

Structural Vector Autoregressive (SVAR) Analysis on Malaria Incidence in Gombe, Nigeria

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Abstract

Vector autoregressive (VAR) models are capable of capturing the dynamic structure of many time series variables. Granger causality test, Impulse response functions and variance decomposition are typically used to investigate the relationships between the variables included in such models. In this context the relevant impulses or innovations or shocks to be traced out in an impulse response analysis have to be specified by imposing appropriate identifying restrictions. Taking into account the cointegration structure of the variables offers interesting possibilities for imposing identifying restrictions. Therefore VAR models which explicitly take into account the cointegration structure of the variables, so-called vector error correction models were considered in the previous work.

Granger causality test showed that Female group Granger cause Pregnant group (i.e Female group is helpful in predicting the future Pregnant malaria cases) while all other pairs were not significant. The results of impulse response functions revealed that almost all the groups had positive and/or negative effects on other groups. Finally variance decomposition analysis conducted indicates that all the groups were largely explained by their own innovations and slightly by the shocks of other groups.

Keywords: Vector Error-correction Model; Granger Causality Test; Impulse Response Function; Variance decomposition.

1. Introduction

Malaria has been a long life-threatening parasitic disease transmitted by female anopheles mosquitoes. It threatens 3.3 billion people, or half of the world population living in the world's poorest countries (WHO, 2013). An estimated 219 million malaria cases were recorded in 2010 with nearly 660,000 deaths, the burden of the disease is higher to the under fives and African region. 81% of the cases and 91% of deaths occurred in Africa where as under fives malaria deaths accounted 86% of all malaria deaths (WHO, 2013).

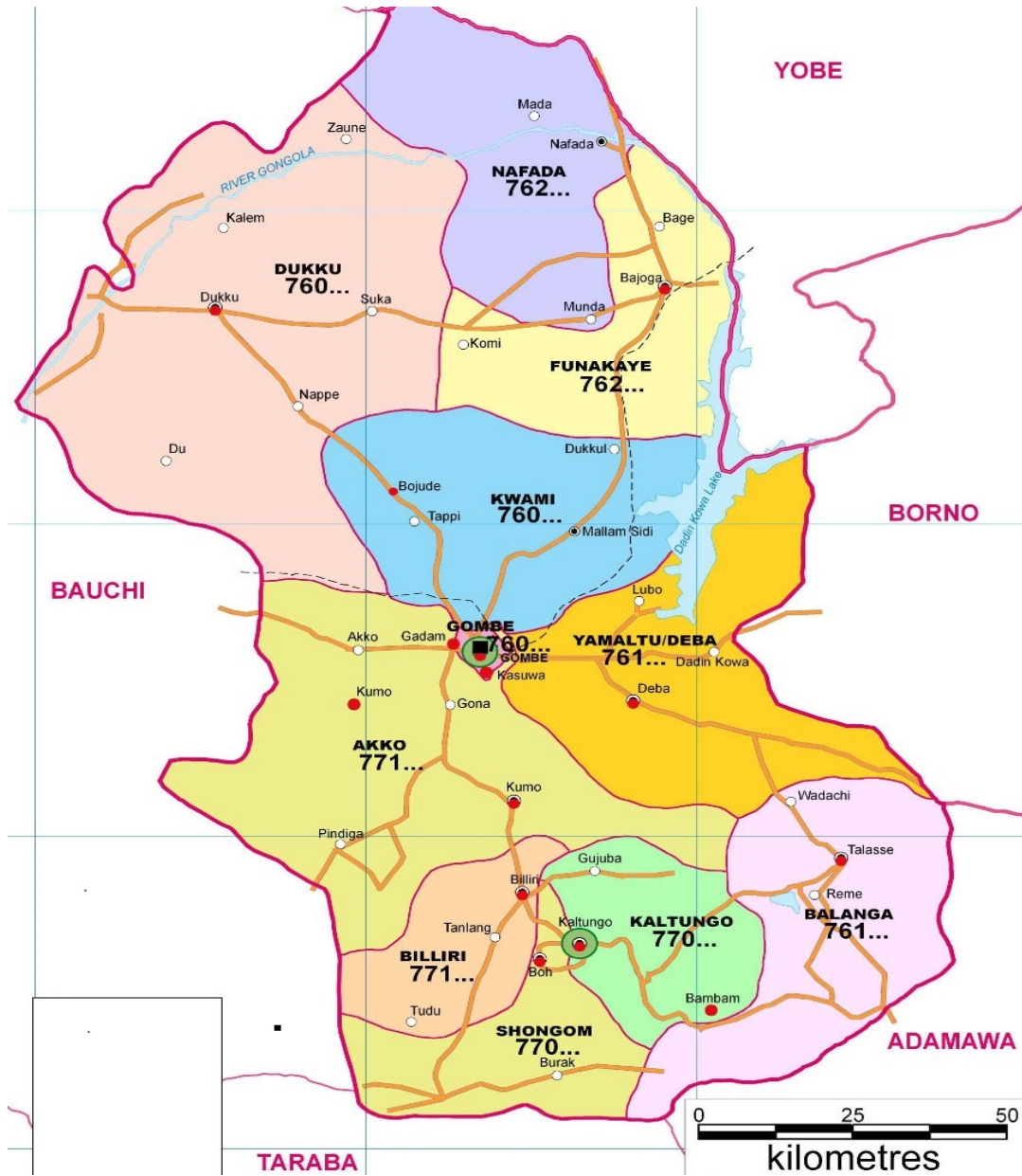
Malaria is the 3rd leading cause of death for children under five years worldwide, after pneumonia and diarrheal disease. Thirty countries in Sub-Saharan Africa account for 90% of global malaria deaths. Nigeria, Democratic Republic of Congo (DRC), Ethiopia, and Uganda account for nearly 50% of the global malaria deaths. Malaria is the 2nd leading cause of death from infectious diseases in Africa, after HIV/AIDS. Almost 1 out of 5 deaths of children under 5 in Africa is due to malaria (USAID/PMI, 2013). In semi-arid and highland regions of Africa, malaria is unstable and epidemic malaria is a common problem causing deaths annually (Worall et al, 2004). However, the risks of morbidity and mortality associated with malaria, particularly in semi-arid and highland regions, vary spatially and temporally (Snow and Marsh,2002). Most malaria infections, particularly in sub-Saharan Africa, are caused by *Plasmodium falciparum*. Malaria presents a major socio-economic challenge to African countries since it is the region most affected. This challenge cannot be allowed to go unnoticed since good health is not only a basic human need but also a fundamental human right and a prerequisite for economic growth (UN, 2003).

