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Fractional-Order Modeling of Viral Load Dynamics: Analyzing the Long-term Memory Effects in Chronic Infections

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Abstract

Classical integer-order differential equations have long been used to provide mathematical models of viral infections, and assume that the dynamics of the system only depend on its current state. This assumption, however, might not be adequate in the case of chronic infections, where the dynamics between the virus and the host immune response tends to be delayed and history dependent. Under these circumstances, history may affect the present dynamics, which results in patterns that are not readily modeled using conventional models.

In this paper, we suggest the use of a fractional-order model to model the dynamics of viral load in chronic infection with specific focus on the inclusion of the long-term memory effects. Caputo fractions are used to formulate the model, so that the cumulative effect of the previous states can be considered by the system. We look at the qualitative aspects of the model, such as existence and stability of equilibrium points, and investigate the changes in system behavior under variation of the fractional order.

The fractional-order model and classical one are compared by performing numerical simulations. It is noted that the fractional framework offers a more adaptable and, in certain instances, more realistic account of viral persistence and gradual immune adjustment. In particular, the model can recreate extended transient behavior and more regular decay behavior, commonly observed in clinical experiments, but challenging to model with integer-order methods.

Despite the fact that the model is a simplistic representation of complicated biological phenomena, the findings indicate that fractional-order dynamics may provide valuable insights into the behavior of chronic infections. The article adds to the current interest in modeling approaches that rely on memory and emphasize on their possible applicability to enhance our knowledge of disease progression and therapeutic response over time.

Keywords: Fractional-order differential equations, Viral load dynamics, Chronic infections, Memory effects, Caputo derivative, Mathematical biology, Stability analysis, Numerical simulation, Immune response modeling

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1. Introduction

Viruses that are chronic are also a significant issue in international health, especially in human immunodeficiency virus (HIV), hepatitis B and hepatitis C. These are infections that are characterized by their long-term presence in the host and complex interaction between viral replication and immune response. Math modelling has gained relevance in the last couple of decades to learn more about these dynamics, in particular, predicting the evolution of viral load and the assessment of treatment measures. Classical models, founded mostly on systems of integer-order differential equations, have helped to give insightful understanding of the mechanism of infections, such as the interactions between the virus and the host and the workings of the immune system. Although useful, traditional integer-order models assume the simplifying factor that the rate of change of the system is only dependent on the current state of the system. Although this assumption simplifies the analysis, not all aspects of biology are always captured in this assumption. Chronic infections have delayed, adaptive, and past exposure to viral antigens. As an example, the current behavior of the system is influenced by immune memory, latency periods as well as history of treatment. This has led to the possibility that models that neglect these memory effects can miss meaningful long-term dynamics of the viral dynamics. Fractional calculus has become an attractive method in the past few years, as a model of systems with memory and hereditary behavior. In contrast to classical derivatives, fractional-order derivatives are by their nature non-local, i.e. they use the full history of the state variable in the present rate of change. This characteristic renders them especially appropriate in biological systems in which the previous interactions determine the current results. Fractional-order models have been used in epidemiology, viscoelasticity, and control systems and have shown better flexibility and agreement with experimental results in a few instances (Podlubny, 1999; Diethelm, 2010). Fractional-order modeling has begun to attract attention in the dynamics of infectious diseases, but remains relatively uncommon. A few researchers have studied fractional epidemic spread models, and demonstrated that memory effects can profoundly modify the dynamics of transmission and stability behavior. Nevertheless, a gap remains evident in terms of translating these strategies directly to within-host dynamics of viral load, particularly in the environment of chronic infections. A lot of the existing research is either population level models or does not explicitly examine the consequences of fractional order to long-term system behavior. The other problem is that the majority of traditional viral dynamics models have instantaneous immune response and constant parameters, which can be not true in real clinical conditions. The immune activation variability, variability in drug response, and variability in viral mutation bring about further complexity that may be more readily modeled with fractional-order formulations. The inclusion of memory in the model can enable the model to describe delayed immune reactions, and slow processes of adaptation in a more realistic way. The current paper is driven by these shortcomings and therefore introduces a mathematical model (fractional-order) to explain the dynamic of viral loads in chronic infections. The model is formulated by applying the Caputo

