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Intracellular Virus Dynamics A Study of The Converting from The Deterministic Model to its Stochastic Counterpart

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Abstract

By examining both deterministic and stochastic models, the intracellular viral movement model explores the complex dynamics of the interaction of viruses with host cells. Since such movement is not deterministic but rather random, the main goal of this study is to build a stochastic model corresponding to the deterministic one, that describes the movement of viruses and their intracellular interactions in a more realistic way. The model helps explain how viruses are produced and reproduce by analyzing the mechanisms that generate and deplete structural proteins and viral nucleic acids, and by examining the effect of the viral template, the findings may help improve methods for treating and preventing viral infections by shedding light on structural proteins and viral DNA. The deterministic and stochastic systems were solved numerically and represented using MATAB tools, to gain deeper insights.

Keywords: Intracellular Virus Movement Model, Stochastic Model, Covariance Matrix, Diffusion Matrix.

INTRODUCTION

Viruses are infectious agents that are tiny, obligate intracellular parasites that are incapable of self-replication. These are also acellular creatures, meaning that their genomes are either RNA or DNA (nucleic acid) and are encased in a protective protein coat that is encoded by the virus. Every virus can only replicate obligately in live cells. They do this by utilizing the metabolic processes and ribosomes of the host to create a collection of parts that come together to form molecules known as VIRIONS, which guard the genome and spread it to new cells (Gelderblom, 1996) https://www.uoanbar.edu.iq/eStoreImages/Bank/14736.

All living things, including bacteria, archaea, plants, and animals, are susceptible to virus infection. https://www.uoanbar.edu.iq/eStoreImages/Bank/14736.The infectious units known as viruses range in diameter from around 16 nanometers for circoviruses to over 300 nanometers for poxviruses. Because of its microscopic size, it is ultra-filterable, meaning that bacteria-resistant filters cannot hold it (Modrow et al., 2013). Humans and other species have been greatly impacted by viruses, yet until recently, little was understood about their nature. Clarifying their nature can be aided by a brief history of their discovery and identification as distinct infectious agents. Despite their ignorance of the nature of their illnesses, the ancients were aware of conditions like rabies, which are understood to have a viral cause today. Indeed, there is considerable evidence that the measles and smallpox vi-

ruses were likely responsible for the enormous epidemics that struck between 165 and 180 AD and between 251 and 266 AD., which significantly damaged the Roman Empire and contributed to its fall (Harley, 2002). Numerous discoveries in several fields of biology during the past 20 years have fundamentally altered our understanding of the viral world. The conventional understanding of viruses as passive biological entities that evolved mainly by choosing genes from their hosts and had a secondary function in evolution is contradicted by several of these findings. It is now known that viruses are extremely varied, and very old—they existed before the Last Universal Cellular Ancestor (LUCA) and that they were crucial to the evolution of life. These new findings have led to the proposal of new definitions and ideas for viruses. The idea of the viral cell, in particular, affirms that viruses are cellular entities and that they are capable of producing their own DNA (Forterre, 2017). Over millions of years, viruses have changed to fit certain creatures or their cells. Proteins make up infectious viral particles, or virions, which are encased in a fatty membrane known as the envelope in certain virus species. Only one kind of nucleic acid—either DNA or RNA—is present in the particles. Unlike bacteria, yeasts, or other cells, viruses multiply within the live cells they infect rather than by dividing (Modrow et al., 2013). Virologists study viruses, which are obviously extremely different from prokaryotic and eukaryotic microbes. Viruses are very significant and require careful consideration, even though they are simpler than biological creatures, (Harley, 2002). Although viruses may cause terrible illnesses in a variety of creatures, they are also straightforward systems that can be used for a wide range of beneficial reasons. Viruses have long been used in medicine to make vaccinations, and they are now utilized as vectors to carry chemicals that are needed to cure illnesses like cancer so that they may target certain cells. They have also been utilized to precipitate certain metals in nanotechnology and agriculture, and they have shown significant promise in the creation of nanomaterials. Additionally, they have a variety of uses in the electronics, cosmetics, pharmaceutical, and other sectors. As a result, viruses are no longer just considered adversaries (Varanda et al., 2021). We also point out that the study of viruses has made significant contributions to the science of molecular biology, as indicated by the recent appearance of AIDS and the fact that numerous viral illnesses in humans are already recognized, with new ones being identified or emerging each. Viral discoveries are the foundation of the entire discipline of genetic engineering (Harley, 2002).

The ability of viruses to infiltrate cells from bacteria, archaea, and eukaryotes is a result of their evolution. The majority of the more than 3,600 identified viruses are linked to illness, and hundreds of them have the ability to infect human cells. Animal viruses attach themselves to host cell receptors to enter the cell. Understanding how viral entry proteins interact with their host cell receptors and change conformation to allow entrance offers previously unheard-of possibilities for creating novel therapies and vaccines. The first and most crucial stage of a virus' life cycle is its entrance into the host cell. After attaching to receptors, viruses enter animal cells by either fusing with cellular membranes (enveloped viruses), penetrating through (non-enveloped viruses), or undergoing significant conformational changes to their proteins. When viral genomes are transferred into host cells, the procedure is complete (Dimitrov, 2004). Viruses are intracellular pathogens, meaning they need the metabolic machinery of their host cell to proliferate. Although there are significant differences in the reproductive life cycle of different viral kinds and classes, attachment, penetration, disassembly, replication, assembly, and virus release are the six fundamental processes required for virus reproduction. https://www.immunology.org/sites/default/files/2022-08/Virus%20replication.pdf

