A New ARMA G (p, q) Model for largest Viral Replication and its Posterior Distribution

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Abstract:A Statistical Models used for quantifying the viral load in the blood plasma of HIV Patients. These Models are mostly non-linear differential equations. Determination of solution of variable in the Differential equation is very complicated. The quantification of viral load by using differential equation is not as easy approach. The hierarchical Bayesian approach is used to find the predictive distribution of viral load, which is other way of finding the solution. If the prior distribution is only conjugate the expression Predictive Distribution is simple. The study of viral replication not at all considering as a single period, it is based on the number of succeeding periods. So, the researcher developed a New Auto Regressive moving average Growth process with (p, q) order for the viral replication and finds its predictive distribution.

Keywords: ARMA, HIV, Statistical Models, Auto Regressive moving average Growth process

1. Introduction

The chronically infected individuals are heterogeneous in the viral infection population. The viral replication is varing person to person. In that situation, treatment becomes the dominant strain. So, the viral replications models are characterising effective deterministic differential equation. But not at all generally a simple solution. So, the some of researcher followed the stochastic model for viral dynamic involving susceptible cell and infected CD_4^+T cell. These models are not satisfied to analyses the treatment effect. This research is concentrating the individual viral load for succeeding periods are modelled as a new ARMA G (p, q) and its predictive distribution is newly derived by Bayesian methodology.

In this paper introduce new HIV replication process and its assumption is different form postulate of the Brownian motion process. This process related to the ARMA $_{(p, q)}$ process. The model is determined the number of replication by newly derived the generating function based on concept of branching process. The following is the some of review of literature relating this study.

2. Review of Literature

Ollivier Hyrien (2005) has explained the progenitor cells give rise to different clones (or clusters) of cells that evolve in parallel so that microscopic examination of their composition at distinct time points provides various count. **Andrei Y. Yakovlev (2008)** has proposed two new models of an age dependent branching process with two types of cells to describe the kinetics of progenitor cell populations cultured in vitro. Their main focus is on the estimation of the offspring distribution from data on individual cell evolutions. **Christine Jacob (2010)** have presented a general class of branching processes in discrete time for modelling in a stochastic way some diseases propagation when the infected period is long respect to the time frequency of births. However when the transitions are population dependent, the long-term prediction of these processes is an open problem in the general case. **Yuan Yuan(2011)** has used the new stochastic models which of stochastic differential equations (SDEs) and continuous-time Markov chain (CTMC) models that, explain the account for the variability in cellular reproduction and death, the infection process, the immune system activation, and viral reproduction. Two viral release strategies are considered: budding and bursting. The CTMC model is used to estimate the probability of virus extinction during the early stages of infection. **Fernando Antoneli (2011)** has referred the initial viral population starts replication constrained by the unavoidable interaction with the host organism and evolves in time towards an eventual equilibrium. **Jessica M. Conway (2018)** has investigated the initial stages of HIV infection within a host and they have developed a multi-type, continuous-time branching process model. This model is a stochastic extension of the standard viral dynamics model, under the assumption that the number of cell targets for viral infection is constant, biologically reasonable since, during the earliest stages of HIV infection, very few cells are infected relative to their total population size. **Abid Ali Lashari (2018)** has developed to present a branching process approach for analysing the early stages of an outbreak of a sexually transmitted infection, or any other infectious disease, spreading along the dynamic network. **Aadrita Nandi and Linda J. S. Allen (2019)** have explained the Multitype branching processes approximate the

dynamics of the CTMC model near the disease-free equilibrium and it is used to estimate the probability of a minor or a major epidemic. **Lubna Pinky (2019)** have proposed the computing time of the direct method scales linearly with the initial number of target population the direct method becomes infeasible to simulate viral infection models with realistic number of target cells, i.e. of order 1×10^8. **Antonio A. Alonso (2020)** has used stochastic models for the estimation of parameters to successfully fit experimental data in particularly challenging problem. For instance, if Monte Carlo methods are employed to model the required distributions of times to division, the parameter estimation problem can become numerically intractable. They overcame this limitation by converting the stochastic description to a partial differential equation (backward Kolmogorov) instead, which relates to the distribution of division times. **Katrin Haeussler (2018)** has developed to present a dynamic MM under a Bayesian framework. They extended a static MM by incorporating the force of infection into the state allocation algorithm. The corresponding output is based on dynamic changes in prevalence and thus accounts for herd immunity. **Verrah Otiende , Thomas Achia (2019)** have identified elevated risk areas for TB/HIV co infection and fluctuating temporal trends which could be a result of improved TB case detection or surveillance bias caused by spatial heterogeneity in the co -infection dynamics. The elevated risk areas indicated the need for focused interventions and continuous TB-HIV surveillance. The following is designed the model for stochastic variation of viral load.