Oliver H. O. in his study titled 'Structural Vector Autoregressive Models and Monetary Policy Analysis' he concluded that the statistical VAR model should be augmented with economic structure in empirical applications. This can be done by cointegration analysis and by analyzing the contemporaneous relationships between the variables using the SVAR approach. Hsiao-Ching Sheng and Anthony H. Tu in their research 'A study of cointegration and variance decomposition among national equity indices before and during the period of the Asian financial crisis' The variance decomposition reveals that the 'degree of exogeneity' for all indices has been reduced, implying that no countries are 'exogenous' to the financial crisis. In addition, Granger's causality test suggests that the US market still 'causes' some Asian countries during the period of crisis, reflecting the US market's persisting dominant role.

This research work intend to study the dynamic relationships between the variables (groups) using

impulse response functions, Granger causality tests and forecast error variance decomposition (FEVD).

1.1 Map of Gombe State, Nigeria



Source: www.ccsenet.org/jgg Journal of Geography and Geology Vol. 4, No. 3; 2012)

2. Methods

The general VAR (p) model has many parameters, and they may be difficult to interpret due to complex interactions and feedback between the variables in the model. As a result, the dynamic properties of a VAR (p) are often summarized using various types of structural analysis. The three main types of structural analysis summaries are: Granger causality test, Impulse response functions and Forecast error variance decompositions.

2.1 Granger Causality tests

One of the main uses of VAR models is forecasting. The structure of the VAR model provides information about a variable's or a group of variables' forecasting ability for other variables. The following intuitive notion of a variable's forecasting ability is due to Granger (1969). If a variable, or group of variables, y_1 is found to be helpful for predicting another variable, or group of variables, y_2 then y_1 is said to Granger-cause y_2 ; otherwise it is said to fail to Granger-cause y_2 . Formally, y_1 fails to Granger-cause y_2 if for all $s > 0$ the MSE of a forecast of $y_2, t+s$ based on $(y_2, t, y_2, t-1, \dots)$ is the same as the MSE of a forecast of $y_2, t+s$ based on $(y_2, t, y_2, t-1, \dots)$ and $(y_1, t, y_1, t-1, \dots)$. Clearly, the notion of Granger causality does not imply true causality. It only implies forecasting ability. If y_1 causes y_2 and y_2 also causes y_1 the process $(y_{1t}, y_{2t})'$ is called a feedback system.

The p linear coefficient restrictions implied by Granger non-causality may be tested using the Wald statistic. Notice that if y_2 fails to Granger-cause y_1 and y_1 fails to Granger-cause y_2 , then the VAR coefficient matrices β_1, \dots, β_p are diagonal.

Suppose we have two time series x_t and y_t , we say x Granger causes y if $E(y_t/y_{t-1}, y_{t-2}, \dots) \neq E(y_t/y_{t-1}, y_{t-2}, \dots, x_{t-1}, x_{t-2}, \dots)$, that is, if we can improve the forecast for y_t based upon its own history by additionally considering the history of x_t .

In the other case $E(y_t/y_{t-1}, y_{t-2}, \dots) = E(y_t/y_{t-1}, y_{t-2}, \dots, x_{t-1}, x_{t-2}, \dots)$, where adding history of x_t does not improve the forecast for y_t , we say that x does not Granger cause y , or x is exogenous to y .

