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NON-INTEGER MATHEMATICAL MODEL OF RESPIRATORY SYNCYTIAL VIRUS

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ABSTRACT
Respiratory syncytial virus (RSV), also known as a human respiratory syncytial virus (hRSV) and human orthopneumovirus, is a common, transmittable virus that roots respiratory tract diseases. It is a negative-sense, single-stranded Ribonucleic acid (RNA) virus. It gets its name from syncytia, which are huge cells that form when infected cells merge. In this paper fractional order model of the respiratory syncytial virus (RSV) virus will be developed. The Caputo fractional derivative operator of the order $\alpha \in (0,1]$will be used to generate the model scheme of non-integer differential equations. To calculate an estimated solution of the system of nonlinear fractional differential equations, the Laplace-Adomian Decomposition Method was used. Infinite series was produced as solutions to fractional differential equations. The model's proposed series solution converges quickly to its precise value. The obtained results are compared to the standard case.

Keywords: Non-Integer, Respiratory Syncytial Virus (RSV) Mathematical model, Ribonucleic acid (RNA)

INTRODUCTION

Respiratory syncytial virus (RSV), commonly known as respiratory syncytial virus, is a prevalent respiratory virus that causes mild, cold-like symptoms. RSV can be dangerous, especially in small children and the elderly, although most people recover within a week or two. RSV is the leading cause of bronchiolitis (inflammation of the tiny airways in the lung) and pneumonia (lung infection) in children under the age of one in the United States (CDC, 2022). RSV was first identified in 1956 when scientists isolated a virus from a group of sick chimps. The CCA (Chimpanzee Coryza Agent) virus was named (Morris, Blount Jr, & Savage, 1956). In 1957, Robert M. Chanock found this virus in newborns with respiratory problems (Chanock & Roizman, 1957). According to investigations of human antibodies in neonates and children (Morris, Blount Jr, & Savage 1956), the infection was common during infancy. The virus is also known as the human orthopneumovirus or the human respiratory syncytial virus (hRSV) (Walsh, & Hall 2015).

According to estimates, there were 1.4 million RSV-associated acute lower respiratory infection hospitalizations among infants under 6 months of age and 6.6 million RSV LRTI episodes worldwide in 2019 (Li et al 2022). RSV hospitalizations made up 9.3% of all infant hospitalizations in the US between October 2015 and December 2019 and were the most frequent cause of infant hospitalizations outside of birth hospitalizations, according to a retrospective analysis of the National Inpatient Sample (NIS) (Suh et al 2022). Due to the low rate of RSV testing, the diagnosis of bronchiolitis may lead to an additional 3.7% of hospitalizations (Suh et al 2022). To better understand the dynamics of RSV, researchers have put forth several mathematical models. Others have evaluated the efficiency of control systems in thwarting RSV using mathematical models. Integer-order differential equations are used in these models. To the best of our knowledge, this is the first non-integer model for RSV.

Using seasonal driving of incidence and transient intra- and inter-group partial immunity, (White et al 2005) developed a unique integer mathematical model for hRSV transmission. They concluded that the variations in these populations' dynamics could be explained solely by the differences in the two populations' seasonal and magnitude-based contact rates. Furthermore, (White et al 2007) established an integer model. They determined that it reduced infectiousness and transient immunity (which could be partial) and proposed a single model structure that captures four different host responses to infection and subsequent reinfection: partial susceptibility and changed infection duration. (Arenas, Moraño & Cortés, 2008), on the other hand, developed a non-standard numerical framework for a SIRS seasonal integer epidemiological model for RSV transmission. This novel numerical method is used to approximate the answer with varying step sizes while maintaining the continuous model's positivity. This unconventional numerical method is used to approximate the solution using various step sizes while maintaining the positivity of the continuous model. They conducted simulations using data from Finland and the Gambia and compared their approach to a few well-known explicit methods.

