07e+60	145.9805	147.9222	146.3595

Note: "* indicates lag order selected by the criterion LR: sequentially modified LR test statistic (each test at 5% level); FPE: Final prediction error; AIC: Akaike information criterion; SC: Schwarz information criterion; HQ: Hannan-Quinn information criterion."

In Table 2, the endogenous variables are EG, PC, HC, FG and the appropriate lag-length was specified as 1 by LR, FPE, AIC, SC and HQ method.

The integration level of the series I (1) suggests that there may be a cointegration between the series and therefore a long-term relationship. Therefore, the existence of a long-term relationship between the series will be examined by cointegration analysis.

4.2.3. Cointegration Analysis

The cointegration between the series will be investigated with the help of "Johansen Cointegration". "No cointegration the null hypothesis" is tested in return for the alternative hypothesis that represents "a cointegration relationship between variables" was tested. So as to specify the existence and the number of vectors of cointegration, the outcomes of the required "trace statistics" and "maximum Eigen statistics" values are displayed in Table 3.

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Table 3. Johansen Co-integration Analysis

Series: EG PC HC FD/		Trace Test		
Hypothesized No. of CE(s)	Eigenvalue	Trace Statistic	0.05 Critical Value	Prob.**
None *	0.791770	56.55451	47.85613	0.0062
At most 1	0.424160	22.03404	29.79707	0.2966
At most 2	0.362115	9.891669	15.49471	0.2892
At most 3	2.45E-05	0.000539	3.841466	0.9834

"Trace test indicates 1 cointegrating eqn(s) at the 0.05 level"

Maximum Eigenvalue Test

Hypothesized No. of CE(s)	Eigenvalue	Max-Eigen Statistic	0.05 Critical Value	Prob.**
None *	0.791770	34.52047	27.58434	0.0055
At most 1	0.424160	12.14237	21.13162	0.5337
At most 2	0.362115	9.891130	14.26460	0.2192
At most 3	2.45E-05	0.000539	3.841466	0.9834

"Max-eigenvalue test indicates 1 cointegrating eqn(s) at the 0.05 level. * denotes rejection of the hypothesis at the 0.05 level. **MacKinnon-Haug-Michelis (1999) p-values"

In Table 3, trace statistics and max-Eigen Statistics' values are greater than their critical value at the 0.05 level for None. As a result, the "trace unrestricted cointegration rank test indicates 1 cointegrating equation at the 0.05 level". Similarly, "max-eigenvalue test indicates 1 cointegrating equation at the 0.05 level".

Based on both results of "trace" and "Maximum Eigenvalue", there is "a co-integrated vector between the variables" and therefore there is a long-term relationship between PC, HC, FD and EG.

Because of Johansen Cointegration analysis predicted a long-term analysis, VECM analysis was performed to conform it and produce more detail regarding the short and the long-term causality.

Model-I, which is seen in Eq.7, shows the relationship between EG, which is

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the predicated variable, and HC, PC and FD, which are the predictors.

$$D(EG) = C(1)*(EG(-1) - 2.88126497451*PC(-1) - 63939.5742426*HC(-1) + 95.136915388*FD(-1) - 317172961189) + C(2)*D(EG(-1)) + C(3)*D(PC(-1)) + C(4)*D(HC(-1)) + C(5)*D(FD(-1)) + C(6) \quad (7)$$

The outcomes of the VECM of Model-I are presented in Table 4.

Table 4. VECM Outcomes (Model-I)

	Coefficient	Std. Error	t-Statistic	Prob.
C(1)	-0.036647	0.037093	-0.987978	0.3379
C(2)	0.998257**	0.398420	2.505539	0.0234
C(3)	-1.087262**	0.459253	-2.367456	0.0309
C(4)	8251.338	13453.85	0.613307	0.5483
C(5)	6.056894**	2.578547	2.348957	0.0320
C(6)	1.05E+10	1.27E+10	0.823542	0.4223

As it is seen in Table 4, the value of C (1) the error correction term is negative; however, the p-value is higher than 0.05 and statistically insignificant. Therefore, there is no long-term causality from PC, HC, FD to EG. The coefficients from C(2) to C(5) show the short-term relationships. To investigate the causality in the short-term, Wald Test was employed and the outcomes presented in Table 5.

