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A CAUSALITY TEST ON THE GIBSON PARADOX IN TURKEY

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ABSTRACT

In general, it is believed that interest rate is an important instrument for central banks while struggling with inflation. However, Gibson Paradox tells the story in a different way. So how does it work in reality? This study examines the validity of Gibson Paradox in Turkey empirically by using monthly data belonging 01:2003 - 05:2015 period. According to analysis results, there is an interaction between floating exchange rate and inflation targeting and also between monetary policy base interest rate (base rate) and consumer price index (CPI). On the account of these results we reach the conclusion that Gibson paradox is valid for Turkey.

Keywords: Interest rate, Consumer price index, Bootstrap causality, Frequency domain causality, Central banking, Monetary policy, Gibson paradox

JEL Classification: E43, E52, E58.

Contribution/ Originality

In literature there is not many works about the validity of Gibson Paradox for Turkey. Existing works have to be revisited and revised for current period. This paper analyzes the validity of Gibson paradox for Turkey for recent past with new econometric tests.

1. INTRODUCTION

Central banks of countries which have chronic inflation problem, will determine monetary policy tools and targets on the basis of information that there is an interaction between general price level and interest rates. As Van Der Merwe (2004) stated, changes in central banks' policies largely depend on price expectations of economic actors. The aim of this study is to research the

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relationship between consumer price index (CPI) and monetary policy base interest rate (base rate) for Turkey from 2003 till now. During this period Turkey has applied inflation targeting policy and has implemented floating exchange rate regime. If Gibson paradox is valid which means inflation has been the main reason of interest rate increase in Turkey, then Central Bank of Turkish Republic will develop alternative strategies in monetary policy implementation. The rest of paper is organized as follows. Theoretical framework and literature review is outlined in Section 2. The econometric methodology is outlined in Section 3. Then the data and the empirical findings are discussed in Section 4. Finally, some concluding remarks and policy implications are offered in Section 5.

2. THEORETICAL FRAMEWORK AND LITERATURE REVIEW

Classical Theory of Economics claims that long-term interest rate is determined by real economy variables like saving rate, productivity, which has effect on loanable funds market. Accordingly, general price level is assumed to be affected by only money supply. This assumption of Classical Theory is contradicted with the relationship between interest rate and general price level. Keynes named this contradiction as "Gibson Paradox" and he implied that this situation is one of the empirical realities in quantitative economics. The most important explanation for Gibson Paradox was made by Fisher (1930). According to Fisher Effect, raises in money supply results in inflation which causes increase in interest rate. In Fisher (1930) hypothesis, nominal interest rate i_t equals to sum of, real interest rate r_t and inflation expectations π_t^e.

$$i_t = r_t + \pi_t^e \qquad (1)$$

The reason of the positive relationship between general price level and nominal interest rate is that in long term real interest rate is not affected from monetary imbalances which have an effect on inflation rate.

Although there have been limited studies on Gibson Paradox, empirical literature has centered on testing Fisher Effect. Some supportive works about validity of Gibson Paradox are; Şimşek and Kadılar (2008) from 1987:Q1 to 2003:Q4 and Yamak and Tanriover (2007) for the period between 1990:Q1 and 2006:Q4. However, Halıcıoğlu (2004) found that Gibson Paradox is invalid between the years 1950 and 2002. In studies about Fisher Effect, Fisher Effect is generally occurred to be effective. For instance, Turgutlu (2004) found that Fisher effect is valid when consumer price index is taken as the basis, but invalid when wholesale price index is used for the period 1978Q4 to 2003Q4. Şimşek and Kadılar (2006) and Gül and Açikalin (2007) support the validity of Fisher Effect with their studies for similar periods. As a final, Yılancı (2009) arrived at the result that Fisher Effect is invalid during the period 1989Q1-2008Q1.