To induce the observed seasonality of RSV in the Philippines, (Paynter, et al 2014) estimated when ecological determinants of respiratory syncytial virus (RSV) transmissibility would need to act using an integer mathematical transmission model. According to their calculations, a seasonal high in transmissibility would need to occur between 49 and 67 days (or about 51 days) before the actual peak in RSV infections. They concluded that the timing of the seasonal patterns of rainfall and nutritional status were both consistent with the projected seasonal pattern of transmissibility and that these were both likely causes of the seasonality of RSV in this environment. A model to replicate the biennial seasonal epidemic curves of RSV identifications in metropolitan Western Australia has been effectively created (Moore et al 2014). The model's quality of data acquired from linked individual-level total population-based data sources is one of its strongest points. They concluded that not all RSV-positive detections result in hospitalizations, so it's crucial to avoid limiting data sources to the clinically severe end of the spectrum.

To understand the mechanisms underlying RSV infection kinetics in the lung, (Wethington et al 2019) combined mathematical modeling using ordinary differential equations (ODEs) with measurements of RSV infection kinetics in

primary well-differentiated human bronchial epithelial cultures in vitro as well as in immunocompetent and immunosuppressed cotton rats. In cotton rats, their combined technique assessed the role of the adaptive immune response in preventing RSV infection, which may help assess potential RSV vaccine candidates. In 2022, (Sungchasit, Tang & Pongsumpun 2022), Built, a model for the spread of the respiratory syncytial virus (RSV) in a constant population of humans is taken into account. This model assumes that there are super-spreading infected individuals (who infect numerous people in a single encounter). The epidemiological data for the illnesses brought on by this virus have shown instances where some people are super-spreaders of the virus. The numerical simulations demonstrate how disease dynamics alter as values for the parameters in the SEI_rI_sRmodel is changed.

Adomian invented the Laplace Adomain decomposition Method (LADM) in 1980, which combines the Adomain decomposition method and the Laplace transform. The approach works effectively for solving several types of differential equations. To investigate the numerical solution of the corruption model, we employ fractional calculus and LADM. In the model, the Caputo derivative is regarded as a differential operator. This effort will make use of various well-known concepts and conclusions from the literature (Farman et al 2018 &Yakubu, Abdullah & Abdullahi 2021).

Preliminaries

This unit emphases on about fundamental non-integer calculus definitions and results. For more details see (Farman et al 2018, Yakubu, Abdullah & Abdullahi 2021, Hassan & El-Tawil (2011), Haq et al 2018, Gökdoğan, Yildirim & Merdan (2011) & Biazar, 2006)

$I_{0+}^{\alpha}f(t) = \frac{1}{\Gamma(\alpha)}\int_0^1 (t-s)^{\alpha-1} f(s)ds,$

Definition 1. The Riemann-Liouville fractional integral of order $\alpha \in (0,1)$of a function $f \in L^1([0,T],\Re)$ is defined as

Definition 2. The Caputo fractional order derivative of an interval function is defined as

$$ {}^{c}D_{0+}^{\alpha}f(t) = \frac{1}{\Gamma(\alpha)}\int_0^1 (t-s)^{n-\alpha-1} f^{(n)}(s)ds, \quad (1)$$

When $n = |x| + 1 and |x|$ signifies the integer part of x. More specifically,$0 < x < 1$, the Caputo derivative changes to

$$ {}^{c}D_{0+}^{\alpha}f(t) = \frac{1}{\Gamma(\alpha)}\int_0^1 \frac{f(s)}{(1-s)} ds. \quad (2)$$

Lemma 1. The following is accurate for fractional differential equations. $I^{\alpha}(\ {}^{c}D^{\alpha}h)(t) = h(t) + \sum_{i=0}^{n-1}\frac{h^i(0)}{i!}t^i.$
$for\ arbitrary\ x > 0, i = 0,1,2,\dots,n-1, when\ n = |x| + 1 and |x| represents$the integer part of x

Definition 2.3. We note that the Laplace transform of Caputo derivative formulation as:

$$\ell\{\ {}^{c}D^{\alpha}y(t)\} = s^{\alpha}h(s) - \sum_{k=0}^{n-1} s^{\alpha-i-1}y^{(k)}(0), n-1 < \alpha < n, n \in N.$$

$for\ arbitrary\ x > 0, i = 0,1,2,\dots,n-1, when\ n = |x| + 1 and |x| represents$the integer part of x