The public's health is still at risk from the seasonal and pandemic influenza virus (IAV). The kinetics of the immune response to IAV infection and the biological factors that significantly affect infection outcomes, however, are not well understood quantitatively. In order to tackle these prob-

lems, we statistically examine innate and adaptive immune responses to primary IAV infection using modeling techniques in conjunction with experimental data. The dynamic interactions between target (epithelial) cells, viruses like influenza virus, cytotoxic T lymphocytes (CTLs), and virus-specific IgG and IgM have been described mathematically. Simulation studies have also been conducted to determine the relative contributions of biological parameters to the clearance of IAV (Miao et al., 2010). This study provides a model of virus movement inside cells, as by adjusting the parameters according to the behaviors of different viruses, it is suitable for many viruses, including influenza virus, HIV, and others. This is to provide precise insights into how different viruses interact with cells and develop effective methods to combat viral diseases.

The basic interactions between viral components may be captured by deterministic models that are based on ordinary differential equations. On the other hand, a single viral particle can start an infection by transferring its genome — a single DNA or RNA molecule — to the host cell. A stochastic model that takes into account the natural variations in viral component levels might produce qualitatively different results in these circumstances (Srivastava et al., 2002). We have created a basic model of the intracellular dynamics of a generic virus that may be implemented either stochastically or deterministically in order to compare modeling techniques (Allen, 2010). Therefore, the proposed mathematical model is

$$\begin{cases} \frac{dT}{dt} = f_1(T,G,S) = K_1 G - K_2 T. \\ \frac{dG}{dt} = f_2(T,G,S) = K_3 T - K_1 G - K_4 GS.. \\ \frac{dS}{dt} = f_3(T,G,S) = K_5 T - K_6 S - K_4 GS. \end{cases}$$

where all constants $K_i, i = 1,2,3,4,5,6.$ are in units of day, and the variables described in Table 1.

Table: (1). Description of state variables of the proposed model

Variable	Description
T	viral template.
G	viral genome.
S	structural proteins

The model reflected the processes that produced and depleted structural proteins and viral nucleic acids. Before creating the stochastic model for the system, we will study the stability of its system at the equilibrium point (T^*, G^*, S^*) (Chou & Friedman, 2015).

1- The equilibrium points:

$$\frac{dT}{dt} = 0 \Rightarrow K_1 G - K_2 T = 0 \Rightarrow K_1 G = K_2 T \implies T^* = \frac{K_1}{K_2} G^* .$$

$$\frac{dG}{dt} = 0 \Rightarrow K_3 T - K_1 G - K_4 GS = 0 \Rightarrow S^* = \frac{K_1 K_3}{K_2 K_4} - \frac{K_1}{K_4} = \frac{K_1 (K_3 - K_2)}{K_2 K_4}.$$

$$\frac{dS}{dt} = 0 \Rightarrow K_5 T - K_6 S - K_4 GS = 0 \Rightarrow G^* = \frac{K_6 (K_3 - K_2)}{K_4 (K_5 - K_3 + K_2)}.$$

2- The Jacobian matrix:

$$J = \begin{pmatrix} \frac{\partial f_1}{\partial T} & \frac{\partial f_1}{\partial G} & \frac{\partial f_1}{\partial S} \\ \frac{\partial f_2}{\partial T} & \frac{\partial f_2}{\partial G} & \frac{\partial f_2}{\partial S} \\ \frac{\partial f_3}{\partial T} & \frac{\partial f_3}{\partial G} & \frac{\partial f_3}{\partial S} \end{pmatrix} = \begin{pmatrix} -K_2 & K_1 & 0 \\ K_3 & -K_1 - K_4 S & -K_4 G \\ K_5 & -K_4 S & -K_6 - K_4 G \end{pmatrix}.$$

$$J(T^*,G^*,S^*)=\begin{pmatrix}-K_2 & K_1 & 0\\ K_3 & -K_1-K_4S^* & -K_4G^*\\ K_5 & -K_4S^* & -K_6-K_4G^*\end{pmatrix}.$$

3- Stability study:

at (T^*,G^*,S^*):

$|J-\lambda I|=0.$

$$\left|\begin{pmatrix}-K_2 & K_1 & 0\\ K_3 & -K_1-K_4S^* & -K_4G^*\\ K_5 & -K_4S^* & -K_6-K_4G^*\end{pmatrix}-\lambda\begin{pmatrix}1&0&0\\0&1&0\\0&0&1\end{pmatrix}\right|=0.$$

$$\left|\begin{pmatrix}-K_2 & K_1 & 0\\ K_3 & -K_1-K_4S^* & -K_4G^*\\ K_5 & -K_4S^* & -K_6-K_4G^*\end{pmatrix}-\begin{pmatrix}\lambda&0&0\\0&\lambda&0\\0&0&\lambda\end{pmatrix}\right|=0.$$

$$\begin{vmatrix}-K_2-\lambda & K_1 & 0\\ K_3 & -K_1-K_4S^*-\lambda & -K_4G^*\\ K_5 & -K_4S^* & -K_6-K_4G^*-\lambda\end{vmatrix}=0.$$

$(-K_2-\lambda)[(-K_1-K_4S^*-\lambda)(-K_6-K_4G^*-\lambda)-(-K_4G^*)(-K_4S^*)]-(K_1)[K_3(-K_6-K_4G^*-\lambda)]+$
$(K_4K_5G^*)=0.$

$(-K_2-\lambda)[(K_1+K_4S^*+\lambda)(K_6+K_4G^*+\lambda)-{K_4}^2G^*S^*]-(K_1)[-K_3(K_6+K_4G^*+\lambda)]+$
$(K_4K_5G^*)=0.$

put $x=K_1+K_4S^*, y=K_6+K_4G^*, z={K_4}^2G^*S^*, h=K_4K_5G^*$.