3. METHODOLOGY

3.1. Toda and Yamamoto (1995) Linear Granger Type Causality Test

Toda and Yamamoto (1995) applies VAR model due to number of the delay and also take into account the degree of integration of the series with χ2 distribution of the Wald test. Toda and Yamamoto (1995) causality analysis of the values β of the variables so that the level of the series by creating a standard VAR model eliminates the problems of determining the rank of cointegration (Zapata and Rambaldi, 1997; Duasa, 2007). The relationship between base rate (i) and inflation rate (cpi) VAR (p) process can be expressed as;

$$i = \phi_1 + \sum_{i=1}^{p+d_{\max}} \alpha_{1i} i_{ti} + \sum_{i=1}^{p+d_{\max}} \beta_{1i} cpi_{ti} + \varepsilon_{1t} \quad (2)$$

$$cpi = \phi_2 + \sum_{i=1}^{p+d_{\max}} \alpha_{2i} cpi_{ti} + \sum_{i=1}^{p+d_{\max}} \beta_{2i} i_{ti} + \varepsilon_{2t} \quad (3)$$

where $d_{\max}$ is the maximum degree of integration of the variables in the model, p is the optimal lag length obtained from the VAR model and ε_t is the term refers to the error correction based on the assumption of white noise. The null hypothesis is tested as $\beta_{1i} = 0$ for i≤k in equation 2. If the alternative hypothesis is accepted, it means that causal relationship running from inflation to base rate. The null hypothesis is tested as $\beta_{2i} = 0$ and i≤k in equation3 again and if the alternative hypothesis accepted, it means that there is a causality between variables running from base rate to inflation rate.

3.2. Hacker and Hatemi (2005; 2006) Bootstrap Process-Based Toda and Yamamoto (1995) Linear Granger Causality Test

Toda and Yamamoto (1995) causality test, applying a number of sampling is less, and if you have autoregressive conditional heteroscedasticity (ARCH) effect in error terms, based on the results of causality is wrong to make comments. Therefore, Hacker and Hatemi-J (2006) and also Hatemi-J (2005) developed a new methodology by using Efron (1979) bootstrap process based on the causality test. The vector autoregressive model of order p VAR(p) can be expressed as where y_t is the number of variables in the VAR model, v is an vector of intercepts and A_r is matrix of parameters for lag r (r=1,…,p) ;

$$y_t = v + A_1 y_{t-1} + A_2 y_{t-2} + ... + A_p y_{t-p} + \varepsilon_t \quad (4)$$

If the variables are cointegration equation 1 and 2 in the VAR ($p + d_{\max}$) model with a simple expression;

$$y_t = v + A_1 y_{t-1} + A_2 y_{t-2} + ... + A_p y_{t-p} + A_{p+d_{\max}} y_{t-p-d_{\max}} + \varepsilon_t \quad (5)$$

(Hacker and Hatemi-J, 2006; Hatemi-J and Roca, 2007).[1] The estimated VAR($p+d_{max}$) model in Equation 4 can be written compactly as: $Y = (y_1, ..., y_T)$,

$$\hat{D} = (\hat{v}, \hat{A}_1, ..., \hat{A}_p, ..., \hat{A}_{p+d_{\max}}), \hat{\delta} = (\hat{\varepsilon}_1, ..., \hat{\varepsilon}_T) \text{ and } Z_t = \begin{bmatrix} 1 \\ y_t \\ y_{t-1} \\ \vdots \\ y_{t-p-d+1} \end{bmatrix};$$

Can be written as,

$$Y = \hat{D}Z + \hat{\delta} \quad (6)$$

Null hypothesis that the there is no Granger causality (causality non Granger) Toda and Yamamoto (1995) developed by the modified Wald test (Modified WALD);

$$MWALD = (C\hat{\beta})'[C((Z'Z)^{-1} \otimes S_U)C']^{-1}(C\hat{\beta}) \square \chi_p^2 \quad (7)$$

Where $\otimes$ the Kronecker product and C is a $pxn(1+(p+d_{\max}))$ selector matrix, S_U is variance-covariance matrix of residuals and $\hat{\beta} = vec(D)$ is vec signifies the column-stacking operator. The error terms are normally and the MWALD test statistic is asymptotically χ^2 distributed (Hacker and Hatemi-J, 2006; Hatemi-J and Roca, 2007; Hatemi-J and Morgan, 2009). Hatemi-J (2005) Monte Carlo experiments testing the error terms in the normal zero smudge MWALD (nonnormality) and ARCH effect is rejected because of the null hypothesis leads to excessive. This is why Hatemi-J (2005); Efron (1979) developed by the leveraged bootstrap developed simulations. We generate the distribution for the MWALD test statistics by running the bootstrap simulation

[1] For choice of optimal lag order Hatemi-J (2003). They are developed new information criteria. For the details of Hatemi-J criterion can be read the study of Hacker and Hatemi-J (2006).