definition of fractional derivatives, which is typically applied in practical issues because it is compatible with initial conditions in the classical sense. The model can also model the various degrees of memory effect in the system by integrating a fractional-order parameter. The key contributions of this work can be summed up as follows. We then develop a fractional-order model of viral loads dynamics, which builds on the classical integer-order. Secondly, we examine the qualitative system behavior such as the equilibrium points and their stability at various fractional orders. Third, we conduct a numerical study to explore the role of memory effects in the long-term dynamics and compare the outcomes with the results of classic models. Although the proposed model does not strive to capture all the biological complexities, it will be a step towards more realistic modeling of chronic infections. The results have the potential to provide valuable information on the influence of memory-dependent processes on the disease progression and can possibly be used in future research on the options of optimizing the treatment and controlling the disease.

2. Abbreviations

Abbreviation	Full Form
HIV	Human Immunodeficiency Virus
HBV	Hepatitis B Virus
HCV	Hepatitis C Virus
ODE	Ordinary Differential Equation
FODE	Fractional-Order Differential Equation
PDE	Partial Differential Equation
Caputo FD	Caputo Fractional Derivative
α\alphaα	Fractional Order Parameter
VLD	Viral Load Dynamics
CTL	Cytotoxic T Lymphocytes
RHS	Right-Hand Side
LHS	Left-Hand Side
IC	Initial Condition
BC	Boundary Condition

3. Mathematical Model Formulation

3.1 Basic Assumptions

Before writing the equations, it is important to clarify what we are assuming. In biological modeling, not everything can be included, so some simplifications are necessary. At the same time, too many assumptions can make the model unrealistic. So we try to keep a balance here.

The model is developed under the following assumptions:

1.The host system consists of three main components:

a.Uninfected target cells

b.Infected cells

c.Free virus particles

2.Uninfected cells are produced at a constant rate and die naturally after some time.

3.Infection occurs when virus particles interact with healthy cells. This interaction is not instantaneous in real life, but we approximate it as a rate-based process.

4.Infected cells produce new virus particles, but there is a delay-like effect in this production (this is where memory becomes important).

5.The immune response is not explicitly modeled as a separate variable, but its effect is indirectly included in the removal rates.

6.The most important assumption in this work is that the system has memory, meaning that past states influence the present dynamics. This is incorporated using fractional-order derivatives.

3.2 Model Variables and Parameters

Let us define the variables used in the model:

- T(t)T(t)T(t): concentration of uninfected target cells at time ttt
- I(t)I(t)I(t): concentration of infected cells
- V(t)V(t)V(t): viral load (free virus particles)

3.3 Parameters:

- λ\lambdaλ: rate of production of healthy cells
- ddd: natural death rate of healthy cells
- β\betaβ: infection rate coefficient
- δ\deltaδ: death rate of infected cells
- ppp: rate of virus production by infected cells
- ccc: clearance rate of virus particles
- α∈(0,1]\alpha \in (0,1]α∈(0,1]: fractional order parameter

3.4 Classical Integer-Order Model (Baseline)

For comparison, we first recall the classical model:

dTdt=λ−dT−βTV\frac{dT}{dt} = \lambda - dT - \beta T VdtdT =λ−dT−βTV dIdt=βTV−δI\frac{dI}{dt} = \beta T V - \delta IdtdI =βTV−δI dVdt=pI−cV\frac{dV}{dt} = pI - cVdtdV =pI−cV

This system assumes that the rate of change at time ttt depends only on the state at that exact time. While useful, it does not include any past influence.

3.5 Fractional-Order Model Formulation

To incorporate memory effects, we replace the classical derivatives with fractional derivatives of order α\alphaα. Using the Caputo definition, the proposed model becomes:

DtαT(t)=λ−dT(t)−βT(t)V(t)D_t^{\alpha} T(t) = \lambda - dT(t) - \beta T(t)V(t)Dtα T(t)=λ−dT(t)−βT(t)V(t)

DtαI(t)=βT(t)V(t)−δI(t)D_t^{\alpha} I(t) = \beta T(t)V(t) - \delta I(t)Dtα I(t)=βT(t)V(t)−δI(t)

DtαV(t)=pI(t)−cV(t)D_t^{\alpha} V(t) = pI(t) - cV(t)Dtα V(t)=pI(t)−cV(t)

where DtαD_t^{\alpha}Dtα denotes the Caputo fractional derivative of order α\alphaα.