$(-K_2-\lambda)[(x+\lambda)(y+\lambda)-z]-(K_1)[-K_3(y+\lambda)]+h=0.$

$(-K_2-\lambda)[(xy+x\lambda+y\lambda+\lambda^2)-z]+(K_1K_3y+K_1K_3\lambda-K_1h)=0.$

$(-K_2-\lambda)[(xy+(x+y)\lambda+\lambda^2)-z]+(K_1K_3(y+\lambda)-K_1h)=0.$

$-K_2xy-K_2(x+y)\lambda-K_2\lambda^2+K_2z-xy\lambda-(x+y)\lambda^2-\lambda^3+z\lambda+K_1K_3y+K_1K_3\lambda-K_1h=0.$

$-\lambda^3-((x+y)+K_2)\lambda^2-(K_2(x+y)+xy-z-K_1K_3)\lambda-(K_2xy-K_2z-K_1K_3y+K_1h)=0.$

$\lambda^3+(x+y+K_2)\lambda^2+(K_2x+K_2y+xy-z-K_1K_3)\lambda+(K_2xy-K_2z-K_1K_3y+K_1h)=0.$

$\lambda^3+((K_1+K_4S^*)+(K_6+K_4G^*)+K_2)\lambda^2+(K_2(K_1+K_4S^*)+K_2(K_6+K_4G^*)+((K_1+K_4S^*)(K_6+K_4G^*)-$
$({K_4}^2G^*S^*)-K_1K_3)\lambda+((K_2(K_1+K_4S^*)(K_6+K_4G^*)-K_2{K_4}^2G^*S^*-K_1K_3(K_6+K_4G^*)+K_1(K_4K_5G^*))=0.$

$\lambda^3+(K_1+K_4S^*+K_6+K_4G^*+K_2)\lambda^2+(K_2K_1+K_2K_4S^*+K_2K_6+K_2K_4G^*+K_1K_6+K_1K_4G^*+K_6K_4S^*+$
$K_4{}^2G^*S^*-K_4{}^2G^*S^*-K_1K_3)\lambda+(K_1K_2K_6+K_1K_2K_4G^*+K_6K_2K_4S^*+K_2K_4{}^2G^*S^*-K_2K_4{}^2G^*S^*-K_1K_3K_6-$
$K_1K_3K_4G^*+K_1K_4K_5G^*)=0.$

$\lambda^3+(K_1+K_4S^*+K_6+K_4G^*+K_2)\lambda^2+(K_2K_1+K_2K_4S^*+K_2K_6+K_2K_4G^*+K_1K_6+K_1K_4G^*+K_6K_4S^*-$
$K_1K_3)\lambda+(K_1K_2K_6+K_1K_2K_4G^*+K_6K_2K_4S^*-K_1K_3K_6-K_1K_3K_4G^*+K_1K_4K_5G^*)=0.$

$\lambda^3+(K_1+K_4(\frac{K_1(K_3-K_2)}{K_2K_4})+K_6+K_4(\frac{K_6(K_3-K_2)}{K_4(K_5-K_3+K_2)})+K_2)\lambda^2+(K_2K_1+K_2K_4(\frac{K_1(K_3-K_2)}{K_2K_4})+K_2K_6+$
$K_2K_4\left(\frac{K_6(K_3-K_2)}{K_4(K_5-K_3+K_2)}\right)+K_1K_6+K_1K_4\left(\frac{K_6(K_3-K_2)}{K_4(K_5-K_3+K_2)}\right)+K_6K_4\left(\frac{K_1(K_3-K_2)}{K_2K_4}\right)-K_1K_3)\lambda+(K_1K_2K_6+$
$K_1K_2K_4(\frac{K_6(K_3-K_2)}{K_4(K_5-K_3+K_2)})+K_6K_2K_4(\frac{K_1(K_3-K_2)}{K_2K_4})-K_1K_3K_6-K_1K_3K_4(\frac{K_6(K_3-K_2)}{K_4(K_5-K_3+K_2)})+$
$K_1K_4K_5(\frac{K_6(K_3-K_2)}{K_4(K_5-K_3+K_2)}))=0.$

$\lambda^3+(K_1+(\frac{K_1(K_3-K_2)}{K_2})+K_6+(\frac{K_6(K_3-K_2)}{(K_5-K_3+K_2)})+K_2)\lambda^2+(K_2K_1+K_1(K_3-K_2)+K_2K_6+K_2\left(\frac{K_6(K_3-K_2)}{(K_5-K_3+K_2)}\right)+$
$K_1K_6+K_1\left(\frac{K_6(K_3-K_2)}{(K_5-K_3+K_2)}\right)+K_6\left(\frac{K_1(K_3-K_2)}{K_2}\right)-K_1K_3)\lambda+(K_1K_2K_6+K_1K_2(\frac{K_6(K_3-K_2)}{(K_5-K_3+K_2)})+K_6K_1(K_3-K_2)-$
$K_1K_3K_6-K_1K_3(\frac{K_6(K_3-K_2)}{(K_5-K_3+K_2)})+K_1K_5(\frac{K_6(K_3-K_2)}{(K_5-K_3+K_2)}))=0.$