10.000 times and calculating the MWALD test statistics for each run. We then find bootstrap critical values pertaining to 1%, 5% and 10% significance levels. Afterwards, we calculate the MWALD statistics using original data. We reject the null hypothesis of no causality in the Granger sense at the α level of significance, if the actual MWALD is greater than. The Monte Carlo simulations are conducted using program procedure written by Hacker and Hatemi (2005; 2006)

3.3. Hatemi and Roca (2014) Aysmmetric Causality Test

P_{1t} and P_{2t} are assumed to be two cointegrated variables (Hatemi and Roca, 2014)

$$P_{1t} = P_{1t-1} + \varepsilon_{1t} = P_{1,0} + \sum_{i=1}^{t} \varepsilon_{1i} \quad (8)$$

Ve

$$P_{2t} = P_{2t-1} + \varepsilon_{2t} = P_{2,0} + \sum_{i=1}^{t} \varepsilon_{2i} \quad (9)$$

In these expressions t=1,2,…,T, $P_{1,0}$ and $P_{2,0}$ are constant terms and $\varepsilon_{1i}, \varepsilon_{2i} \sim iid(0, \delta^2)$. For all variables expressed, when positive and negative changes are defined as $\varepsilon_{1i}^{+} = \max(\varepsilon_{1i}, 0)$, $\varepsilon_{2i}^{+} = \max(\varepsilon_{2i}, 0)$, $\varepsilon_{1i}^{-} = \min(\varepsilon_{1i}, 0)$ and $\varepsilon_{2i}^{-} = \min(\varepsilon_{2i}, 0)$ $\varepsilon_{1i} = \varepsilon_{1i}^{+} + \varepsilon_{1i}^{-}$ ve $\varepsilon_{2i} = \varepsilon_{2i}^{+} + \varepsilon_{2i}^{-}$ is reached. Thus

$$P_{1t} = P_{1t-1} + \varepsilon_{1t} = P_{1,0} + \sum_{i=1}^{t} \varepsilon_{1i}^{+} + \sum_{i=1}^{t} \varepsilon_{1i}^{-} \quad (10)$$

$$P_{2t} = P_{2t-1} + \varepsilon_{2t} = P_{2,0} + \sum_{i=1}^{t} \varepsilon_{2i}^{+} + \sum_{i=1}^{t} \varepsilon_{2i}^{-} \quad (11)$$

Cumulative sum of positive and negative shocks in each variables is shown as $P_{1t}^{+} = \sum_{i=1}^{t} \varepsilon_{1t}^{+}$, $P_{1t}^{-} = \sum_{i=1}^{t} \varepsilon_{1t}^{-}$, $P_{2t}^{+} = \sum_{i=1}^{t} \varepsilon_{2t}^{+}$ and $P_{2t}^{-} = \sum_{i=1}^{t} \varepsilon_{2t}^{-}$ (Hatemi and Roca, 2014). Here when causality between positive shocks is tested, vector $P_t^{+} = (P_{1t}^{+}, P_{2t}^{+})$ is used. If this vector is written as k-th order VAR(L) then it is:

$$P_t^+ = v + A_1 P_{t-1}^+ + A_2 P_{t-2}^+ + ... + A_L P_{t-k}^+ + u_t^+ \quad (12)$$

In this expression v stands for the 2x1 constant terms vector , u_t^+ represents the 2x1 error terms vector including positive shocks where t=1,2…k and 2x2 parameter matrix is defined as A_r (Hatemi, 2012). Optimal lag length (k) is determined by test statistic developed by Hatemi-J (2003); Hatemi (2008):

$$HJC = In(\left|\hat{\Omega}_f\right|) + k2T^{-1}(m^2 InT + 2mIn(InT)) \quad (13)$$

If lag length, $\left|\hat{\Omega}_f\right|$, is k then error terms stand for variance-covariance matrix . m is the equation number in VAR model and T is the sample size (Hatemi and Roca, 2014). The null hypothesis of asymmetric causality test is the equality of j-th row and k-th column. If test statistic is greater than critical value, null hypothesis of no causality is rejected.