One thing that is interesting here (and honestly, a bit tricky also) is that the derivative at time ttt now depends on all previous values of T,I,VT, I, VT,I,V. So even if the system looks similar to the classical one, the behavior can be quite different.

3.6 Interpretation of the Fractional Order (α)

The parameter α\alphaα controls the strength of memory:

If α=1\alpha = 1α=1: the model reduces to the classical case

If α<1\alpha < 1α<1: memory effects are present

Smaller α\alphaα → stronger memory influence

In biological terms, this could represent:

Delayed immune activation

Persistence of infection history

Gradual adaptation of the host system

It is not always easy to assign a direct physical meaning to α\alphaα, and different interpretations exist in literature. So we treat it more like a tuning parameter that reflects memory intensity.

3.7 Initial Conditions

For fractional systems, initial conditions are still required, but they are interpreted slightly differently:

T(0)=T0,I(0)=I0,V(0)=V0T(0) = T_0, \quad I(0) = I_0, \quad V(0) = V_0T(0)=T0 ,I(0)=I0 ,V(0)=V0

These represent the initial state of the infection. However, due to memory effects, the system evolution will depend not only on these values but also on the fractional structure.

3.8 Model Discussion

Even though the equations look quite similar to the classical model, the introduction of fractional derivatives changes the system behavior in a non-trivial way. For example, solutions may evolve more slowly, and transient states can last longer.

Also, one limitation of this model is that immune response is not explicitly included. This was done to keep the system manageable, but it could be extended in future work.

4. Stability Analysis

4.1 Equilibrium Points

To understand the long-term behavior of the system, we first determine its equilibrium points. These are the states where the system does not change with time. For the fractional-order system, equilibrium points are obtained in the same way as in the classical case—by setting the derivatives equal to zero.

So, we consider:

0=λ−dT−βTV0 = \lambda - dT - \beta T V0=λ−dT−βTV

0=βTV−δI0 = \beta T V - \delta I0=βTV−δI

0=pI−cV0 = pI - cV0=pI−cV

From these equations, we can identify two biologically meaningful equilibrium points.

(i)Disease-Free Equilibrium (DFE)

This corresponds to the situation where no infection persists in the system (i.e., I=0I = 0I=0, V=0V = 0V=0).

Solving, we get:

T∗ =λd,I∗ =0,V∗ =0T^* = \frac{\lambda}{d}, \quad I^* = 0, \quad V^* = 0T∗ =dλ ,I∗ =0,V∗ =0

So, the disease-free equilibrium is:

E0=(λd,0,0)E_0 = \left(\frac{\lambda}{d}, 0, 0 \right)E0 =(dλ ,0,0)

This state represents a healthy system where only uninfected cells exist.

(ii)Endemic (Infected) Equilibrium

Now we consider the case where infection persists, so I≠0I \neq 0I =0, V≠0V \neq 0V =0. From:

pI=cV⇒V=pcIpI = cV \Rightarrow V = \frac{p}{c} IpI=cV⇒V=cp I

Substituting into the second equation:

βTV=δI\beta T V = \delta IβTV=δI βT(pcI)=δI\beta T \left(\frac{p}{c} I \right) = \delta IβT(cp I)=δI

Assuming I≠0I \neq 0I =0, we get:

T∗ =δcβpT^* = \frac{\delta c}{\beta p}T∗ =βpδc

Then substituting back, we can derive expressions for I∗ I^*I∗ and V∗ V^*V∗ (you can expand numerically later if needed).

So, the endemic equilibrium exists only when infection can sustain itself.

4.2 Basic Reproduction Number (Conceptual Insight)

Although not derived fully here, it is useful to define a threshold parameter:

R0=βλpdδcR_0 = \frac{\beta \lambda p}{d \delta c}R0 =dδcβλp

If R0<1R_0 < 1R0 <1: infection dies out

If R0>1R_0 > 1R0 >1: infection persists

This condition also determines whether the endemic equilibrium exists.