$.\lambda^3+(K_1+(\frac{K_1(K_3-K_2)}{K_2})+K_6+(\frac{K_6(K_3-K_2)}{(K_5-K_3+K_2)})+K_2)\lambda^2+(K_1K_3+K_2K_6+K_2\left(\frac{K_6(K_3-K_2)}{(K_5-K_3+K_2)}\right)+K_1K_6+$
$K_1\left(\frac{K_6(K_3-K_2)}{(K_5-K_3+K_2)}\right)+K_6\left(\frac{K_1(K_3-K_2)}{K_2}\right)-K_1K_3)\lambda+(K_1K_2K_6+K_1K_2(\frac{K_6(K_3-K_2)}{(K_5-K_3+K_2)}-K_6K_1K_2-$

$$K_1K_3\left(\frac{K_6(K_3-K_2)}{(K_5-K_3+K_2)}\right)+K_1K_5\left(\frac{K_6(K_3-K_2)}{(K_5-K_3+K_2)}\right))=0\,.$$

$$a_3=1>0.$$

$$a_2=K_1+\left(\frac{K_1(K_3-K_2)}{K_2}\right)+K_6+\left(\frac{K_6(K_3-K_2)}{(K_5-K_3+K_2)}\right)+K_2.$$

$$a_1=K_1K_3+K_2K_6+K_2\left(\frac{K_6(K_3-K_2)}{(K_5-K_3+K_2)}\right)+K_1K_6+K_1\left(\frac{K_6(K_3-K_2)}{(K_5-K_3+K_2)}\right)+K_6\left(\frac{K_1(K_3-K_2)}{K_2}\right)-K_1K_3.$$

$$a_0=K_1K_2K_6+K_1K_2\left(\frac{K_6(K_3-K_2)}{(K_5-K_3+K_2)}\right)-K_6K_1K_2-K_1K_3\left(\frac{K_6(K_3-K_2)}{(K_5-K_3+K_2)}\right)+K_1K_5\left(\frac{K_6(K_3-K_2)}{(K_5-K_3+K_2)}\right).$$

If $a_i>0, i=0,1,2$ *and* $a_2a_1-a_3a_0>0$

Then by the Routh-Hurwitz criterion, the system at (T^*,G^*,S^*) is stable.

Despite our continued efforts in analyzing the system, we were unable to reach a consistent result or a clear analytical solution. We faced multiple challenges that complicated the results, making it difficult to determine the system's behavior definitively. We therefore intend to resort to numerical methods as an alternative means to explore the system dynamics more precisely. By using numerical solutions, we hope to gain deeper insights into the stability of the system and its behaviors under different conditions, which may help us understand complex phenomena that we have not been able to analyze Figure 1.

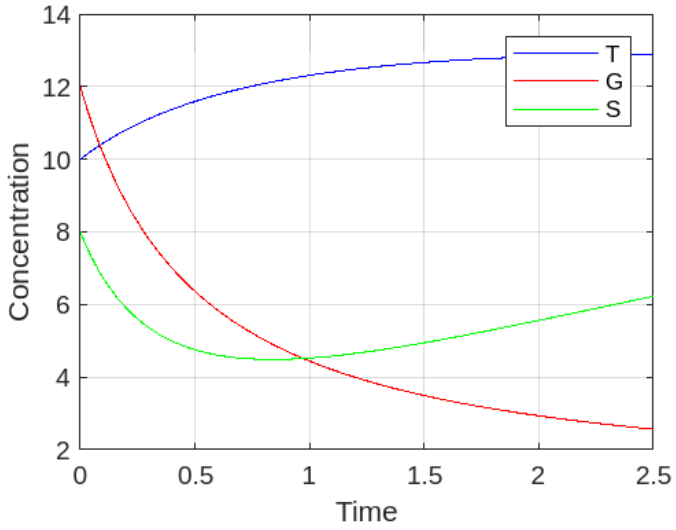

Figure: (1). intracellular viral kinetics**;** $K_1=0.5, K_2=0.1. K_3=0.3, K_4=0.2, K_5=0.1, K_6=0.4.$

Note that the viral template *T* appears concave upward, and this indicates that the amount of template increases over time, which indicates stability in growth with increasing template production. Being concave upward, this means that the increase in the amount of *T* accelerates over time, which reflects a positive dynamic in the reproduction of the virus.

As for the viral genome, it decreases over time as it is concave downwards. This decrease also indicates consumption or degradation of the viral genome. This dynamic may reflect an interaction process between the genome and the target cells. As for the amount of structural proteins, it decreases over time, and this indicates that the structural proteins are either used to build new viruses or are broken down due to cell processes. Based on the results extracted from the drawing, the system appears unstable. This is based on the data that was entered.

In fact, viruses and the host cell are exposed to random fluctuations. These fluctuations can affect the rates of interaction and reproduction. To provide a deeper understanding of how random factors

affect the movement of the virus inside cells, we can create a stochastic model that takes into account random factors and provides a better understanding of how viruses evolve and become resistant. For treatments or vaccines.