Table 5. Wald Test Outcomes (Model-I)

H_0: C(4)=0				H_0: C(5)=0			
Statistics	Value	df	Prob.	Statistics	Value	df	Prob.
t-stat.	0.613307	16	0.5483	t-stat.	2.348957	16	0.0320**
F-stat.	0.376145	(1, 16)	0.5483	F-stat.	5.517597	(1, 16)	0.0320**
Chi-sqr.	0.376145	1	0.5397	Chi-sqr.	5.517597	1	0.0188**

H_0: C(3)=0				**Results:**		
Statistics	Value	df	Prob.	H_0: C(3)=0	**PC⇏EG**	**Rejected**
t-stat.	-2.367456	16	0.0309*	H_0: C(4)=0	**HC⇏EG**	**Confirmed**
F-stat.	5.604849	(1, 16)	0.0309*	H_0: C(5)=0	**FD⇏EG**	**Rejected**
Chi-sqr.	5.604849	1	0.0179*			

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H_0: C(3)=0 means that there is no short-term causality form PC to EG. The prob. value of $\chi 2$ is 0.0179 and lower than 0.05 the significance level. Therefore, "H0: C(3)=0 is rejected" and there is a causality from PC to EG.

H_0: C(4)=0 means that there is no short-term causality form HC to EG. The prob. value of $\chi 2$ is 0.5397 and higher than 0.05. Therefore, "H0: C(4)=0 is confirmed" that there is no causality from HC to EG.

H_0: C(5)=0 means that there is no short-term causality form FD to EG. The prob. value of $\chi 2$ is 0.0188 and lower than 0.05. Therefore, "H0: C(5)=0" is rejected that there is a causality from FD to EG.

To examine whether Model-I has any statistical error, the diagnostic tests consist of serial correlation, heteroscedasticity, and histogram normality was employed and the outcomes are summarized in Table 6.

Table 6. Diagnostic Tests Outcomes (Model-I)

Breusch-Godfrey Serial Correlation LM Test:

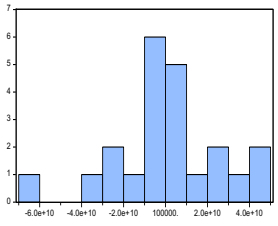
F-statistic	1.560513	Prob. F(1,15)	0.2307
Obs*R-squared	2.073081	Prob. Chi-Square(1)	0.1499

Heteroskedasticity Test: Breusch-Pagan-Godfrey

F-statistic	0.582387	Prob. F(8,13)	0.7757
Obs*R-squared	5.804380	Prob. Chi-Square(8)	0.6691
Scaled explained SS	4.438694	Prob. Chi-Square(8)	0.8155

Histogram Normality

Jarque-Bera	1.609681	Probability	0.447159



"Breusch-Pagan-Godfrey Test" was employed to test the heteroscedasticity. This test has a reverse hypothesis. If the F and $\chi 2$ probability values are greater than

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0.05, there is a multivariate variance that means there is not heteroscedasticity, but homoscedasticity, which refers to constant variance belongs to error terms. Table 6 shows the prob. values of F and $\chi 2$ are greater than 0.05, so there is no heteroscedasticity.

Likewise, a reverse hypothesis is used in the Jarque-Bera test. In other words, if the probability result is greater than 0.05, hence residuals are normally distributed. The outcomes of "Histogram Normality shows that the "prob. value of Jarque-Bera" is approximately 0,45 and is higher than 0.05. This result illustrates that "there is not a multicollinearity in the model".

Similarly, the probability values of F and $\chi 2$ of "Breusch-Godfrey Serial Correlation LM Test" are higher than 0.05 the significance level and consequently, there is no serial correlation.

As a result of diagnostic tests, it is concluded that Model-I is statistically significant.