A New model for stochastic variation of viral load

Let $\{X(t), t\epsilon T\}$ be a HIV replication process at continuous time interval with satisfying the following assumptions.

(i) $P(X(t) = 1) = 0$, if $t = 1$.
(ii) $\{X(t), t\epsilon T\}$ has dependent increments., $t = 1,2, \dots n\epsilon T$
(iii) $\{X(t), t\epsilon T\}$ has not stationary increments.
(iv) $P[X(t) - X(s) \leq u] = \int_0^\infty e^{-\beta u} u^{\alpha-1} du$,$0 < u < \infty$

Where $u = X(t) - X(s)$ (number of replication between the time t and s), T is time.$T \in R$.

Viral replication depends on sensing and responding to diverse environment factors, often involving the activation and expression of multiple genes. The viral transformation process from virus to the $CD_4^+ T$ cell by the nucleus reactor of the human body. But replication of viral particles RNA reactor with DNA of $CD_4^+ T$ cell is connected with the branching process.

Let $t_1$is assumed that the initial time of infection of $CD_4^+ T$ cell. At the time t_i, the initial infection of $CD_4^+ T$ cell by single the HIV is denoted by $X_0 = 1$, then it replication from the $CD_4^+ T$ cell is denoted by $X_1 = \varepsilon_1$, with probability $P(X_1 = \varepsilon_1) = p_1$, is called as first stage viral replication then at time t_2 the second duration of period , the second stage viral replication is denoted by X_2,with probability $P(X_2 = \varepsilon_2) = p_2$ so on.

At time t_{n+1} $(n+1)^{th}$ stage viral replication only depends on $(n)^{th}$ stage replication. But n^{th} stage replication depends on the previous $(n-1)^{th}$ stage replication.

Let $\{X_1, X_2, \dots, X_n\}$ is denoted by viral replication process and its generating function of the viral replication is represented by

$$E(e^{-s_i X_i}) = \sum_{i=1}^{n} e^{-s_i X_i} P(X = x_i)$$

Where $1 < s_i < s_0 \epsilon R$ and at each stage viral replication is increasing nature. So it is assumed to be the exponential distribution. Therefore the generating function becomes,

$$Z(s_i) = \int_1^{s_1} \int_{s_1}^{s_2} \cdots \int_{s_n}^{s_0} e^{-s_i x_i} \theta e^{-\theta_{x_i}} dx_1 \dots dx_n$$

Where the $1/\theta$ is the average viral replication over the n period.

The average viral replication of n stages for n $CD_4^+ T$ cells is given by

$$\begin{bmatrix} \lambda_{11} & \lambda_{12} & \cdots & \lambda_{1\mathrm{N}} \\ \lambda_{i1} & \lambda_{i2} & \cdots & \lambda_{ij} \\ \vdots & \vdots & \ddots & \vdots \\ \lambda_{n1} & \lambda_{n2} & \cdots & \lambda_{nN} \end{bmatrix} \qquad i = 1,2,\dots,n, j = 1,2,\dots,N.$$

Since average replication of each stage is considered as increasing nature such that, $\lambda_{i1} < \lambda_{i2} < \cdots < \lambda_{in}$ and their "n" stage transition probabilities is given by

$$P_1 < P_2 < P_3 < \cdots < P_n \qquad (i.e.,) \sum_{i=1}^{n} p_i = 1,$$

The probability generating function of the i stage is denoted by$Z_i = e^{-s_j} s_j^{x_i}$, where $0 < s_j < 1$, and X_i is the i^{th} stage viral replication as X_i the n[th] stage. The probability generation function of viral replication is denoted by $Z_n = Z_{n-1}(Z_{n-2}) \dots Z_1$ if $Z_i > Z_{i-1}, (n+1)^{th}$ stage probabilities generation function of viral replication is given by.