To determine Wald test statistic VAR(k) model is built as $Y = DZ + \delta$ where Y, D, Z and matrices such as $Y := (P_1^+, P_2^+, ..., P_T^+)$ mxT, $D := (v, A_1, A_2, ..., A_k)$ (mx(1+mk)),

$Z_t := \begin{bmatrix} 1 \\ P_t^+ \\ P_{t-1}^+ \\ \vdots \\ P_{t-p+1}^+ \end{bmatrix}$ ((1+mk)x1), $Z := (Z_0, Z_1, ..., Z_{T-1})$ ((1+mk)xT) and

$\delta := (u_1^+, u_2^+, ..., u_T^+)$ (mxT). In null hypothesis of no Granger causality $H_0 : R\beta = 0$ test statistic is calculated as;

$$Wald = (R\beta)'\left[R((Z'Z)^{-1} \otimes S_U)R'\right]^{-1}(R\beta)$$

In this expression $\beta = vec(D)$ and vec is column-stacking operator, $\otimes$ Kronecker multiplier, R is the kxm(1+mk) indicator matrix that includes restricted parameters defined as 1 and unrestricted parameters defined as 0. Unrestricted var-cov matrix of VAR model is

$S_U = \frac{\hat{\delta}_U' \hat{\delta}_U}{T-c}$. C stands for number of parameters in each equation inside the VAR model. Under normality assumption, Wald test statistic asymptotically shows kth degree χ^2 distribution. If test statistic is greater than all critical values, the null hypothesis of no causality is rejected.

3.4. Frequency Domain Causality Test

While conventional time domain causality tests produce a single test statistic for the interaction between variables in concern, frequency domain methodology generates tests statistics at different frequencies across spectra. Frequency domain approach to causality thereby permits to investigate causality dynamics at different frequencies rather than relying on a single statistics as is the case with the conventional time domain analysis (Ciner, 2011). Hence, it seems to be very meaningful to carry out frequency domain causality to better understand temporary and permanent linkages between policy rate and credit rates. To test for causality based on frequency domain, Geweke (1982) and Hosoya (1991) defined two-dimensional vector of time series $z_t = [x_t, y_t]'$ and z_t has a finite-order VAR;

$$\Theta(L) z_t = \varepsilon_t \tag{14}$$

Where $\Theta(L) = I - \Theta_1 L - \ldots - \Theta_p L_p$ and lag polynomial with $L^k z_t = z_{t-1}$. Then Granger causality at different frequencies is defined as;

$$M_{y \to x} = \log \left[\frac{2\pi f_x(\omega)}{\left| \psi_{11}(e^{-i\omega}) \right|^2} \right] = \left[1 + \frac{\left| \psi_{12}(e^{-i\omega}) \right|^2}{\left| \psi_{11}(e^{-i\omega}) \right|^2} \right] \tag{15}$$

If $\left| \psi_{12}(e^{-i\omega}) \right|^2 = 0$ that y does not cause x at frequency ω. If components of z_t are I(1) and co-integrated, then the autoregressive polynomial $\Theta(L)$ has a unit root. The remaining roots are outside the unit circle. Extracting z_{t-1} from both sides of equation 9 gives;

$$\Delta z_t = (\Theta_1 - I) z_{t-1} + \Theta_1 z_{t-2} + \ldots + \Theta_p z_{t-p} + \varepsilon_t = \hat{\Theta}(L) z_{t-1} + \varepsilon_t \tag{16}$$

Where $\hat{\Theta}(L) = \Theta_1 - I + \Theta_2 L + \ldots + \Theta_p L^p$ (Breitung and Bertrand, 2006). Geweke (1982) and Hosoya (1991) propose causality measure at a particular frequency based on a decomposition of the spectral density. Breitung and Bertrand (2006) who has using a bivariate vector