2.2 Impulse Response Analysis

Traditionally, VAR studies do not report estimated parameters or standard test statistics. Coefficients of estimated VAR systems are considered of little use in themselves and also the high (i.e. $P \times (k \times k)$ autoregressive coefficients) number of them does not invite for individual reporting. Instead, the approach of Sims (1980) is often used to summarize the estimated VAR systems by IRA. IRA traces out the effect of an exogenous shock or an innovation in an endogenous variable on all the endogenous variables in the

system over time, to provide an answer to the following question: “What is the effect of a shock of size δ in the system at time t on the state of the system at time $t + \tau$, in the absence of other shocks?

Any covariance stationary VAR (p) process has a Wold representation of the form

$$Y_t = \mu + \mathcal{E}_t + \psi_1 \mathcal{E}_{t-1} + \psi_2 \mathcal{E}_{t-2} + \dots \tag{2.1}$$

where the $(k \times k)$ moving average matrices ψ_s are determined recursively. It is tempting to interpret the (i, j) th element, ψ_{ij}^s of the matrix Y s as the dynamic multiplier or impulse response.

$$\frac{\partial y_{i,t+s}}{\partial \mathcal{E}_{j,t}} = \frac{\partial y_{i,t}}{\partial \mathcal{E}_{j,t-s}} = \psi_{ij}^s \quad i, j = 1, 2, \dots, k \tag{2.2}$$

However, this interpretation is only possible if $\text{Var}(\mathcal{E}_t) = \Sigma$ is a diagonal matrix so that the elements of \mathcal{E}_t are uncorrelated. One way to make the errors uncorrelated is to follow Sims (1980) and estimate the triangular structural VAR (p) model.

$$\begin{aligned} y_{1t} &= \alpha_1 + \mathbf{Y}'_{21} Y_{t-1} + \dots + \mathbf{Y}'_{1p} Y_{t-p} + \eta_{1t} \\ y_{2t} &= \alpha_2 + \beta_{21} y_{1t} + \mathbf{Y}'_{11} Y_{t-1} + \dots + \mathbf{Y}'_{2p} Y_{t-p} + \eta_{2t} \\ y_{3t} &= \alpha_3 + \beta_{31} y_{1t} + \beta_{32} y_{2t} + \mathbf{Y}'_{31} Y_{t-1} + \dots + \mathbf{Y}'_{3p} Y_{t-p} + \eta_{3t} \\ y_{nt} &= \alpha_n + \beta_{n1} y_{1t} + \dots + \beta_{n,n-1} y_{n-1,t} + \mathbf{Y}'_{n1} Y_{t-1} + \dots + \mathbf{Y}'_{np} Y_{t-p} + \eta_{nt} \end{aligned} \tag{2.3}$$

In matrix form, the triangular structural VAR (p) model is

$$BY_t = \alpha + \Gamma_1 Y_{t-1} + \Gamma_2 Y_{t-2} + \dots + \Gamma_p Y_{t-p} + \eta_t \tag{2.4}$$

Where

$$B = \begin{pmatrix} 1 & 0 & \dots & 0 \\ -\beta_{21} & 1 & 0 & 0 \\ \vdots & \vdots & \ddots & \vdots \\ -\beta_{n1} & -\beta_{n2} & \dots & 1 \end{pmatrix} \tag{2.5}$$

is a lower triangular matrix with 1's along the diagonal. The algebra of least squares will ensure that the estimated covariance matrix of the error vector η_t is diagonal. The uncorrelated/orthogonal errors η_t are referred to as structural errors.

The triangular structural model [3] imposes the recursive causal ordering

$$y_1 \rightarrow y_2 \rightarrow \dots \rightarrow y_n \tag{2.6}$$

The ordering means that the contemporaneous values of the variables to the left of the arrow \rightarrow affect the contemporaneous values of the variables to the right of the arrow but not vice-versa. These contemporaneous effects are captured by the coefficients β_{ij} in [3]. For example, the ordering $y_1 \rightarrow y_2 \rightarrow y_3$ imposes the restrictions: y_{1t} affects y_{2t} and y_{3t} but y_{2t} and y_{3t} do not affect y_{1t} , y_{2t} affects y_{3t} but y_{3t} does not affect y_{2t} .

Similarly, the ordering $y_2 \rightarrow y_3 \rightarrow y_1$ imposes the restrictions: y_{2t} affects y_{3t} and y_{1t} but y_{3t} and y_{1t} do not affect y_{2t} ; y_{3t} affects y_{1t} but y_{1t} does not affect y_{3t} . For a VAR (p) with n variables there are n! possible recursive causal orderings. orderings can always be compared to determine the sensitivity of results to the imposed ordering.