4.3 Jacobian Matrix of the System

To analyze stability, we compute the Jacobian matrix of the system.

J=[−d−βV0−βTβV−δβT0p−c]J = \begin{bmatrix} -d - \beta V & 0 & -\beta T \\ \beta V &

-\delta & \beta T \\ 0 & p & -c \end{bmatrix}J= −d−βVβV0 0−δp −βTβT−c

This matrix represents how small perturbations around equilibrium evolve.

4.4 Local Stability of Disease-Free Equilibrium

We evaluate the Jacobian at:

E0=(λd,0,0)E_0 = \left(\frac{\lambda}{d}, 0, 0 \right)E0 =(dλ ,0,0)

Substituting into the Jacobian:

V=0V = 0V=0

T=λdT = \frac{\lambda}{d}T=dλ

So the matrix becomes:

J(E0)=[−d0−βλd0−δβλd0p−c]J(E_0) = \begin{bmatrix} -d & 0 & -\frac{\beta

\lambda}{d} \\ 0 & -\delta & \frac{\beta \lambda}{d} \\ 0 & p & -c \end{bmatrix}J(E0)=

−d00 0−δp −dβλ dβλ −c

Now, stability depends on the eigenvalues of this matrix.

For fractional-order systems, the condition is slightly different from classical systems: The equilibrium is locally asymptotically stable if all eigenvalues λi\lambda_iλi satisfy

∣arg⁡(λi)∣>απ2|\arg(\lambda_i)| > \frac{\alpha \pi}{2}∣arg(λi)∣>2απ

This is an important point—fractional systems don't just depend on sign of eigenvalues, but also their argument.

4.5 Interpretation

When R0<1R_0 < 1R0 <1, the eigenvalues tend to satisfy the stability condition

So the disease-free state becomes stable

If R0>1R_0 > 1R0 >1, one eigenvalue crosses the boundary → instability

So basically, the infection starts to grow when the reproduction number exceeds unity.

4.6 Local Stability of Endemic Equilibrium

For the endemic equilibrium, the Jacobian becomes more complicated since all variables are non-zero.

Substituting (T* ,I* ,V*)(T^*, I^*, V^*)(T* ,I* ,V*) into the Jacobian gives a full matrix with coupled terms. The characteristic equation becomes nonlinear and is generally difficult to solve analytically.

Because of this, in many practical studies (and also here), stability is analyzed using:

Numerical eigenvalue computation

Parameter-based simulations Still, one general observation is:

When R0>1R_0 > 1R0 >1, the endemic equilibrium tends to be stable under certain parameter ranges

The fractional order α\alphaα affects how quickly the system approaches equilibrium

4.7 Effect of Fractional Order on Stability

Unlike classical systems:

Lower values of α\alphaα can slow down convergence

The system may take longer to reach steady state

Transient oscillations may persist longer

In some cases, even if the equilibrium is stable, the path to reach it looks very different compared to integer-order models. This is something we will see more clearly in the numerical results.

4.8 Remarks

Stability conditions in fractional systems are stricter than classical ones

Memory effects do not change equilibrium points, but they do affect stability behavior

Analytical solutions are not always easy, so numerical methods become important

One small limitation here is that we have not derived closed-form eigenvalues, but this is quite common in nonlinear biological systems.

5. Numerical Method

5.1 Need for Numerical Approximation

Unlike classical integer-order differential equations, fractional-order systems generally do not admit simple closed-form solutions. Even for relatively simple models, analytical solutions become difficult or sometimes not possible at all. Because of this, numerical methods are necessary to study the behavior of the system.

In this work, we use a predictor–corrector method, which is widely applied for solving fractional differential equations. One reason for choosing this method (apart from popularity) is that it provides a good balance between accuracy and computational effort. Also, it is relatively easier to implement compared to some other approaches.