Model II which is seen in Eq.8 shows the relationship between PC which is the predicated variable and HC, EG and FD which are the predictor variables.

$$D(PC) = C(1)*(PC(-1) - 0.347069779713*EG(-1) + 22191.4939473*HC(-1) - 33.0191482663*FD(-1) + 110081149771) + C(2)*D(PC(-1)) + C(3)*D(EG(-1)) + C(4)*D(HC(-1)) + C(5)*D(FD(-1)) + C(6) \quad (8)$$

Table 7: VECM Test Outcomes (Model-II)

	Coefficient	Std. Error	t-Statistic	Prob.
C(1)	0.158131	0.081837	1.932269	0.0712
C(2)	-0.589641	0.351664	-1.676717	0.1130
C(3)	0.517167	0.305082	1.695173	0.1094
C(4)	6017.172	10302.02	0.584077	0.5673
C(5)	6.180711	1.974471	3.130312	0.0065
C(6)	-3.77E+09	9.74E+09	-0.387287	0.7036

Table 7 shows that the value of C (1) the ec term is positive and the p-value is higher than 0.05 and so statistically insignificant. Thus, there is no long-term causality from EG, HC, FD to PC. The coefficients from C(2) to C(5) show the short-term relationships. To investigate the causality in the short-term, Wald Test

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was employed and the outcomes presented in Table 8.

Table 8. Wald Test Outcomes (Model-II)

H_0: C(3)=0				H_0: C(4)=0			
Statistics	Value	df	Prob.	Statistics	Value	df	Prob.
t-stat.	1.695173	16	0.1094	t-stat.	0.584077	16	0.5673
F-stat.	2.873612	(1, 16)	0.1094	F-stat.	0.341146	(1, 16)	0.5673
Chi-sqr.	2.873612	1	0.0900	Chi-sqr.	0.341146	1	0.5592

H_0: C(5)=0				**Results**		
Statistics	Value	df	Prob.	H_0: C(3)=0	**EG⇏PC**	**Confirmed**
t-stat.	3.130312*	16	0.0065	H_0: C(4)=0	**HC⇏PC**	**Confirmed**
F-stat.	9.798855*	(1, 16)	0.0065	H_0: C(5)=0	**FD⇏PC**	**Rejected**
Chi-sqr.	9.798855*	1	0.0017			

H_0: C(3)=0 means that there is no short-term causality form EG to PC. The prob. value of $\chi 2$ is 0.090 and higher than 0.05 the significance level. Therefore, "H0: C(3)=0 is confirmed" and there is no causality from EG to PC.

H_0: C(4)=0 means that there is no short-term causality form HC to PC. The prob. value of $\chi 2$ is 0.5592 and higher than 0.05. Therefore, "H0: C(4)=0 is confirmed" that there is no causality from HC to PC.

H_0: C(5)=0 means that there is no short-term causality form FD to PC. The prob. value of $\chi 2$ is 0.0017 and lower than 0.05. Therefore, "H0: C(5)=0" is rejected that there is a causality from FD to PC.

To examine whether Model-II has any statistical error, the diagnostic tests consist of serial correlation, heteroscedasticity, and histogram normality was conducted and the outcomes are summarized in Table 9.

Table 9. Diagnostic Tests Outcomes (Model-II)

Breusch-Godfrey Serial Correlation LM Test:

F-statistic	1.503053	Prob. F(1,15)	0.2391
Obs*R-squared	2.003700	Prob. Chi-Square(1)	0.1569

Heteroskedasticity Test: Breusch-Pagan-Godfrey

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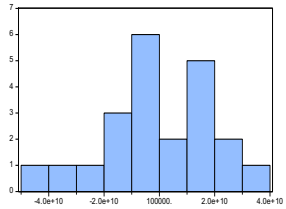
F-statistic	1.861763	Prob. F(8,13)	0.1534
Obs*R-squared	11.74694	Prob. Chi-Square(8)	0.1629
Scaled explained SS	5.620974	Prob. Chi-Square(8)	0.6896