Once a recursive ordering has been established, the Wold representation of Y_t based on the orthogonal errors η_t is given by

$$Y_t = \mu + \Theta_0 \eta_t + \Theta_1 \eta_{t-1} + \Theta_2 \eta_{t-2} + \dots \quad (2.7)$$

where $\Theta_0 = B^{-1}$ is a lower triangular matrix. The impulse responses to the orthogonal shocks η_{it} are

$$\frac{\partial y_{i,t+s}}{\partial \eta_{j,t}} = \frac{\partial y_{i,t}}{\partial \eta_{j,t-s}} = \theta_{ij}^s \quad i, j = 1, 2, \dots, n \quad s > 0 \quad (2.8)$$

where θ_{ij}^s is the (i, j) th element of Θ_s . A plot of θ_{ij}^s against s is called the orthogonal impulse response function (IRF) of y_i with respect to η_t . With n variables there are n^2 possible impulse response functions.

In practice, the orthogonal IRF [7] based on the triangular VAR (p) [3] may be computed directly from the parameters of the non-triangular VAR (p) [1] as follows. First, decompose the residual covariance matrix Σ as

$$\Sigma = ADA' \quad (2.9)$$

where A is an invertible lower triangular matrix with 1's along the diagonal and D is a diagonal matrix with positive diagonal elements. Next, define the structural errors as $\eta_t = A^{-1} \varepsilon_t$.

These structural errors are orthogonal by construction since

$$\text{var}(\eta_t) = A^{-1} \Sigma A^{-1} = A^{-1} ADA' A^{-1} = D.$$

Finally, re-express the Wald representation [1] as

$$Y_t = \mu + AA^{-1} \varepsilon_t + \psi_1 AA^{-1} \varepsilon_{t-1} + \psi_2 AA^{-1} \varepsilon_{t-2} + \dots = \mu + \Theta_0 \eta_t + \Theta_1 \eta_{t-1} + \Theta_2 \eta_{t-2} + \dots$$

where $\Theta_j = \psi_j A$. Notice that the structural B matrix in [2] is equal to A^{-1} .

2.3 Forecast Error Variance Decomposition (FEVD)

Another way of characterizing the dynamic behaviour of a system is through FEVD (see, e.g., Hamilton (1994), Franses (1998), Chapter 9, and Lütkepohl (1993)). While IRFs trace the effects of a shock in one variable on other variables in the VAR-system, the FEVD separates the variation in an endogenous variable into component shocks to the system. If, for example, shocks to one variable fail to explain the forecast error variances of another variable (at all horizons), the second variable is said to be exogenous with respect to the first one. The other extreme case is if the shocks to one variable explain all forecast variances of the second variable at all horizons, so that the second variable is entirely endogenous with respect to the first. The FEVD can be derived from the VMA representation of the model described in Equation (1) (Lütkepohl 1993, p. 56).

The forecast error variance decomposition (FEVD) answers the question: what portion of the variance of the forecast error in predicting $Y_{i,T+h}$ is due to the structural shock η_t ? Using the orthogonal shocks η_t the h-step ahead forecast error vector, with known VAR coefficients, may be expressed as $Y_{i,T+h} - Y_{T+h/T} = \sum_{s=0}^{h-1} \Theta_s \eta_{T+h-s}$.

For a particular variable $Y_{i,T+h}$, this forecast error has the form

$$Y_{i,T+h} - Y_{T+h/T} = \sum_{s=0}^{h-1} \Theta_{i1}^s \eta_{1,T+h-s} + \dots + \sum_{s=0}^{h-1} \Theta_{in}^s \eta_{n,T+h-s}$$

Since the structural errors are orthogonal, the variance of the h-step forecast error is

$$\text{var}(Y_{i,T+h} - Y_{T+h/T}) = \sigma_{\eta_1}^2 \sum_{s=0}^{h-1} (\Theta_{i1}^s)^2 + \dots + \sigma_{\eta_n}^2 \sum_{s=0}^{h-1} (\Theta_{in}^s)^2$$

where $\sigma_{\eta_j}^2 = \text{var}(\eta_{jt})$. The portion of $\text{var}(y_{i,T+h} - y_{T+h/T})$ due to shock η_j is then

$$FEVD_{ij}(h) = \frac{\sigma_{\eta_j}^2 \sum_{s=0}^{h-1} (\Theta_{ij}^s)^2}{\sigma_{\eta_1}^2 \sum_{s=0}^{h-1} (\Theta_{i1}^s)^2 + \dots + \sigma_{\eta_n}^2 \sum_{s=0}^{h-1} (\Theta_{in}^s)^2} \quad i, j = 1, \dots, n \quad (2.10)$$

In a VAR with n variables there will be $n^2 FEVD_{ij}(h)$ values. It must be kept in mind that the FEVD in [10] depends on the recursive causal ordering used to identify the structural shocks η_t and is not unique. Different causal orderings will produce different FEVD values.

3. Discussion of Results

3.1 Granger-Causality Test

Granger causality test is considered a useful technique for determining whether one time series is good for forecasting the other. The concept of Granger causality test is explored when the coefficients of the lagged of the other variables is not zero. Table A1 presents results from the pair wise Granger-causality

tests which were obtained with two lag for each variable.

The result in Table A1 show that in all pairs the null hypothesis is not rejected (i.e the variables does not Granger cause each other) except that the pair Female does not Granger Pregnant is rejected (i.e Female Granger cause Pregnant). This implies that the Female group is helpful in predicting future values of the Pregnant group but not true in all other pairs at five percent significance level.