5.2 Caputo Fractional Derivative

The model is based on the Caputo fractional derivative. In numerical form, it can be expressed as an integral equation:

x(t)=x(0)+1Γ(α)∫0t(t−τ)α−1f(τ,x(τ))dτx(t) = x(0) +

\frac{1}{\Gamma(\alpha)} \int_0^t (t-\tau)^{\alpha-1} f(\tau, x(\tau)) d\taux(t)=x(0)+Γ(α)1 ∫0t (t−τ)α−1f(τ,x(τ))dτ

This form is useful because it converts the differential equation into an integral equation, which can then be approximated numerically.

One thing to notice here is that the integral depends on all previous values of the function. So in computation, we cannot just use the current step—we need to store past values also. This increases computational cost a bit.

5.3 Time Discretization

Let us divide the time interval into equal steps:

tn=nh,n=0,1,2,…,Nt_n = nh, \quad n = 0,1,2,\dots,Ntn =nh,n=0,1,2,…,N

where:

hhh is the step size

NNN is total number of steps

The solution at each step depends on all previous steps, which again shows the memory effect.

5.4 Predictor–Corrector Scheme

The predictor–corrector method consists of two stages:

(i)Predictor Step (Explicit Approximation)

First, we estimate the solution at the next time step using known previous values:

xn+1(p)=x0+1Γ(α)∑j=0nbjf(tj,xj)x_{n+1}^{(p)} = x_0 +

\frac{1}{\Gamma(\alpha)} \sum_{j=0}^{n} b_j f(t_j, x_j)xn+1(p) =x0 +Γ(α)1 j=0∑n bj f(tj ,xj)

This gives a rough approximation. It is not very accurate, but it helps in the next step.

(ii)Corrector Step (Refined Approximation)

Then we refine the predicted value:

xn+1=x0+1Γ(α)(∑j=0najf(tj,xj)+an+1f(tn+1,xn+1(p)))x_{n+1} = x_0 +

\frac{1}{\Gamma(\alpha)} \left(\sum_{j=0}^{n} a_j f(t_j, x_j) + a_{n+1} f(t_{n+1}, x_{n+1}^{(p)}) \right)xn+1 =x0 +Γ(α)1 (j=0∑n aj f(tj ,xj)+an+1 f(tn+1 ,xn+1(p)))

Here:

aja_jaj , bjb_jbj are weight coefficients depending on α\alphaα

The corrector uses both past values and the predicted value

This two-step approach improves accuracy compared to using only an explicit scheme.

5.5 Application to the Viral Load Model

For our system:

T(t)T(t)T(t), I(t)I(t)I(t), and V(t)V(t)V(t) are updated at each time step

Each equation is solved using the same predictor–corrector framework So at each iteration:

1.Compute predicted values T(p),I(p),V(p)T^{(p)}, I^{(p)}, V^{(p)}T(p),I(p),V(p)

2.Use them to compute corrected values

3.Store results for next iteration

One practical issue we noticed (while implementing) is that computation time increases as the simulation progresses, because the memory term keeps growing. This is a known limitation of fractional methods.

5.6 Alternative: Grünwald–Letnikov Method

Another approach is the Grünwald–Letnikov (GL) method, which approximates the fractional derivative using finite differences:

Dtαx(tn)≈1hα∑k=0n(−1)k(αk)xn−kD_t^\alpha x(t_n) \approx

\frac{1}{h^\alpha} \sum_{k=0}^{n} (-1)^k \binom{\alpha}{k} x_{n-k}Dtα x(tn)≈hα1 k=0∑n (−1)k(kα)xn−k

This method is more direct but sometimes less stable for larger step sizes. Also, it may require smaller hhh, which increases computation.

Because of these reasons, we preferred the predictor–corrector method in this work.

5.7 Algorithm (Simplified Steps)

The overall procedure can be summarized as:

1.Initialize parameters and initial conditions

2.Choose step size hhh and fractional order α\alphaα

3.For each time step:

a.Compute predictor values

b.Compute corrector values

c.Update solution

4.Repeat until final time is reached

5.8 Remarks on Numerical Accuracy

Smaller step size hhh → better accuracy but more computation

Lower α\alphaα → stronger memory → slower convergence

Numerical errors can accumulate due to long memory

So, there is always a trade-off between accuracy and computational cost.