The stochastic model for the system

1- Probabilities associated with changes in the model, Table 2.

Table: (2). Probabilities associated with changes in the model

Changes, Δx_i	Probability, p_i
$(1,-1,0)^{tr}$.	$K_1 G\,\Delta t$.
$(-1,0,0)^{tr}$.	$K_2 T\,\Delta t$.
$(0,1,0)^{tr}$.	$K_3 T\,\Delta t$.
$(0,-1,-1)^{tr}$.	$K_4 GS\Delta t$.
$(0,0,1)^{tr}$.	$K_5 T\Delta t$.
$(0,0,-1)^{tr}$.	$K_6 S\Delta t$.

2- The expectation $E(\Delta x) = \sum_{i=1}^{6} p_i \Delta x_i$ is 3×1 matrix, the expectation can be expressed as follows.

$$E(\Delta x) = \sum_{i=1}^{6} p_i \Delta x_i = p_1\Delta x_1 + p_2\Delta x_2 + p_3\Delta x_3 + p_4\Delta x_4 + p_5\Delta x_5 + p_6\Delta x_6 ,$$

$$E(\Delta x) = \sum_{i=1}^{6} p_i \Delta x_i = K_1 G\begin{pmatrix}1\\-1\\0\end{pmatrix} + K_2 T\begin{pmatrix}-1\\0\\0\end{pmatrix} + K_3 T\begin{pmatrix}0\\1\\0\end{pmatrix} + K_4 GS\begin{pmatrix}0\\-1\\-1\end{pmatrix} + K_5 T\begin{pmatrix}0\\0\\1\end{pmatrix} +$$

$$K_6 S\begin{pmatrix}0\\0\\-1\end{pmatrix}.$$

$$E(\Delta x) = \begin{pmatrix}K_1 G\\-K_1 G\\0\end{pmatrix} + \begin{pmatrix}-K_2 T\\0\\0\end{pmatrix} + \begin{pmatrix}0\\K_3 T\\0\end{pmatrix} + \begin{pmatrix}0\\-K_4 GS\\-K_4 GS\end{pmatrix} + \begin{pmatrix}0\\0\\K_5 T\end{pmatrix} + \begin{pmatrix}0\\0\\-K_6 S\end{pmatrix}.$$

$$E(\Delta x) = \begin{pmatrix}K_1 G - K_2 T\\-K_1 G + K_3 T - K_4 GS\\-K_4 GS + K_5 T - K_6 S\end{pmatrix}\Delta t.$$

3- The covariance matrix, can be expressed as follows

$$E(\Delta x(\Delta x)^T) = \sum_{i=1}^{6} p_i \Delta x_i\,(\Delta x_i)^T.$$

$$= p_1\Delta x_1(\Delta x_1)^T + p_2\Delta x_2(\Delta x_2)^T + p_3\Delta x_3(\Delta x_3)^T + p_4\Delta x_4(\Delta x_4)^T +$$

$$p_5\Delta x_5(\Delta x_5)^T + p_6\Delta x_6(\Delta x_6)^T.$$

$$E(\Delta x(\Delta x)^T) = \begin{pmatrix}K_1 G\\-K_1 G\\0\end{pmatrix}(1\ -1\ \ 0) + \begin{pmatrix}-K_2 T\\0\\0\end{pmatrix}(-1\ 0\ \ 0) + \begin{pmatrix}0\\K_3 T\\0\end{pmatrix}(0\ 1\ \ 0) +$$

$$\begin{pmatrix}0\\-K_4 GS\\-K_4 GS\end{pmatrix}(0\ -1\ -1) + \begin{pmatrix}0\\0\\K_5 T\end{pmatrix}(0\ 0\ \ 1) + \begin{pmatrix}0\\0\\-K_6 S\end{pmatrix}(0\ 0\ -1)\,.$$

$$E(\Delta x(\Delta x)^T) = \begin{pmatrix}K_1 G & -K_1 G & 0\\-K_1 G & K_1 G & 0\\0 & 0 & 0\end{pmatrix} + \begin{pmatrix}K_2 T & 0 & 0\\0 & 0 & 0\\0 & 0 & 0\end{pmatrix} + \begin{pmatrix}0 & 0 & 0\\0 & K_3 T & 0\\0 & 0 & 0\end{pmatrix} +$$

$$\begin{pmatrix}0 & 0 & 0\\0 & K_4 GS & K_4 GS\\0 & K_4 GS & K_4 GS\end{pmatrix} + \begin{pmatrix}0 & 0 & 0\\0 & 0 & 0\\0 & 0 & K_5 T\end{pmatrix} + \begin{pmatrix}0 & 0 & 0\\0 & 0 & 0\\0 & 0 & K_6 S\end{pmatrix}.$$

$$E(\Delta x(\Delta x)^T) = \begin{pmatrix} K_1G + K_2T & -K_1G & 0 \\ -K_1G & K_1G + K_3T + K_4GS & K_4GS \\ 0 & K_4GS & K_4GS + K_5T + K_6S \end{pmatrix}\Delta t.$$

$$E(\Delta x(\Delta x)^T) = \begin{pmatrix} K_1G + K_2T & -K_1G & 0 \\ -K_1G & K_1G + K_3T + K_4GS & K_4GS \\ 0 & K_4GS & K_4GS + K_5T + K_6S \end{pmatrix}\Delta t = V\Delta t.$$