$$Z_{n+1} = \prod_{i=1}^{n} Z_{i\,,}\ i = 1,2,\dots,n$$

$$= \prod_{i=1}^{n} e^{-s_j} s_j^{x_i},\ 0 < s_j < 1$$

and the first stages $j = 1,2,\dots,m$ and initial replication as assume that $Z_0 = 1$. At each stage j can be randomly choose n and it is fixed for each stage. if j=1 at first stage $s_j = s_1 = 0.1 and\ if\ j = 2, s_j = s_2 = 0.2\ and\ s_j\ is\ 0 < s_j < 1$. Therefore $Z_{n+1}$is the $n + 1^{th}$ stage probability generating function of the viral replication process by under taking the concept of branching process. X_{n+1} is the viral replication at the $(n+1)^{th}$ stage.

Let the viral replication at the i^{th} stage is considered as Auto Regressive Model of $X_i = \alpha_0 + \alpha X_{i-1} + \varepsilon_i$. where ε_i is distribution normal with mean zero and varianceσ^2, and

$$\varepsilon_i = \beta_0 \varepsilon_{i-1} + e_0$$

$$\varepsilon_1 = \beta_1 \varepsilon_0 + e_0$$

$$\varepsilon_2 = \beta_2 \varepsilon_1 + e_1$$

$$\vdots$$

$$\varepsilon_n = \beta_n \varepsilon_{n-1} + e_n$$

Where $\varepsilon_0 < \varepsilon_1 < \cdots < \varepsilon_n$ and $e_0 < e_1 < e_2 < \cdots < e_n$.

Where ε_i's are extraneous factor of biological reaction during process of the viral replication depends on the previous stage replication process and number of infected CD_4^+T cells so on so this processes is increasing nature in the current stage compare to the previous stage.

$\alpha_0 -$ is the constant replication per each stage.

$X_{i-1} -$ is the viral load at the previous stage.

$\varepsilon_i -$is DNA capacity due to the biological reaction.

$\alpha_i -$is the proportion of viral replication in the current stage.

Then $(n+1)^{th}$ stage viral load is given by,

$X_{n+1} = \alpha_0 + \alpha_1 X_1 + \alpha_2 X_2 + \cdots + \alpha_p X_\beta + \beta_0 + \beta_1 \varepsilon_0 + \beta_2 \varepsilon_1 + \beta_3 \varepsilon_2 + \cdots + \beta_q \varepsilon_{q-1} + e_{pq}$. $e_{pq} = e_0 + e_1 + \cdots + e_n, e_{pq} \sim N(0, \sigma^2)$

Where $e_p = \beta_0 + \beta_1 \varepsilon_0 + \beta_2 \varepsilon_1 + \beta_3 \varepsilon_2 + \cdots + \beta_q \varepsilon_{q-1} + e_{pq}$.

$\varepsilon_i \sim exp(\sigma_i)$ and $e_i \sim exp(\delta_i)$

From the above assumption the $(n+1)^{th}$ stage viral replication process of growth order (p, q) is given byX_{n+1}. It is a new auto regressive moving average processes of order (p. q), it is denoted by ARMA G (p, q).

Let us assume that the initial check up one virus particle infect the one $CD_4^+ T$ cell as denoted as $X_0 = 1$ and $i = 1, \alpha_0 = 1$and every six month duration viral check up is considered as a stage. It is a random variable denoted by X_i

(i) Initial infection is assumed that one $CD_4^+ T$ cell is infected.

(ii) In the first generation one $CD_4^+ T$ broken out, number of virus with range [c, d]
Where α_1 is the co-efficient of generation,
Therefore, $X_1 = \alpha_1(a^{X_0})$,

$$X_1 = f(X_0) = \alpha_1(a^{x_0}),$$

Where $a \epsilon [c, d] \epsilon [10,100]$; and $\alpha_i = 1,2, \ldots,$ nϵR > 0

$$\Rightarrow X_1 = \int_{10}^{100} \alpha_1 a^{x_0} da$$

$$= \alpha_1 \frac{[a^2]}{2} \Big|_{10}^{100} = 5000 - 50 = 4500.$$

(iii) Second generating $X_2 = f(X_1)$

$$= \alpha_2 f(X_0)$$

$$= \alpha_2 \alpha_1 \int_{10}^{100} (a^{X_0}) da$$

$$\vdots$$

$$X_{n+1} = f(X_n)$$
$$= \alpha_n \alpha_{n-1} \ldots \alpha_1 (a^{X_0})$$
$$= \int_c^d \alpha_n \alpha_{n-1} \ldots \alpha_1 (a^{X_0}) da$$