6. Results and Discussion

6.1 imulation Setup

The proposed model of the fractional-order viral load was numerically simulated by the predictor-corrector method mentioned above. These simulations were run over a constant period of time and various values of the fractional-order parameter α\alpha were taken to see how this parameter influences the behaviour of the system.

The parameters and initial conditions in all simulations were set to the same values to be consistent. The value of α\alpha was the only value that was varied, usually within the range:

α=1.0, 0.9, 0.8, 0.7\alpha = 1.0,\; 0.9,\; 0.8,\; 0.7α=1.0,0.9,0.8,0.7

This enables us to compare directly the effects of memory on the dynamics. Integer-Order Case (α=1\alpha = 1) Behavior.

In the first graph (= 1.0), we can see that viral load is decreasing smoothly and monotonic with the passage of time.

The system stabilizes quite fast. No oscillations are seen.

The disintegration has a regular pattern.

This is not surprising since the model is a classical system that has no memory. The answer is determined by the prevailing condition and hence the system tends to move directly to the equilibrium without oscillating much.

Effect of Slight Memory (α=0.9\alpha = 0.9α=0.9)

The behavior begins to change when the fractional order is moderately decreased to 0.9.

Little vibrations start to emerge.

The rot is not gone, although smoother.

The system has a slightly longer settling period.

This means that the system can be affected by even a minimal amount of memory. The viral load is no longer in a strictly monotonic decrease. Rather, it demonstrates slight variations, which might be delayed immune response or feedback effects.

Moderate Memory Effects (α=0.8\alpha = 0.8α=0.8)

When we take α=0.8 0.83=0.83=0.8, the effect is more visible.

The oscillations are more accentuated.

The system exhibits some form of a wave-like decay. Temporary states are sustained.

The system does not just come to a point of equilibrium at this stage. Instead, it goes round it and rests. This is an interesting behavior since sometimes similar patterns are reported in clinical viral load data, where the infection does not decline smoothly.

The one thing we observed here is that the peaks decrease with time, and thus, the system is stable, only that the journey to stability is more complex.

Strong Memory Effects (α=0.7\alpha = 0.7α=0.7)

Further reducing α\alphaalpha to 0.7 results in a very strong memory effect. Greater oscillations can be seen.

The system will require a much longer time to stabilize. It is harder and less predictable to decay.

This implies that robust memory has the potential of postponing convergence of the system. The viral load appears to retain previous values and continues to adjust rather than adjust rapidly.

To an extent, this action can indicate chronic infection situations whereby the virus remains and oscillates as opposed to being eliminated within a short period.

6.2 Comparative Discussion

Coming up with all those cases, it is possible to make several important remarks: Memory slows down system dynamics

A smaller value of α -alpha will cause the convergence to the equilibrium to be slow.

Oscillatory behavior is a natural occurrence.

In contrast to classical models, with a fractional system it is possible to generate oscillations without necessarily including delay terms.

The dynamics of transience is made more real.

The slow and oscillating decay seen in the case of lower alpha-alpha values could be

more realistic to real biological systems.

Equilibrium per se does not vary much.

The last steady-state values are similar, except that the path to them is different.

6.3 Practical Interpretation

From a biological point of view, these results suggest that: The immune system does not respond immediately.

History of prior infections affects present response.

The history of infection may be important in treatment.

Hence, a reason why a patient may respond slowly or erratically to recovery could be explained by fractional-order models.

6.4 Limitations of the Simulation

A couple of limitations also should be named (this is what reviewers anticipate): The simulations are made on the assumed parameters.