3.2 Impulse-Response Functions

Impulse responses trace out the responsiveness of the dependent variables in the VAR to shocks to each of the variables. So, for each variable from each equation separately, a unit shock is applied to the error, and the effects upon the VAR system over time are noted. Thus, if there are k variables in a system, a total of k^2 impulse responses could be generated. A standard Choleski decomposition is used in order to identify the short run effects of shocks on the levels of the endogenous variables in the VECM.

Impulse responses are presented in Figures 1 with the Cholesky ordering Male, Female, Pediatric and Pregnant groups. The x-axis gives the time horizon or the duration of the shock whilst the y-axis gives the direction and intensity of the impulse or the percent variation in the dependent variable away from its base line level. Figure 1 shows the responses of Male, Female, Paediatric and Pregnant groups with respect to one standard deviation innovation in Female. The result indicates Female group innovations has a positive impact on Male. It started with a very high level (192.2575) and reduces in 3, 7, 8 and 9 month. Female group innovations have a negative effect on Paediatric group. Moreover, the shocks of Female group have initially positive effect on Pregnant group and then become negative in 3 month time horizon with still positive effect in 6, 8 and 10 month horizon.

Impulse responses for Male group in Figure 1 show that the effect of a one standard deviation shock to Female group is positive which fluctuate throughout the time horizon. It also had a positive effect to Paediatric except the 3 and 4 months horizon. The effect of one standard deviation innovations of male to pregnant group changes from positive to negative throughout the time horizon.

Also the impulse responses for Paediatric group in figure 1 indicates that the effect of a one standard deviation shock to Female group is negative effect, the shocks of Male group have positive at the beginning then negative effect at 2 and 3 month and a decreasing positive effect. It also has a positive effect on pregnant group.

Finally it is observed that, the effect of a one standard deviation shock to pregnant group exhibits a changing positive and negative effect on the shocks of other groups.