4- Formulate the stochastic system as

$$dX(t) = f(X(t),t)dt + h(X(t),t)dW(t).$$

where

$$dX(t) = \begin{bmatrix} dT_t \\ dG_t \\ dS_t \end{bmatrix}, f(X(t),t) = \left[\frac{E(\Delta X)}{\Delta t}\right], h(X(t),t) = \sqrt{V} \text{ and } dW(t) = \begin{bmatrix} dW_1(t) \\ dW_2(t) \\ dW_3(t) \end{bmatrix}.$$

$$\begin{pmatrix} dT_t \\ dG_t \\ dS_t \end{pmatrix} = \begin{pmatrix} K_1G - K_2T \\ -K_1G + K_3T - K_4GS \\ -K_4GS + K_5T - K_6S \end{pmatrix}dt +$$

$$\begin{pmatrix} \sqrt{(K_1G + K_2T)} & -\sqrt{K_1G} & 0 \\ -\sqrt{K_1G} & \sqrt{(K_1G + K_3T + K_4GS)} & \sqrt{K_4GS} \\ 0 & \sqrt{K_4GS} & \sqrt{(K_4GS + K_5T + K_6S)} \end{pmatrix}.\begin{pmatrix} dW_1(t) \\ dW_2(t) \\ dW_3(t) \end{pmatrix}.$$

$$\begin{cases} dT_t = (K_1G_t - K_2T_t)dt + \sqrt{(K_1G_t + K_2T_t)}\, dW_1(t) - \sqrt{K_1G_t}dW_2(t). \\ dG_t = (-K_1G_t + K_3T_t - K_4G_tS_t)dt - \sqrt{K_1G_t}dW_1(t) + \sqrt{(K_1G_t + K_3T_t + K_4G_tS_t)}dW_2(t) + \sqrt{K_4G_tS_t}dW_3(t) \\ dS_t = (-K_4G_tS_t + K_5T_t - K_6S_t)dt + \sqrt{K_4G_tS_t}\, dW_2(t) + \sqrt{(K_4G_tS_t + K_5T_t + K_6S_t)}dW_3(t). \end{cases}.$$

The equivalent system for the former system.

The diffusion matrix G of dimension 3×6 is

$$G = \begin{pmatrix} \sqrt{K_1G} & -\sqrt{K_2T} & 0 & 0 & 0 & 0 \\ -\sqrt{K_1G} & 0 & \sqrt{K_3T} & -\sqrt{K_4GS} & 0 & 0 \\ 0 & 0 & 0 & -\sqrt{K_4GS} & \sqrt{K_5T} & -\sqrt{K_6S} \end{pmatrix}.$$

$$dX(t) = f(X(t),t)dt + g(X(t),t)dW(t).$$

Where:

$$dX(t) = \begin{bmatrix} dT_t \\ dG_t \\ dS_t \end{bmatrix}, f(X(t),t) = \left[\frac{E(\Delta X)}{\Delta t}\right], g(X(t),t) = G \text{ and } dW(t) = \begin{bmatrix} dW_1(t) \\ dW_2(t) \\ dW_3(t) \\ dW_4(t) \\ dW_5(t) \\ dW_6(t) \end{bmatrix}.$$

Thus, the system takes the following form:

$$\begin{pmatrix} dT_t \\ dG_t \\ dS_t \end{pmatrix} = \begin{pmatrix} K_1G - K_2T \\ -K_1G + K_3T - K_4GS \\ -K_4GS + K_5T - K_6S \end{pmatrix}dt + \begin{pmatrix} \sqrt{K_1G} & -\sqrt{K_2T} & 0 & 0 & 0 & 0 \\ -\sqrt{K_1G} & 0 & \sqrt{K_3T} & -\sqrt{K_4GS} & 0 & 0 \\ 0 & 0 & 0 & -\sqrt{K_4GS} & \sqrt{K_5T} & -\sqrt{K_6S} \end{pmatrix}.\begin{pmatrix} dW_1(t) \\ dW_2(t) \\ dW_3(t) \\ dW_4(t) \\ dW_5(t) \\ dW_6(t) \end{pmatrix}.$$

$$\begin{cases} dT_t = (K_1G - K_2T)dt + \sqrt{K_1G}\, dW_1(t) - \sqrt{K_2T} dW_2(t) . \\ dG_t = (-K_1G + K_3T - K_4GS)dt - \sqrt{K_1G}\, dW_1(t) + \sqrt{K_3T} dW_3(t) - \sqrt{K_4GS}\, dW_4(t). \\ dS_t = (-K_4GS + K_5T - K_6S)dt - \sqrt{K_4GS}\, dW_4(t) + \sqrt{K_5T}\, dW_5(t) - \sqrt{K_6S} dW_6(t). \end{cases} .$$

A stochastic drawing shows how the viral template interacts with the genome and structural proteins in the system. While the viral template remains stable, the genome and proteins exhibit fluctuations that reflect response to random factors and changes in the cellular environment, Figure 2 and Figure3.