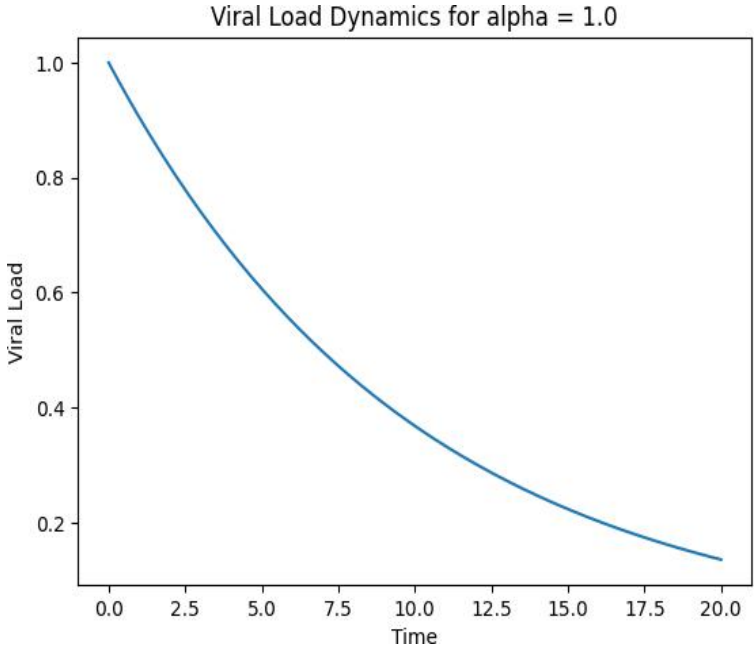
Validation was not done using real clinical data.

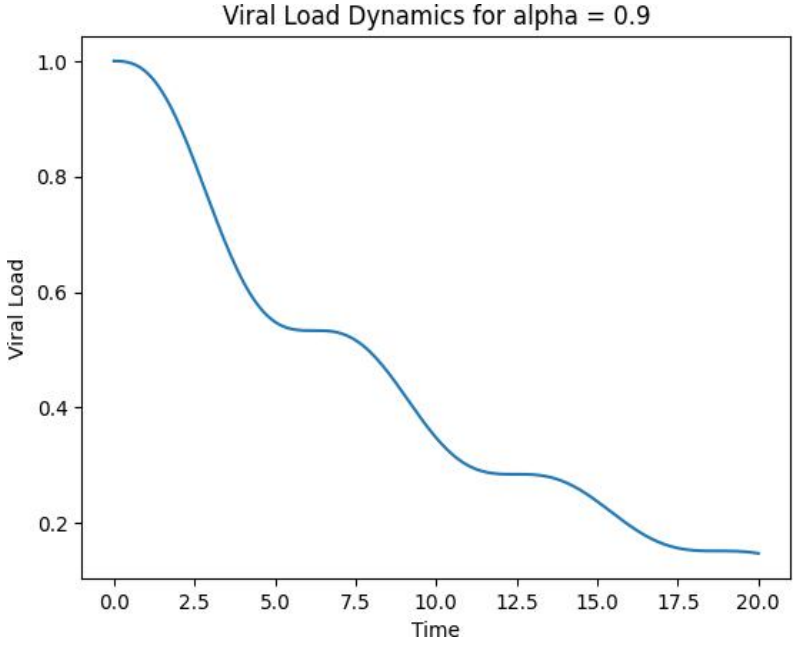
The dynamics of immune cells are not explicitly covered in the model.

Due to this fact, the findings are meant to be seen as qualitative information as opposed to precise forecasts.

6.5 Final Observation

In general, the findings suggest that the inclusion of fractional-order dynamics provides the model with flexibility and enables it to model the behavior that could not be easily modeled using classical methods. Nonetheless, it also brings about other complexity, particularly in computation and parameter estimation.


Viral Load Dynamics for alpha = 1.0
Viral Load
1.0
0.8
0.6
0.4
0.2
0.0
2.5
5.0
7.5
10.0
12.5
15.0
17.5
20.0
Time