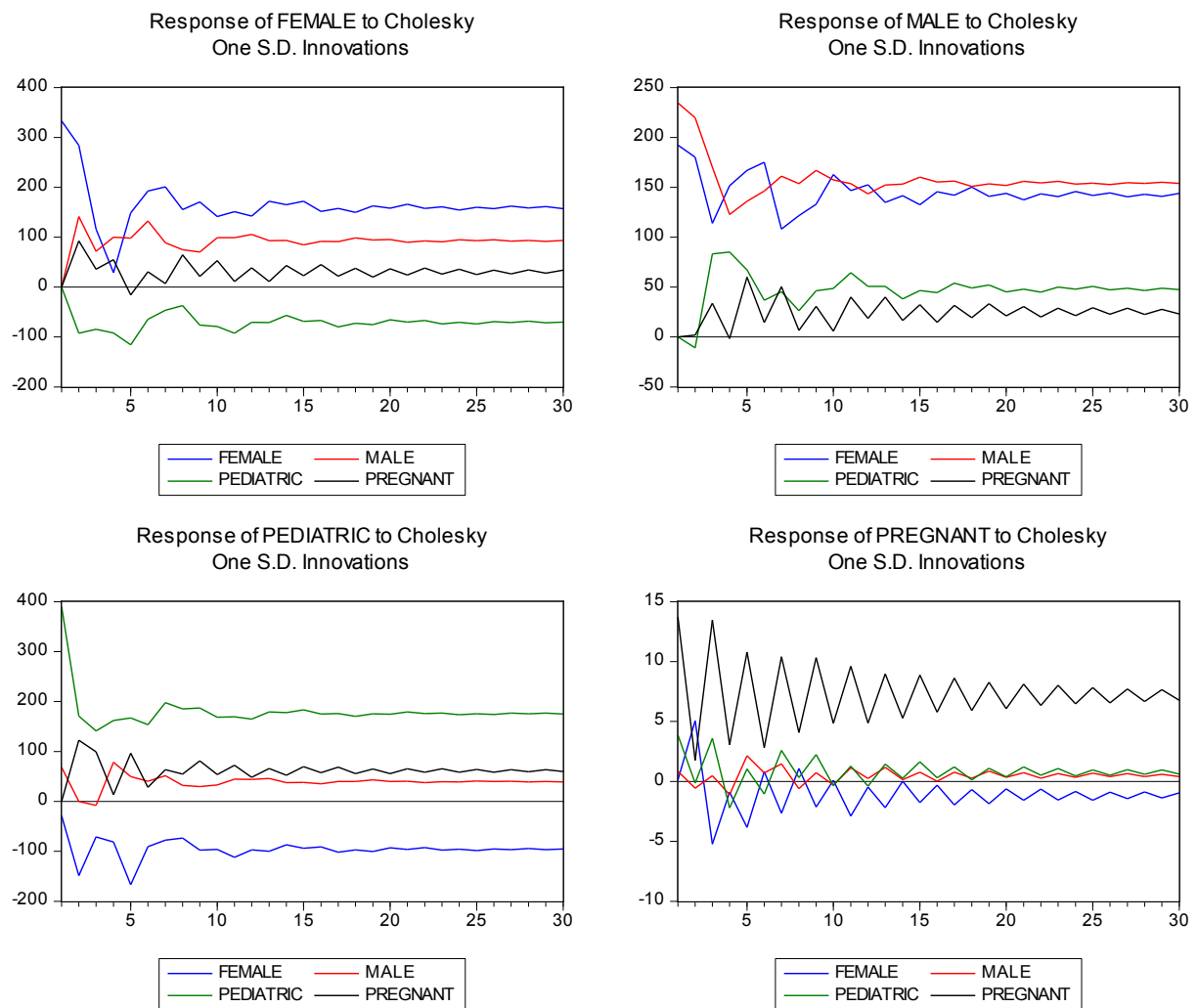


Figure 1: Response of Female, Male, Paediatric and Pregnant groups to Cholesky one S.D. Innovations.

3.3 Forecast Error Variance Decomposition

Variance decompositions offer a slightly different method for examining VAR system dynamics. The decomposition used to understand the proportion of the fluctuation in a series explained by its own shocks versus shocks from other variables. In general we expect a variable to explain almost all its forecast error variance at short horizons and smaller proportions at longer horizons. The results of the decomposition of the endogenous variables of the model are presented in Table 6, Table 7, Table 8 and Table 9 and plotted in Figure 2. These two results provide the percentage of the forecast error in each variable that could be attributed to innovations of the other variables, for different time period.

The variance decomposition analysis result in table 6 shows that, at the first horizon variation of

Female group explained only by its own shock. In the second month 83.78 % of the variability in the Female group fluctuations is explained by its own innovations. The proportion decreases for the following months to 69.06% after 10 months. Male group shocks increase the percentage as the contribution of Female group shock decreases and it reaches 16.53% after 10 months. Proportions of Paediatric group shocks decreases from and increased to 11.61% at 5 month time horizon then decreases afterwards. The role of pregnant group shocks is not significant in determining Female group.

The result in table 7 shows that, in the first month, 59.82% of the variability of the Male group is explained by its own shocks and 40.18% by Female group shocks. After 10 months, its variability decreases to 51.70% and the Female group increase to 41.88%. The Paediatric and Pregnant groups do not have significant variability to forecast Male group shocks.

The variance decomposition of Paediatric group in table A8 shows that, at the beginning 96.51% of its variability is explained by its own fluctuations which decreases to 70.59% at 10 month horizon. The effect of Female group is not significant at first horizon which becomes pronounced at higher horizons; 17.69 % at 6 month which reduces to 17.13 at 10 months . The effect of Pregnant group is not significant at the first horizon and peaked up to 8.77% at 10 month. However, the effect of Male group is not significant.

Table 9 shows the forecast variance decomposition of Pregnant group. At first horizon 92.30% is explained by its own shocks and decreases to 81.91% at 6 month and increases to 84.50% at 10 month. Paediatric group has 7.34% effect at first horizon which decreases to 5.21% at 10 month. The effect of Female group is negligible at first horizon and peaked up to 11.69% at 3 month then decreases to 9.11% at 10 month while the effect of Male group is not significant.

Table 1. Pair-wise Granger-causality tests (Eviews 7 software)

Null Hypothesis:	Obs	F-statistic	P-value
Male does not Granger cause Female	46	1.71374	0.1928
Female does not Granger cause Male		0.54811	0.5822
PEDIATRIC does not Granger Cause FEMALE	46	1.28705	0.2870
FEMALE does not Granger Cause PEDIATRIC		0.87414	0.4249
PREGNANT does not Granger Cause FEMALE	46	1.29005	0.2862
FEMALE does not Granger Cause PREGNANT		3.94142	0.0272

PEDIATRIC does not Granger Cause MALE	46	0.25515	0.7760
MALE does not Granger Cause PEDIATRIC		2.69583	0.0794
PREGNANT does not Granger Cause MALE	46	0.61967	0.5431
MALE does not Granger Cause PREGNANT		0.89965	0.4146
PREGNANT does not Granger Cause PEDIATRIC	46	0.28867	0.7508
PEDIATRIC does not Granger Cause PREGNANT		0.53508	0.5897

Table 2. Response of Female

Period	Female	Male	Pediatric	Pregnant
1	332.6339	0.000000	0.000000	0.000000
2	284.2358	141.3146	-92.36692	92.56457
3	116.1487	71.61474	-84.61359	35.74009
4	28.78291	100.1577	-91.80749	55.14606
5	148.8941	98.22280	-115.8593	-15.93530
6	192.4044	132.6584	-64.71251	30.90792
7	200.7976	88.80937	-46.27203	6.842596
8	155.4285	75.11743	-37.21601	64.69047
9	170.5691	70.22174	-76.31714	21.56236
10	141.3301	98.88151	-78.98181	52.95801