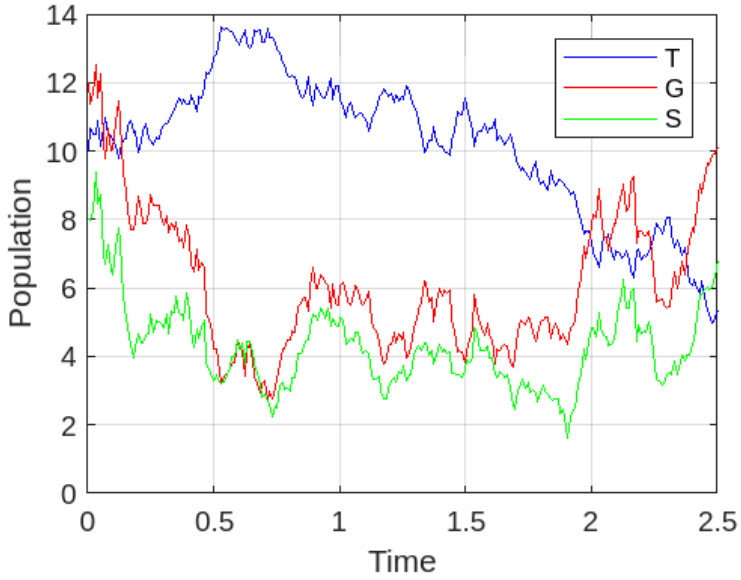

Figure: (2). intracellular viral kinetics**;** $K_1 = 0.5, K_2 = 0.1. K_3 = 0.3, K_4 = 0.2, K_5 = 0.1, K_6 = 0.4$.

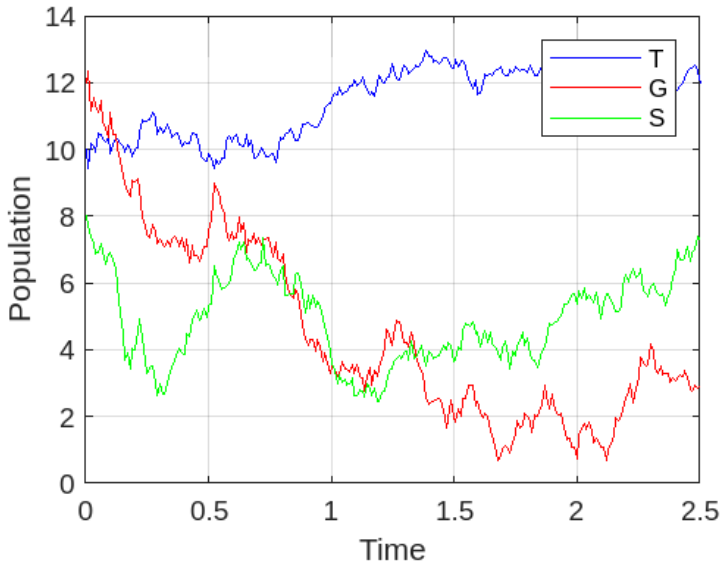

Figure: (3). intracellular viral kinetics**;** $K_1 = 0.5, K_2 = 0.1. K_3 = 0.3, K_4 = 0.2, K_5 = 0.1, K_6 = 0.4$.

Numerical Representation

The deterministic and stochastic systems solved numerically, (see the Appendix), their solutions represented using MATAB tools, and hence the following figures were obtained.

CONCLUSION

In this study, the movement of the virus inside cells was analyzed, using an advanced mathematical model, where the stability of the virus's dynamics was studied and the random model of the virus's movement inside the cells was discovered, which allowed us to better understand the dynamics of the virus and how the system works, and its influence on random factors. This study provides important insights that contribute to the development of effective treatment strategies.

The study can also be used to analyze different types of viruses, such as the immunodeficiency virus, influenza virus, coronavirus, etc., which confirms the importance of model in viral researches.

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Appendix

Code of intracellular viral kinetics model (Deterministic model):

```
% Parameters
K1 = 0.5; % Parameter K1
K2 = 0.1; % Parameter K2
K3 = 0.3; % Parameter K3
K4 = 0.2; % Parameter K4
K5 = 0.4; % Parameter K5
```

```
K6 = 0.1; % Parameter K6

% ODE system
ode_system = @(t, Y) [
    K1 * Y(2) - K2 * Y(1);              % dT/dt
    K3 * Y(1) - K1 * Y(2) - K4 * Y(2) * Y(3); % dG/dt
    K5 * Y(1) - K6 * Y(3) - K4 * Y(2) * Y(3)  % dS/dt
];

% Initial conditions
T0 = 10; % Initial condition for T
G0 = 12; % Initial condition for G
S0 = 8; % Initial condition for S
Y0 = [T0; G0; S0];

% Time span
tspan = [0 2.5];

% Solve ODE
[t, Y] = ode45(ode_system, tspan, Y0);

% Plot results
figure;
plot(t, Y(:,1), '-b', 'DisplayName', 'T');
hold on;
plot(t, Y(:,2), '-r', 'DisplayName', 'G');
plot(t, Y(:,3), '-g', 'DisplayName', 'S');
xlabel('Time');
ylabel('Concentration');
legend;
grid on;
hold off;
```