Normally, the viral infection extraneous factor involvement variation is increasing nature. So, the Biological error is considered as exponential growth. But infected patient's viral growth is usually distributed as normal. Then the largest replication is denoted by y_n and its prediction density for future replication of a particular patient is given by

Let $f(y_n)$ is the density of the largest viral replication in the current period (stage).

$$y_n \sim N(0, \sigma_n^2)$$

$$f(y_n) = \frac{1}{\sqrt{2\pi\sigma_n^2}} e^{-1/2\left(\frac{y_n^2}{\sigma_n^2}\right)}$$

$$f(y_n) = \int_{-\infty}^{y_n} \frac{1}{\sqrt{2\pi}\sigma_n} e^{\frac{-1}{2\sigma_n^2}(y_n)} dy_n$$

Current stage, largest viral load density is denoted by

$$f_\alpha(y_n) = n[F(y_n)]^{n-1} f(y_n)$$

$$= n\left[\int_{-\infty}^{y_n} c.e^{-\frac{1}{2\sigma_n^2}(y_n)^2} dy_n\right]^{n-1} \frac{1}{\sqrt{2\pi}\sigma_n} e^{-1/2y_n^2}$$

The prior density of σ_n^2 is also exponential growth of viral load, its density follows the gamma random variable with parameter $(\alpha, \beta) > 0$, and it is denoted by

$$f(\sigma_n^2) = \frac{\beta^\alpha}{\Gamma\alpha} e^{-\beta\sigma_n^2}(\sigma_n^2)^{\alpha-1} d\sigma_n^2$$

The posterior density function of the viral load variation in the current stage is denoted by

$$P(\sigma_n^2/y_n) = f(y_n) \cdot P(\sigma_n^2) \quad \ldots (1)$$

$$= n\left[\int_{-\infty}^{y_n} c\, e^{\frac{-y_n^2}{2\sigma_n^2}} dy_n\right]^{n-1`} \frac{1}{c} e^{\frac{-y_n^2}{2\sigma_n^2}} \frac{\beta^\alpha}{\Gamma\alpha} e^{-\beta\sigma_n^2}(\sigma_n^2)^{\alpha-1}.$$

$$= \frac{n}{c}\frac{\beta^\alpha}{\Gamma\alpha} e^{-y_n^2} e^{-\left(1/2\sigma_n^2+\beta\sigma_n^2\right)}(\sigma_n^2)^{\alpha-1}(F)^{n-1}$$

Let

$$F = \int_{-\infty}^{y_n} \frac{1}{c} e^{\frac{-1}{2\sigma_n^2}y_n^2} dy_n - (2\beta - 1)\sigma_n^{-1}$$

$$= \int_{-\infty}^{y_n} f(y_n) dy_n + \int_{0}^{y_n} f(y_n) dy_n.$$

$$= 0.5 + \frac{1}{c}\int_{-\infty}^{y_n} e^{\frac{-1}{2\sigma_n^2}y_n^2} dy_n.$$

Where $\frac{1}{2\sigma_n^2} = a$

$$= \frac{1}{c}\int_{0}^{y_n} e^{\frac{-y_n^2}{a}} dy_n$$

Where $\frac{y_n^2}{a} = y \Rightarrow y_n^2 = {}^{y}/_{a}$, $y_n = \sqrt{{}^{y}/_{a}}$

$2y_n dy_n = dya$

$dy_n = \frac{dy}{2\sqrt{{}^{y}/_{a}}}$

$$= \frac{1}{c}\int_{0}^{\sqrt{{}^{y}/_{a}}} e^{-{}^{y}/_{a}} \frac{dy}{2\sqrt{{}^{y}/_{a}}} n$$

$$= 2c \int_0^{\sqrt{y/a}} e^{-y/a} \left(y/a\right)^{1/2} dy$$

Since $\int_0^{\infty} e^{-y/a} \left(y/a\right)^{1/2-1} dy = \frac{\Gamma 1/2}{(1/a)^{1/2}} = \frac{\Gamma 1/2 \times 2c}{\sqrt{1/a}}$