Histogram Normality

Jarque-Bera	0.769391
Probability	0.680658



The prob. values of the F statistic and $\chi 2$ are higher than 0.05 and therefore "Breusch-Godfrey Serial Correlation LM Test" revealed that there is no serial correlation. The outcomes show that the prob. values of F statistic and $\chi 2$ of "Breusch-Pagan-Godfrey Heteroscedasticity test" are higher than 0.05, so there is no heteroscedasticity. The outcomes of "Histogram Normality show that the probability value of "Jarque-Bera" is approximately 0.68 and is higher than 0.05 and therefore, there is no a multicollinearity in the model. As a result, it is concluded that Model-II is statistically significant.

Model III which is seen in Eq.9 shows the relationship between HC which is the predicated variable and PC, EG and FD which are the predictors.

$$D(HC) = C(1)*(HC(-1) - 1.56397663238e\text{-}05*EG(-1) + 4.50623109185e\text{-}05*PC(-1) - 0.00148791912544*FD(-1) + 4960510.99723) + C(2)*D(HC(-1)) + C(3)*D(EG(-1)) + C(4)*D(PC(-1)) + C(5)*D(FD(-1)) + C(6) \quad (9)$$

Table 10. VECM Test Outcomes (Model-III)

	Coefficient	Std. Error	t-Statistic	Prob.
C(1)	-0.049375	0.040169	-1.229176	0.2368
C(2)	-0.238389	0.227866	-1.046183	0.3110
C(3)	-1.66E-06	7.78E-06	-0.213128	0.8339
C(4)	-2.97E-06	6.75E-06	-0.439894	0.6659
C(5)	3.64E-05	4.37E-05	0.833122	0.4170
C(6)	382150.5	215531.5	1.773061	0.0953

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As it is seen in Table 10, even though the value of C (1), which is the ec term, is negative, the p-value of C(1) is higher than 0.05 and statistically insignificant. Therefore, there is no long-term causality from EG, PC, FD to HC. The coefficients from C(2) to C(5) show the short-term relationships. To investigate the causality in the short-term, Wald Test was employed and the outcomes presented in Table 11.

Table 11. Wald Test Outcomes (Model-III)

H_0: C(3)=0				H_0: C(4)=0			
Statistics	Value	df	Prob.	Statistics	Value	df	Prob.
t-stat.	-0.439894	16	0.6659	t-stat.	-0.213128	16	0.8339
F-stat.	0.193506	(1, 16)	0.6659	F-stat.	0.045423	(1, 16)	0.8339
Chi-sqr.	0.193506	1	0.6600	Chi-sqr.	0.045423	1	0.8312

H_0: C(5)=0				**Results**		
Statistics	Value	df	Prob.	H_0: C(3)=0	**EG⇏HC**	**Confirmed**
t-stat.	0.833122	16	0.4170	H_0: C(4)=0	**PC⇏HC**	**Confirmed**
F-stat.	0.694092	(1, 16)	0.4170	H_0: C(5)=0	**FD⇏HC**	**Confirmed**
Chi-sqr.	0.694092	1	0.4048			

H_0: C(3)=0 means that there is no short-term causality form EG to HC. The prob. value of $\chi 2$ is 0.66 and higher than 0.05 the significance level. Therefore, "H0: C(3)=0 is confirmed" and there is no causality from EG to HC.

H_0: C(4)=0 means that there is no short-term causality form PC to HC. The prob. value of $\chi 2$ is 0.8312 and higher than 0.05. Therefore, "H0: C(4)=0 is confirmed" that there is no causality from PC to HC.

H_0: C(5)=0 means that there is no short-term causality form FD to HC. The prob. value of $\chi 2$ is 0.4048 and lower than 0.05. Therefore, "H0: C(5)=0" is confirmed that there is no causality from FD to HC.