autoregressive model propose a simple test procedure that is based on a set of linear hypothesis on the autoregressive parameters. So that test procedure can be generalized to allow for cointegration relationships and higher-dimensional systems. Breitung and Bertrand (2006) assume that ε_t is white noise with $E(\varepsilon_t)=0$ and $E(\varepsilon_t,\varepsilon_t')=\Sigma$, where Σ is positive definite. Let G be the lower triangular matrix of the Cholesky decomposition $G'G=\Sigma^{-1}$ such that $E(\eta_t\eta_t')=I$ and $\eta_t=G\varepsilon_t$. If the system is stationary, let $\phi(L)=\Theta(L)^{-1}$ and $\psi(L)=\phi(L)G^{-1}$ the MA representation;

$$z_t=\phi(L)\varepsilon_t=\begin{pmatrix}\phi_{11}(L) & \phi_{12}(L)\\ \phi_{21}(L) & \phi_{22}(L)\end{pmatrix}\begin{pmatrix}\varepsilon_{1t}\\ \varepsilon_{2t}\end{pmatrix}=\begin{pmatrix}\psi_{11}(L) & \psi_{12}(L)\\ \psi_{21}(L) & \psi_{22}(L)\end{pmatrix}\begin{pmatrix}\eta_{1t}\\ \eta_{2t}\end{pmatrix} \quad (17)$$

Let we can use this representation for the spectral density of x_t;

$$f_x(\omega)=\frac{1}{2\pi}\{|\psi_{11}(e^{-i\omega})|^2+|\psi_{12}(e^{-i\omega})|^2\} \quad (18)$$

Breitung and Bertrand (2006) investigate the causal effect of $M_{y\to x}(\omega)=0$ if $|\psi_{12}(e^{-i\omega})|^2=0$. The null hypothesis is equivalent to a linear restriction on the VAR coefficients. $\psi(L)=\Theta(L)^{-1}G^{-1}$ and $\psi_{12}(L)=-\frac{g^{22}\Theta_{12}(L)}{|\Theta(L)|}$, with g^{22} as the lower diagonal element of G^{-1} and $|\Theta(L)|$ as the determinant of $\Theta(L)$, it follows y does not cause at frequency ω if

$$|\Theta_{12}(e^{-i\omega})|=\left|\sum_{k=1}^{p}\theta_{12,k}\cos(k\omega)-\sum_{k=1}^{p}\theta_{12,k}\sin(k\omega)i\right|=0 \quad (19)$$

with $\theta_{12,k}$ denoting the (1,2)-element of Θ_k. Thus for $|\Theta_{12}(e^{-i\omega})|=0$,

$$\sum_{k=1}^{p}\theta_{12,k}\cos(k\omega)=0 \quad \text{and} \quad \sum_{k=1}^{p}\theta_{12,k}\sin(k\omega)=0 \quad (20.\ 21.)$$

Breitung and Bertrand (2006) applied to linear restrictions (20) and (21) for $\alpha_j=\theta_{11,j}$ and $\beta_j=\theta_{12,j}$. Then the VAR equation for x_t can be implied as

$$x_t = \alpha_1 x_{t-1} + ... + \alpha_p x_{t-p} + \beta_1 y_{t-1} + ... + \beta_p y_{t-p} + \varepsilon_{1t} \quad (22)$$

and the null hypothesis $M_{y \to x}(\omega) = 0$ is equivalent to the linear restriction with

$$\beta = [\beta_1, ..., \beta_p]'$$

$$H_0: \quad R(\omega)\beta = 0 \text{ and } \quad R(\omega) = \begin{bmatrix} \cos(\omega) & \cos(2\omega) & ... & \cos(\mathrm{p}\omega) \\ \sin(\omega) & \sin(2\omega) & ... & \sin(\mathrm{p}\omega) \end{bmatrix} \quad (23.24.)$$

The causality measure for $\omega \in (0, \pi)$ can be tested with the conventional F-test for the linear restrictions imposed by Eq.(23) and Eq. (24). The test procedure follows an F- distribution with (2, T-2p) degrees of freedom.

4. DATA AND EMPIRICAL FINDINGS

When testing Gibson Paradox starting from 2003:M1 till 2015:M5, monetary policy base interest rate and consumer price index are the variables. Both variables are obtained from IFS. Taking natural log of both, they are seasonally adjusted by Tramo-Seats method.

Table-1. Descriptive Statistics

	Mean	Max.	Min.	Standard Deviations	Skewness	Kurtosis	Jarque-Bera
cpi	5.07	5.55	4.54	0.28	-0.07	1.82	8.737 (0.01)
i	2.81	3.89	0.41	0.79	-0.36	2.87	3.386 (0.183)

Note: Values in parenthesis show probabilities.