$$= \frac{\Gamma\, 1/2 \times 2\sqrt{2\pi}\sigma_n}{\sqrt{1/\sigma_n}}$$

$$= 2\pi(\sigma_n)^{3/2}$$

$$= \sqrt{2/\sigma_n}$$

$$\int_0^{y_n/\sigma_n} f(x)dx < \int_0^{\infty} f(x)\, dx < 2\pi\sigma_n^{3/2}$$

$$\int_{-\infty}^{y_n} f(y_n)dy_n = 2\pi(\sigma_n)^{3/2}\ maximum$$

Since, human viral replication nonnegative therefore the density of highest replication is truncated as

$$\int_0^{y_n} f(y_n)dy_n < \sqrt{2/\sigma_n}$$

Therefore the posterior density (1) become

$$P(\sigma_n^2/y_n) = \frac{n}{\sqrt{2\pi}\sigma_n}\frac{\beta^{\alpha}}{\Gamma\alpha} e^{-y_n^2} e^{-\left(\frac{1}{2\sigma_n^2}+\beta\sigma_n^2\right)(\sigma_n^2)^{\alpha-1}\left(2\pi\sigma_n^{3/2}\right)}$$

$$= n\sqrt{2\pi}\frac{\beta^{\alpha}}{\Gamma\alpha} e^{-y_n^2} e^{-\left(1/2\sigma_n^2+\beta\sigma_n^2\right)} (\sigma_n^2)^{\alpha-1/2}\frac{1+\beta}{2\sigma_n^2}$$

Integrated out the σ_n^2 of the posterior density

$$\int_0^{\infty} P(\sigma_n^2/y_n)d\sigma_n^2$$

$$= \int_0^{\infty} e^{-y_n^2} e^{-\left(1/2\sigma_n^2+\beta\sigma_n^2\right)(\sigma_n^2)^{\alpha-1/2}} d\sigma_n^2$$

$$= \int_0^{\infty} ce^{-y_n^2} e^{-\left(1/2\sigma_n^{2+\beta\sigma_n^2}\right)(\sigma_n^2)^{\alpha-1/2}} d\sigma_n^2$$

$$= \int_0^{\infty} ce^{-\left(\frac{1}{2\sigma_n^2}+\beta\sigma_n^2\right)(\sigma_n^2)^{\alpha-1/2}} \mathrm{e}^{-\mathrm{y}_n^2}\, d\sigma_n^2$$

$$= \int_0^{\infty} ce^{-y_n^2}\, e^{-(2\beta-1)\sigma_n^2}(\sigma_n^2)^{\alpha+1/2-1} d\sigma_n^2$$

$$= \frac{\Gamma\alpha}{n\sqrt{2\pi}\beta^{\alpha}} \mathrm{e}^{-\mathrm{y}_n^2}$$

Where, $c = n\sqrt{2\pi}\frac{\beta^{\alpha}}{\Gamma\alpha}$

The largest viral replication depends on the extraneous factors CD_4^+T DNA and it's the next stage viral density is given by;

$$= \frac{\Gamma\alpha}{n\sqrt{2\pi}\beta^{\alpha}} e^{-y_n^2} . \frac{\Gamma\alpha + {}^1/_2}{(2\beta - 1)^{\alpha + {}^1/_2}}$$

$$= \frac{\Gamma\alpha e^{-y_n^2} \alpha! \, \Gamma\, {}^1/_2}{n\sqrt{2}\, \Gamma\, {}^1/_2 \, (2\beta - 1)^{\alpha + {}^1/_2}}$$

$$= \frac{\Gamma\alpha e^{-y_n^2} \alpha!}{n\sqrt{2}\, \beta^{\alpha} (2\beta - 1)^{\alpha + {}^1/_2}}$$

$$= \frac{\Gamma\alpha + 1 \; e^{-y_n^2}}{n\sqrt{2}\, \beta^{\alpha} (2\beta - 1)^{\alpha} (2\beta - 1)^{{}^1/_2}}$$

The viral density is illustrated through the sample data assumed for the scale parameter of prior distribution.