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Table 12. Diagnostic Tests Outcomes (Model-III)

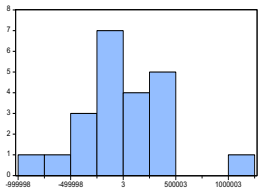
Heteroskedasticity Test: Breusch-Pagan-Godfrey

F-statistic	3.659735	Prob. F(8,13)	0.0188
Obs*R-squared	15.23523	Prob. Chi-Square(8)	0.0547
Scaled explained SS	15.65935	Prob. Chi-Square(8)	0.0475

Breusch-Godfrey Serial Correlation LM Test:

F-statistic	1.507892	Prob. F(1,15)	0.2384
Obs*R-squared	2.009562	Prob. Chi-Square(1)	0.1563

Histogram Normality



Jarque-Bera	4.468776	Probability	0.107058

The prob. values of $\chi 2$ are higher than 0.05 and therefore "Breusch-Godfrey Serial Correlation LM Test" implied that there is no serial correlation. "Breusch-Pagan-Godfrey Heteroscedasticity test" indicates that here is no heteroscedasticity due to the prob. value of $\chi 2$ belong to Obs*R2 > 0.05. The outcomes of "Histogram Normality show that the probability value of "Jarque-Bera" is approximately 0.11 and is higher than 0.05 and therefore, "there is no a multicollinearity in the model. As a result, it is concluded that Model-III is statistically significant.

Model IV which is seen in Eq.9 shows the relationship between FD which is the predicated variable and PC, EG and HC which are the predictors.

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$$D(FD) = C(1)*(FD(-1) - 672.079539071*HC(-1) + 0.0105111669421*EG(-1) - 0.0302854571516*PC(-1) - 3333857944.58) + C(2)*D(FD(-1)) + C(3)*D(HC(-1)) + C(4)*D(EG(-1)) + C(5)*D(PC(-1)) + C(6) \quad (9)$$

Table 13: VECM Outcomes (Model-IV)

	Coefficient	Std. Error	t-Statistic	Prob.
C(1)	-1.687331	0.346605	-4.868163	0.0002
C(2)	0.740594	0.253263	2.924210	0.0099
C(3)	3433.165	1321.426	2.598075	0.0194
C(4)	0.114498	0.039132	2.925896	0.0099
C(5)	-0.120499	0.045107	-2.671378	0.0167
C(6)	-3.05E+09	1.25E+09	-2.442437	0.0266

As it is seen in Table 13, the value of C (1), which is the ec term, is negative, the p-value of C(1) is lower than 0.05 and statistically significant. Hence, there is a long-term causality from EG, PC, HC to FD. The coefficients from C(2) to C(5) show the short-term relationships. To investigate the causality in the short-term, Wald Test was employed and the outcomes presented in Table 14.

Table 14: Wald Test Outcomes (Model-IV)

H_0: C(3)=0				H_0: C(4)=0			
Statistics	Value	df	Prob.	Statistics	Value	df	Prob.
t-stat.	2.598075	16	0.0194	t-stat.	2.925896	16	0.0099
F-stat.	6.749996**	(1, 16)	0.0194	F-stat.	8.560870*	(1, 16)	0.0099
Chi-sqr.	6.749996*	1	0.0094	Chi-sqr.	8.560870*	1	0.0034

H_0: C(5)=0				**Results**		
Statistics	Value	df	Prob.	H_0: C(3)=0	**HC⇏FD**	**Rejected**
t-stat.	-2.671378	16	0.0167	H_0: C(4)=0	**EG⇏FD**	**Rejected**
	7.136263*			H_0: C(5)=0	**PC⇏FD**	**Rejected**
F-stat.	*	(1, 16)	0.0167			
Chi-sqr.	7.136263*	1	0.0076			

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H_0: C(3)=0 means that there is no short-term causality form HC to FD. The prob. value of $\chi 2$ is 0.0094 and lower than 0.05, therefore, "H0: C(3)=0 is rejected" and there is a causality from HC to FD.

H_0: C(4)=0 means that there is no short-term causality form EG to FD. The prob. value of $\chi 2$ is 0.0034 and lower than 0.05. Therefore, "H0: C(4)=0 is rejected" that there is a causality from EG to FD.