Table 3. Response of Male

Period	Female	Male	Pediatric	Pregnant
1	192.2575	234.5912	0.000000	0.000000
2	180.0820	219.6338	-10.76949	1.828630
3	113.8720	170.1112	83.21708	33.85797
4	151.5528	122.8061	85.08480	-1.473420
5	166.8280	135.9808	67.30238	60.00516
6	174.9885	146.2149	36.72056	14.38532
7	108.0655	161.1756	45.32124	50.49297
8	121.5548	153.7934	26.40549	6.536897

9	132.9436	166.8239	46.28004	30.82895
10	162.8521	157.3402	48.74335	5.777552

Table 4. Response of Pediatric

Period	Female	Male	Pediatric	Pregnant
1	-28.81076	68.40985	390.1382	0.000000
2	-148.6579	-0.129698	171.0191	122.2990
3	-71.21590	-8.024070	140.9756	99.22395
4	-81.45054	78.61581	162.1030	13.30634
5	-167.0030	49.41435	167.0001	96.66319
6	-90.76060	40.35857	153.5948	28.46038
7	-77.66917	51.45190	197.7675	63.77494
8	-73.66598	31.96919	185.3660	54.56867
9	-97.60406	29.85467	187.1873	81.33303
10	-96.06315	32.81043	168.5322	53.98008

Table 5. Response of Pregnant

Period	Female	Male	Pediatric	Pregnant
1	0.127556	0.841204	3.864999	13.70413
2	5.067098	-0.573920	-0.148774	1.757790
3	-5.239868	0.486839	3.592253	13.46753
4	-0.897303	-1.106423	-2.217950	3.060324
5	-3.828234	2.146249	1.039210	10.79301
6	0.801218	0.702373	-1.042734	2.827663
7	-2.649490	1.456767	2.592366	10.40286
8	1.076678	-0.600675	0.317851	4.082863
9	-2.138905	0.722864	2.235163	10.32648
10	0.088256	-0.287218	-0.356463	4.843132

Table 6. Variance Decomposition of Female

Period	S.E.	Female	Male	Pediatric	Pregnant
1	332.6339	100.0000	0.000000	0.000000	0.000000
2	478.0219	83.77730	8.739335	3.733683	3.749679
3	505.5303	80.18666	9.820938	6.139870	3.852527
4	527.1533	74.04144	12.64168	8.679574	4.637307
5	568.6696	70.48057	13.84658	11.60941	4.063445
6	618.9878	69.14942	16.27997	10.89163	3.678984
7	658.4380	70.41160	16.20682	10.11945	3.262139
8	684.7708	70.25230	16.18766	9.651498	3.908535
9	713.6002	70.40395	15.87448	10.03117	3.690408
10	740.2835	69.06483	16.53488	10.45936	3.940924

Table 7. Variance Decomposition of Male

Period	S.E.	Female	Male	Pediatric	Pregnant
1	303.3084	40.17887	59.82113	0.000000	0.000000
2	415.6727	40.16143	59.76951	0.067125	0.001935
3	471.9745	36.97222	59.35083	3.160831	0.516119
4	517.7365	39.29382	54.94893	5.327525	0.429723
5	567.8940	41.28917	51.40468	5.832523	1.473626
6	613.2363	43.55172	49.76902	5.360465	1.318792
7	646.7751	41.94374	50.95126	5.309958	1.795039
8	676.3771	41.58243	51.75912	5.007752	1.650696
9	711.3947	41.08180	52.28811	4.950103	1.679989
10	748.1768	41.87956	51.69581	4.899797	1.524828

Table 8. Variance Decomposition of Pediatric

Period	S.E.	Female	Male	Pediatric	Pregnant
1	397.1370	0.526295	2.967267	96.50644	0.000000
2	473.3092	10.23528	2.089051	80.99905	6.676612
3	508.7997	10.81631	1.832649	77.77027	9.580776
4	546.0277	11.61683	3.664227	76.34067	8.378271
5	604.7403	17.09688	3.654946	69.86282	9.385358
6	632.4385	17.69161	3.749038	69.77556	8.783788
7	672.1886	16.99618	3.904644	70.42338	8.675797
8	704.0061	16.58954	3.765890	71.13445	8.510121
9	740.0652	16.75168	3.570586	70.76891	8.908822
10	767.6706	17.13446	3.501080	70.59039	8.774065

Table 9. Variance Decomposition of Pregnant

Period	S.E.	Female	Male	Paediatric	Pregnant
1	14.26412	0.007997	0.347786	7.341904	92.30231
2	15.25064	11.04632	0.445867	6.432291	82.07552
3	21.32027	11.69234	0.280279	6.130108	81.89727
4	21.69949	11.45823	0.530550	6.962448	81.04877
5	24.65155	11.28989	1.169095	5.572481	81.96854
6	24.85794	11.20708	1.229600	5.656292	81.90703
7	27.23966	10.27902	1.309985	5.616121	82.79487
8	27.57335	10.18421	1.325927	5.494298	82.99557
9	29.61451	9.350353	1.209029	5.332669	84.10795
10	30.01154	9.105459	1.186411	5.206616	84.50151