When coefficients of skewness and kurtosis are examined, both of them seem to be left-skewed and peaked. According to Jarque-Bera test results, CPI is not distributed normally but base rate shows normal distribution. To investigate the dynamic relations between variables, Vector Autoregression (VAR) model is used. For this reason, first of all (Dickey and Wayne, 1981) ve Phillips and Perron (1988) linear unit root tests are applied.

Table-2. Dickey and Wayne (1979;1981) and Phillips and Perron (1988) Unit Root Test Results for Gibson Paradox

		Variables	ADF	PP		Variables	ADF	PP
Levels	Constant	cpi	-1.14 (0)	-1.13 (7)	First Differences	cpi	-10.67 (0)***	-10.68 (11)***
		i	-2.02 (0)	-2.09 (2)		i	-10.25 (0)***	-10.27 (2)***
	Constant +Trend	cpi	-3.03 (0)	-3.28 (3)*		cpi	-10.66 (0)***	-10.69 (11)***
		i	-2.20 (1)	-2.05 (2)		i	-10.31 (0)***	-10.32 (3)***

Note: ***, ** and * show respectively stationary condition of series at %1, %5 and %10 significance levels. Values in parenthesis stand for optimal lag length with respect to Schwarz information criterion. If lag length is zero then Dickey-Fuller unit root test. Values in brackets show the probability values. For ADF test: MacKinnon (1996) critical values, for 1 %, 5 % and 10 %, levels are respectively 3.485, -2.885 and -2.579; for model with constant and trend probability values are respectively -3.483, -2.884 and -2.579 corresponding to 1 %, 5 % and 10 % significance levels. For PP test: MacKinnon (1996) critical values for model with constant are respectively 3.485, -2.885 and -2.579 corresponding to 1 %, 5 % and 10 % significance levels and for model with constant and trend probability values are -4.033,-3.446 and -3.148.

Both Dickey and Wayne (1979;1981) and Phillips and Perron (1988) tests give the same result that CPI and base rate are non-stationary except with constant and trend [I(1)]. Thereafter to continue the VAR model analysis, first differences of the variables have to be applied. Additionally trend variable and seasonal dummies are added to the model. Then optimal lag length is chosen where autocorrelation problem disappear.

Table-3. Toda and Yamamoto (1995) and Bootstrapped Hacker and Hatemi (2005; 2006) Granger Causality Test Results

Hypothesis	Lag-Length $k+d_{\max}$	MWALD	%1 Bootstrapped Critical Value	%5 Bootstrapped Critical Value	%10 Bootstrapped Critical Value
cpi≠> i	6	10.131 (0.071)*	15.609	11.442	9.368*
i≠>cpi	6	6.487 (0.261)	16.311	11.760	9.549

Note: ***.** and * respectively show causality relations at %1, %5 and %10 significance levels. $k+d_{\max}$ values stand for the sum of lag-length chosen according to AIC criterion and stationary level of series. Values in parenthesis show the asymptotically distributed probability values.

The results of asymptotically distributed Toda and Yamamoto (1995) Granger causality test reveal that there is causality from CPI to base rate at 10% significance level. According to Yamak and Tanriover (2007) under the circumstances of inflation targeting strategy and price stability target with financial stability target, anti-inflationary monetary policies will affect interest rates. And so the high interest rate increased the burden of both the private and public debt. Similarly the

same result is obtained from Bootstrapped Hacker and Hatemi (2005; 2006) causality test which is robust.

Hatemi and Roca (2014) asymmetric causality test results show that there is asymptotical Granger causality between positive CPI shocks and positive base rate shocks at %5 significance level and bootstrapped Granger causality between two variables at %10 significance level. Causality among positive shocks leads to the result that a rise in CPI brings about a rise in base rate.