H_0: C(5)=0 means that there is no short-term causality form PC to FD. The prob. value of $\chi 2$ is 0.0076 and lower than 0.05. Therefore, "H0: C(5)=0" is rejected that there is a causality from PC to FD.

Table 12. Diagnostic Tests Outcomes (Model-IV)

Breusch-Godfrey Serial Correlation LM Test:

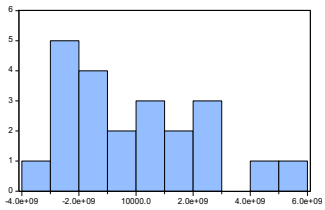
F-statistic	0.045833	Prob. F(1,15)	0.8334
Obs*R-squared	0.067017	Prob. Chi-Square(1)	0.7957

Heteroskedasticity Test: Breusch-Pagan-Godfrey

F-statistic	1.379509	Prob. F(8,13)	0.2910
Obs*R-squared	10.10122	Prob. Chi-Square(8)	0.2580
Scaled explained SS	4.127497	Prob. Chi-Square(8)	0.8454

Histogram Normality

Jarque-Bera	1.584200
Probability	0.452893



The prob. value of $\chi 2$ is 0.7957 and is higher than 0.05. Therefore, "Breusch-Godfrey Serial Correlation LM Test" indicates that there is no a serial correlation. "Breusch-Pagan-Godfrey Heteroscedasticity test" indicates that there is no heteroscedasticity due to the prob. value of $\chi 2$ belong to Obs*R2 > 0.05. The

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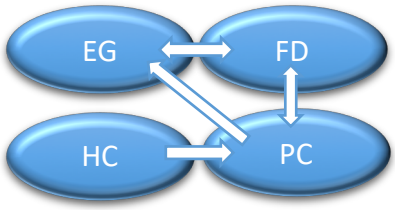
outcomes of "Histogram Normality show that the probability value of Jarque-Bera" is approximately 0.45 and is higher than 0.05. Therefore, "there is no a multicollinearity in the model. As a result, it is concluded that Model-IV is statistically significant.

The outcomes of the four models are presented in Table 13.

Table 13. Short-term Relationships of the Variables



Variable	The direction of The Causality	Variable
PC	⇒	EG
HC	⇏	EG
FD	⇔	EG
HC	⇏	PC
FD	⇔	PC
FD	⇐	HC

5. Conclusion

In this article, the long-term and short-term relationships between economic growth and financial deepening in Turkey were investigated. The dataset covers 24 observations between 1994-2017 that belong to the series GDP (constant 2010 US$), gross fixed capital formation (current US$), secondary education, general pupils, and portfolio investment, bonds (PPG + PNG) (NFL, current US$).

Primarily, functional, statistical and VAR models were established. Then, "Augmented Dickey-Fuller Unit Root Test" was employed to define the integration level of the series. It is observed that all the series are stationary at the first difference level, therefore the integration level of the series is I(1). To define the proper lag-length, "Lag Order Selection Criteria Test" was employed and it is indicated that the proper lag-length value is one.

To investigate whether a long-term relationship among the series, "Johansen Cointegration Test" was conducted. "Vector Error Correction Model (VECM)" employed to reveal both the short term and the long term causality. To discover the consistency of the cointegration models "Breusch-Godfrey Serial Correlation LM

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Test", "Breusch-Pagan-Godfrey Heteroscedasticity Test" and "Histogram Normality Test" were conducted.

The results of VECM did not indicate a long-term relationship. The short – term analysis of VECM revealed that there is "(a) a bidirectional causality between economic growth and financial deepening, (b) a unidirectional causality from physical capital to economic growth, (c) a bidirectional causality between financial deepening and physical capital (d) a unidirectional causality from human capital to financial deepening. There is not a causality (e) between human capital and economic growth, and (f) between human capital and physical capital in the short-term.

The results of the analyses in this research support the Mutual Interaction Hypothesis that asserts a bidirectional relationship between FD and EG, which is hypothesised by Lewis and Patric. In this context, financial deepening has an important place in terms of sustainable economic growth.

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