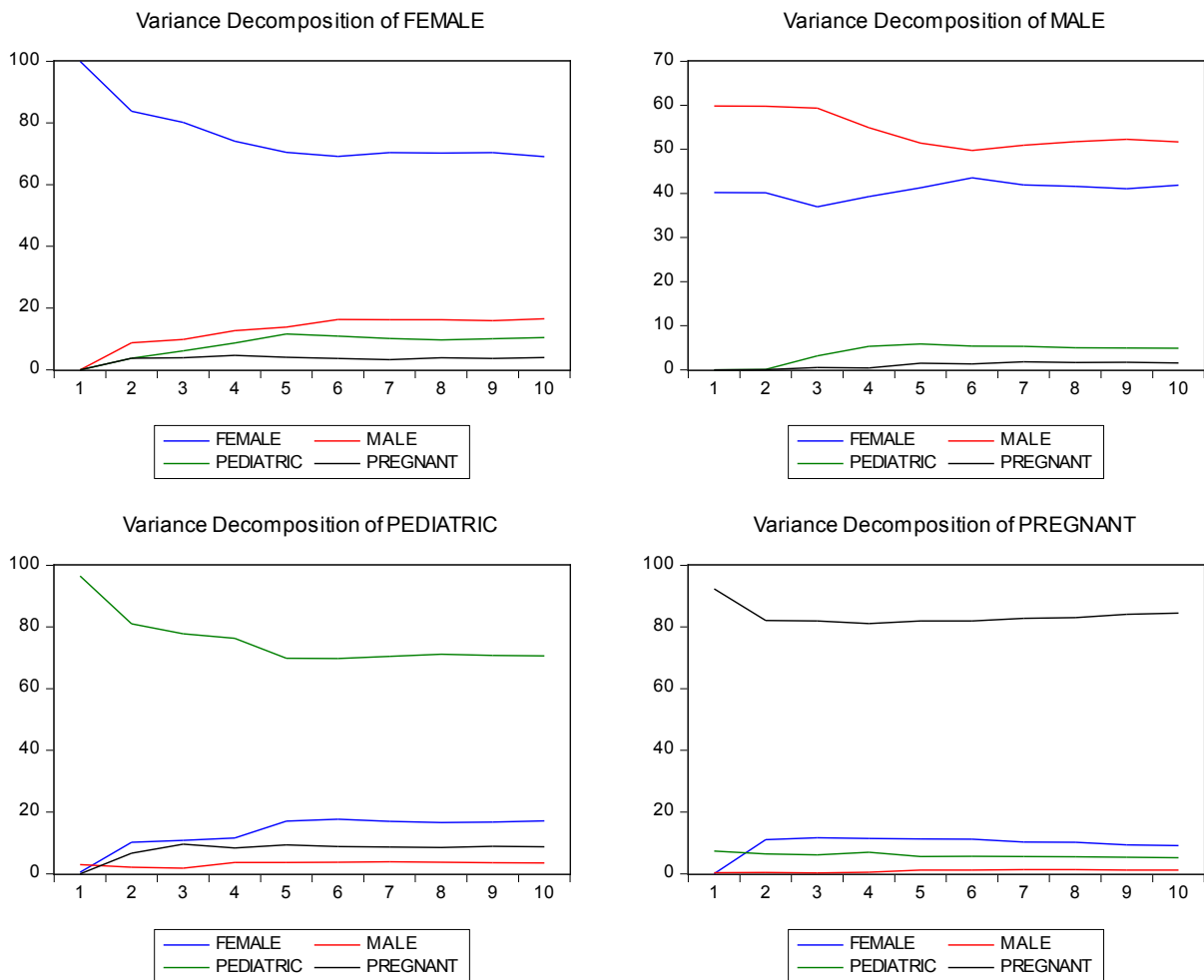


Figure 2. Variance decomposition of Female, Male, Paediatric and Pregnant groups

4. Conclusion

Granger causality test shows that Female group Granger cause Pregnant group (i.e Female group is helpful in predicting the future Pregnant malaria cases) while all other pairs were not significant.

The results of impulse response functions obtained by applying a standard Choleski decomposition indicates Female group has a positive impact on Male group, a negative effect on Paediatric group and initially positive effect on Pregnant group and then become negative. On the other hand the shocks of Male group has a positive effect on Female and Paediatric groups and a positive effect on Pregnant group which became negative at larger horizons. However, shocks of Paediatric group has negative effect on

Female group, positive effect on Pregnant group and on Male group it has positive effect and negative effect. Furthermore, the shocks of Pregnant group has positive and negative effect on all groups.

The results from variance decomposition analysis conducted showed the Female group is mainly explained by its own innovations and slightly by the shocks of Male and Paediatric groups. While Male group is explained by its own shocks and the shocks of Female group. Moreover, the variability of Paediatric group is explained largely by its own innovations and slightly by the shocks of Female group. Finally, Pregnant group is highly explained by its own innovations and slightly by Female and Paediatric groups.

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