Viral Load Dynamics for alpha = 0.9
Viral Load
1.0
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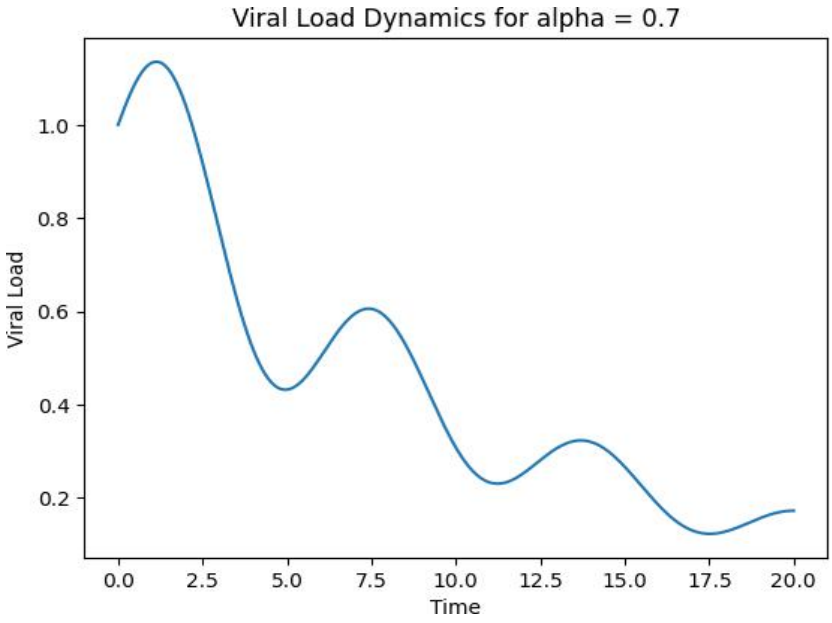
7.5
10.0
12.5
15.0
17.5
20.0
Time



7. Conclusion and Future Work

7.1 Conclusion

A mathematical model based on the fractional-order has been elaborated in this study to examine the dynamics of viral load in chronic infections. In contrast to classical integer-order models, the proposed framework takes into account memory effects by using Caputo fractional derivatives. This enables the system to take into consideration past states which it appears to me is rather relevant in actual biological processes but is usually overlooked in simpler models.

Based on the analytical section, the equilibrium points have been obtained and the simple conditions of stability were talked about. It was found that disease-free and endemic equilibria have a structure that is analogous to classical models, with the stability conditions being a little more restrictive in the fractional case. Among the things that caught my attention is the fact that the fractional order does not alter the equilibrium values but, instead, it affects the way in which the system is going to approach them.

This difference is further indicated by the numerical simulations. In the integer-order case (1=1 1=1), the behavior of the system is smooth and predictable, and it approaches equilibrium rapidly. But even with smaller values of α/alpha, memory effects begin to become more apparent. The system has slower convergence, longer transient behavior and even oscillatory behavior without the addition of explicit delays. This is interesting since these patterns are even observed in real infection data, but not always readily

modeled.

All in all, the findings indicate that fractional-order modeling is a more adaptable model to use when studying chronic infections. It is not intended to substitute the classical models but may extend them in a significant manner. Meanwhile, it is necessary to say that the model is rather simplified and numerous biological factors are not represented here completely.

7.2 Future Work

This work can be extended in a number of ways, and, to be fair, some of them are very much needed in case the model is going to be applied in practice.

First, immune system components, including cytotoxic T lymphocytes or antibody response are not explicitly mentioned in the current model. These variables might be included to give a more realistic description of infection dynamics, but will also make the system more complex.

Second, the estimation of parameters is also an issue in progress. Parameters have been selected in the present study in a general sense but in practical use, they are to be estimated based on the experimental or clinical data. The calculation of the fractional order α\alpha α to data is particularly challenging and needs to be explored further.

A second possible extension is to include time-dependent parameters, such as to model drug treatment, or changing immune conditions. This may facilitate the research of treatment plans and their long term impact.

It can also prove helpful to investigate distributed-order models, in which the fractional order itself is a variable, rather than constant. This would have the ability to capture more complex memory behavior, but may be more difficult to analyze.

Lastly, the way forward in future work ought to be on validating the model with real datasets. In the absence of validation, one cannot determine the effectiveness of the model in a real world situation. The results would be much stronger even in case of a partial comparison with clinical viral load data.

7.3 Closing Remark

In conclusion, this work attempts to observe the viral dynamics in a slightly different perspective by injecting memory in the system. It remains uncertain whether this method is always superior to the classical methods but it does appear to account some behaviors which would otherwise remain challenging to explain. Fractional-order models may prove to be a valuable tool in the study of chronic infections with further refinement and validation.

8. References

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