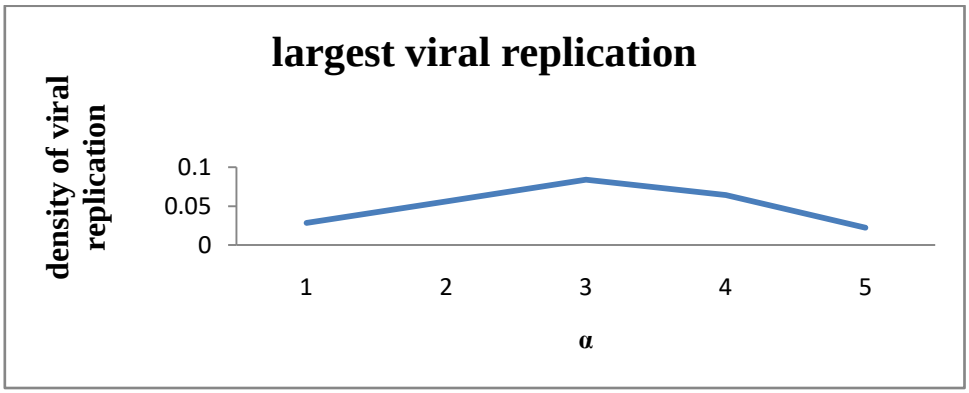
Numerical results

Table: 1

Table: 1 illustrates density of viral load for the largest viral replication for different time periods with special case $n = 10, \beta = 1$ based on the scale parameter of the prior distribution.

Prior distribution parameter $\boldsymbol{\alpha}$	**Posterior density** $\frac{\boldsymbol{\Gamma\alpha + 1 e^{-y_n^2}}}{\sqrt{2}(2\beta - 1)^{\alpha}(2\beta - 1)^{1/2}\beta^{\alpha}}$
1	0.63
2	1.72
3	2.54
4	1.67
5	0.55

Graph: 1



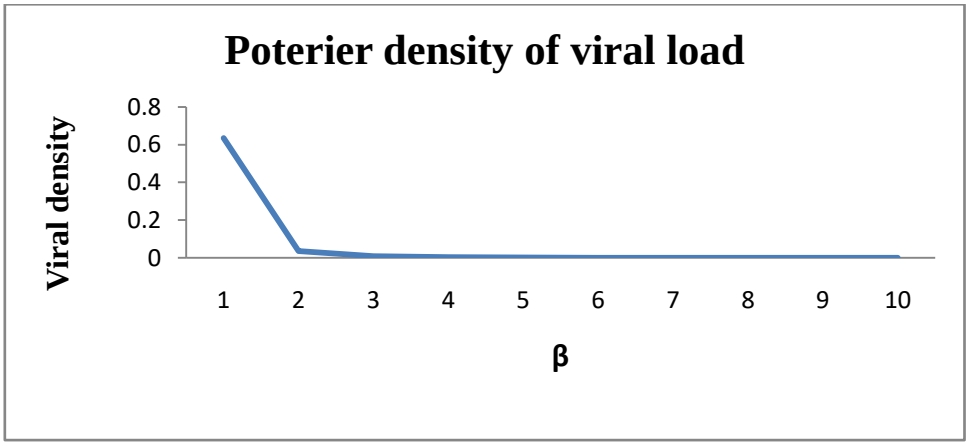
Graph: 1 illustrate that density viral load various scale parameter of prior distribution.

Table: 2

Table: 2 illustrates largest the viral load for different time periods with special case $n = 10, \alpha = 1$ based as the various shape parameter of the prior distribution.

Prior Parameter $\boldsymbol{\beta}$	**Posterior Density** $\dfrac{\Gamma\alpha + 1\, e^{-y_n^2}}{\sqrt{2}(2\beta - 1)^{\alpha}(2\beta - 1)^{1/2}\beta^{\alpha}}$
1	0.635
2	0.035
3	0.0084
4	0.0032
5	0.0015
6	0.0008
7	0.0005
8	0.0003
9	0.0002
10	0.0001

Graph: 2



Graph: 2 illustrate that viral density based on the various Shape Parameter of the Prior Distribution.

Conclusion

The major challenge for worldwide health department to treat the HIV infection. If there is no specific medicine to the treatment of HIV infection. World Health Organization (WHO) and other related sectors are planning to how optimize the cast and extended the HIV patient's future life time. In that situation, Development of a Statistical Model useful to predict the future replication of the virus in the human body. So, this research is concentrated to develop a new ARMA G (p, q) model for largest viral replication and also find the prediction distribution based on the prior distribution. The prediction viral replication for future period essential for determination of medicine and patient life time. This kind of prediction is very much useful for health and related departments in the Government sector for the Budget Planning.

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