Table-4. Hatemi and Roca (2014) Asymmetric Causality Test Results

Causality Direction	MWALD	%1	%5	%10	Causality Direction	MWALD	%1	%5	%10
$(cpi)^{+} \not\Rightarrow (i)^{+}$	4.174 (0.041)**	7.715	4.709	3.186*	$(i)^{+} \not\Rightarrow (cpi)^{+}$	1.332 (0.249)	7.427	4.132	2.738
$(cpi)^{+} \not\Rightarrow (i)^{-}$	0.564 (0.453)	12.213	4.754	2.307	$(i)^{+} \not\Rightarrow (cpi)^{-}$	0.604 (0.437)	6.899	4.002	2.744
$(cpi)^{-} \not\Rightarrow (i)^{-}$	0.568 (0.451)	12.702	4.627	2.516	$(i)^{-} \not\Rightarrow (cpi)^{-}$	2.644 (0.104)	13.987	4.677	2.497*
$(cpi)^{-} \not\Rightarrow (i)^{+}$	0.318 (0.573)	12.941	4.427	2.699	$(i)^{-} \not\Rightarrow (cpi)^{+}$	0.120 (0.729)	6.343	3.886	2.698

Note: $\not\Rightarrow$ notation shows the null hypothesis of no causality. Values in parenthesis show asymptotically distributed probability values. ***.** and * respectively show causality relations between variables at %1, %5 and %10 significance levels. Bootstrap number is 10.000.

Therefore Hatemi and Roca (2014) asymmetric causality test tells us that Gibson paradox is effective. Moreover it can be said that Fisher effect is valid, too. So contrary to Classical Theory, the reason of high interest rates seems to be high inflation rates. However Taylor (1993) makes a suggestion that the relation between inflation and interest rate means that interest rate will be an effective policy tool. According to Ravenna and Walsh (2006) the type of inflation (cost-push or demand-pull) determines the effectiveness of interest rate in monetary transmission mechanism. For this reason, it is important to clear up the basic dynamics of inflation in Turkey because central banks strongly influence short term interest rate with their monetary policy. However long term rates are set by the market and reflect the inflation rate.

Table-5. Breitung and Bertrand (2006) Frequency Domain Causality Test Results

	Long Term		Medium Term		Short Term	
ω_i	0.01	0.05	1.00	1.50	2.0	2.50
$cpi \not\Rightarrow i$	4.299*	4.310*	9.996*	1.985	0.660	2.677
$i \not\Rightarrow cpi$	1.534	1.565	1.431	3.134*	5.360*	2.148

Note: The lag lengths for the VAR models are determined by SIC. F- distribution with (2, T-2p) degrees of freedom equals 3.06.

According to frequency causality test results, which is applied to examine short, middle and long term relations between variables, there is a causality from consumer price index to monetary policy base interest rate in short and medium terms. Many cost-push factors like rising energy prices, higher tax rates, effect of devaluation or interest rate may prove temporary. Therefore, results shown in Table 5 a sight that's there is causality from interest rate to consumer price index

in the short and medium run. On the other hand, temporary cost-push factors like interest rate may influence inflation expectations and thus the temporary cost-push inflation became sustained. Therefore interest rates increases in the long run. And also we can find in that table Fisher effect is valid for Turkey in long run. For Bayat *et al.* (2013) New Keynesian monetary policy arguments implying short run effectiveness of monetary policy actions on real activity henceforth real interest rate and long run effectiveness of monetary policy actions on inflation because of price and wage stickiness. After the second quartile of 2008, the switch of Turkish Republic Central Bank (TRCB) from short-term interest rate, the only monetary policy tool, to alternative tools like liquidity management, reserve option mechanism etc., blocked the causality from base rate to CPI in the long run.

5. CONCLUSIONS

After suffering many years from high interest rate and chronic inflation problems, Turkey has switched to inflation targeting strategy by the year 2002. However Fisher (1930) argues that raises in money supply (to lower interest rate) causes inflation and high inflation results in high interest rate. This vicious cycle is called Gibson Paradox. In this study, validity of Gibson Paradox is investigated from 2003 till now. To accomplish this task, Augmented Dickey and Wayne (1981) and Phillips and Perron (1988) unit root tests and Toda and Yamamoto (1995) and bootstrapped Hacker and Hatemi (2005; 2006) Granger causality, Hatemi and Roca (2014) and Breitung and Bertrand (2006) frequency domain causality tests are applied. According to empirical results, Gibson Paradox is valid in Turkey during this period. So it can be concluded that in Turkey high inflation rates are the causes of high interest rates.

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