Code of intracellular viral kinetics model (Stochastic model):

```
% Parameters
K1 = 0.5; % Parameter K1
K2 = 0.1; % Parameter K2
K3 = 0.3; % Parameter K3
K4 = 0.2; % Parameter K4
K5 = 0.4; % Parameter K5
K6 = 0.1; % Parameter K6

% Initial conditions
T0 = 10; % Initial condition for T
G0 = 12; % Initial condition for G
S0 = 8; % Initial condition for S
total_time = 2.5;  % Total time
dt = 0.01; % Time step
N = total_time / dt; % Number of time steps
```

```
% Preallocate arrays
T_values = zeros(1, N);
G_values = zeros(1, N);
S_values = zeros(1, N);
t = linspace(0, total_time, N);
T_values(1) = T0;
G_values(1) = G0;
S_values(1) = S0;

% Simulation using Euler-Maruyama method
for i = 1:N-1
    % Deterministic part
    dT_det = (K1 * G_values(i) - K2 * T_values(i)) * dt;
    dG_det = (-K1 * G_values(i) + K3 * T_values(i) - K4 * G_values(i) * S_values(i)) * dt;
    dS_det = (-K4 * G_values(i) * S_values(i) + K5 * T_values(i) - K6 * S_values(i)) * dt;

    % Stochastic part
    dW1 = sqrt(dt) * randn;
    dW2 = sqrt(dt) * randn;
    dW3 = sqrt(dt) * randn;

    dT_sto = sqrt(K1 * G_values(i) + K2 * T_values(i)) * dW1 - sqrt(K1 * G_values(i)) * dW2;
    dG_sto = -sqrt(K1 * G_values(i)) * dW1 + sqrt(K1 * G_values(i) + K3 * T_values(i) + K4 *
G_values(i) * S_values(i)) * dW2 + sqrt(K4 * G_values(i) * S_values(i)) * dW3;
    dS_sto = sqrt(K4 * G_values(i) * S_values(i)) * dW2 + sqrt(K4 * G_values(i) * S_values(i) + K5
* T_values(i) + K6 * S_values(i)) * dW3;

    % Update populations
    T_values(i+1) = T_values(i) + dT_det + dT_sto;
    G_values(i+1) = G_values(i) + dG_det + dG_sto;
    S_values(i+1) = S_values(i) + dS_det + dS_sto;

    % Ensure populations remain non-negative
    T_values(i+1) = max(T_values(i+1), 0);
    G_values(i+1) = max(G_values(i+1), 0);
    S_values(i+1) = max(S_values(i+1), 0);
end

% Plot results
figure;
plot(t, T_values, '-b', 'DisplayName', 'T');
hold on;
plot(t, G_values, '-r', 'DisplayName', 'G');
plot(t, S_values, '-g', 'DisplayName', 'S');
xlabel('Time');
ylabel('Population');
legend;
grid on;
```

```
hold off;
```

Code of intracellular viral kinetics model (equevelant Stochastic model):

```
% Parameters
K1 = 0.5; % Parameter K1
K2 = 0.1; % Parameter K2
K3 = 0.3; % Parameter K3
K4 = 0.2; % Parameter K4
K5 = 0.4; % Parameter K5
K6 = 0.1; % Parameter K6

% Initial conditions
T0 = 10; % Initial condition for T
G0 = 12; % Initial condition for G
S0 = 8; % Initial condition for S
total_time = 2.5;  % Total time
dt = 0.01; % Time step
N = total_time / dt; % Number of time steps

% Preallocate arrays
T_values = zeros(1, N);
G_values = zeros(1, N);
S_values = zeros(1, N);
t = linspace(0, total_time, N);
T_values(1) = T0;
G_values(1) = G0;
S_values(1) = S0;

% Simulation using Euler-Maruyama method
for i = 1:N-1
    % Deterministic part
    dT_det = (K1 * G_values(i) - K2 * T_values(i)) * dt;
    dG_det = (-K1 * G_values(i) + K3 * T_values(i) - K4 * G_values(i) * S_values(i)) * dt;
    dS_det = (-K4 * G_values(i) * S_values(i) + K5 * T_values(i) - K6 * S_values(i)) * dt;

    % Stochastic part
    dW1 = sqrt(dt) * randn;
    dW2 = sqrt(dt) * randn;
    dW3 = sqrt(dt) * randn;
    dW4 = sqrt(dt) * randn;
    dW5 = sqrt(dt) * randn;
    dW6 = sqrt(dt) * randn;

    dT_sto = sqrt(K1 * G_values(i)) * dW1 - sqrt(K2 * T_values(i)) * dW2;
    dG_sto = -sqrt(K1 * G_values(i)) * dW1 + sqrt(K3 * T_values(i)) * dW3 - sqrt(K4 * G_values(i)
* S_values(i)) * dW4;
    dS_sto = -sqrt(K4 * G_values(i) * S_values(i)) * dW4 + sqrt(K5 * T_values(i)) * dW5 - sqrt(K6
* S_values(i)) * dW6;
```

```
    % Update populations
    T_values(i+1) = T_values(i) + dT_det + dT_sto;
    G_values(i+1) = G_values(i) + dG_det + dG_sto;
    S_values(i+1) = S_values(i) + dS_det + dS_sto;

    % Ensure populations remain non-negative
    T_values(i+1) = max(T_values(i+1), 0);
    G_values(i+1) = max(G_values(i+1), 0);
    S_values(i+1) = max(S_values(i+1), 0);
end

% Plot results
figure;
plot(t, T_values, '-b', 'DisplayName', 'T');
hold on;
plot(t, G_values, '-r', 'DisplayName', 'G');
plot(t, S_values, '-g', 'DisplayName', 'S');
xlabel('Time');
ylabel('Population');
legend;
grid on;
hold off;
```