The model formulation

Based on the integer mathematical model developed by (Gökdoğan, Yildirim & Merdan 2011), the (susceptible-exposed–infected–recovered) model where the infected humans is split in two classes the normal infection and the super spreader infected class. However, we adapt the model of (Gökdoğan, Yildirim & Merdan 2011) convert to non-integer mathematical model and also make some adjustments which include the recruitment (birth and immigration) rate of the susceptible humans instead of the birth rate of the human population since the disease-affected both children and older adults and both migrate. We also included the infectivity death rate of the infected class since there are human deaths resulting from the infectivity (Savic et al 2023). Thus yield

$$\begin{aligned}
D^{\alpha_1}S(t) &= \Lambda - \beta S(t)\big(I_r(t) + I_s(t)\big) - \mu S(t)\\
D^{\alpha_2}E(t) &= \beta S(t)\big(I_r(t) + I_s(t)\big) - \left(\frac{1}{\eta}\right)\rho E(t) - \left(\frac{1}{\eta}\right)(1-\rho)E(t) - \mu E(t)\\
D^{\alpha_3}I_r(t) &= \left(\frac{1}{\eta}\right)\rho E(t) - r_1 I_r(t) - (\mu + \mu_0)I_r(t)\\
D^{\alpha_4}I_s(t) &= \left(\frac{1}{\eta}\right)(1-\rho)E(t) - r_2 I_s(t) - (\mu + \mu_0)I_s(t)\\
D^{\alpha_5}R(t) &= r_1 I_r(t) + r_2 I_s - \mu R(t)
\end{aligned} \quad (3)$$

Tables 1 and 2 summarize the corresponding model variables and parameters.

Wherever all other parameters are positive and the stated initial conditions are given below

$$\begin{cases} S(0) = N_1\\ E(0) = N_2\\ I_r(0) = N_3\\ I_s(0) = N_4\\ R(0) = N_5, \end{cases} \quad (4)$$

Stability Investigation and Equilibria

Disease-free equilibrium (DFE)

The model (3) has a DFE, which may be determined by setting the right-hand sides of the equations in (3) to zero, as shown by

$$\begin{cases} D^{\alpha_1}S(t) = 0\\ D^{\alpha_2}E(t) = 0\\ D^{\alpha_3}I_r(t) = 0\\ D^{\alpha_4}I_s(t) = 0\\ D^{\alpha_5}R(t) = 0 \end{cases} \quad (5)$$

$$E_0 = (S^*, E^*, I_r^* I_s^*, R^*) = \left(\frac{\Lambda}{\mu}, 0,0,0,0\right) \quad (6)$$

The Laplace–Adomian Decomposition Method

This section describes the numerical method for our model (3) using the initial circumstances provided. Using the Caputo fractional derivative system, which entails applying the Laplace transform to both sides of the equation (3), we obtain:

$$L\{D^{\alpha_1}S(t)\} = L\{\Lambda - \beta S(t)(I_r(t) + I_s(t)) - \mu S(t)\}$$
$$L\{D^{\alpha_2}E(t)\} = L\left\{\beta S(t)(I_r(t) + I_s(t)) - \left(\frac{1}{\eta}\right)\rho E(t) - \left(\frac{1}{\eta}\right)(1-\rho)E(t) - \mu E(t)\right\}$$
$$L\{D^{\alpha_3}I_r(t)\} = L\left\{\left(\frac{1}{\eta}\right)\rho E(t) - r_1 I_r(t) - (\mu + \mu_0)I_r(t)\right\}$$
$$L\{D^{\alpha_3}I_s(t)\} = L\left\{\left(\frac{1}{\eta}\right)(1-\rho)E(t) - r_2 I_s(t) - (\mu + \mu_0)I_s(t)\right\} \quad (7)$$
$$L\{D^{\alpha_4}R(t)\} = L\{r_1 I_r(t) + r_2 I_s - \mu R(t)\}$$

This implies that

$$S^{\alpha_1}L\{D^{\alpha_1}S(t)\} = L\{\Lambda - \beta S(t)(I_r(t) + I_s(t)) - \mu S(t)\}$$
$$S^{\alpha_2}L\{D^{\alpha_2}E(t)\} = L\left\{\beta S(t)(I_r(t) + I_s(t)) - \left(\frac{1}{\eta}\right)\rho E(t) - \left(\frac{1}{\eta}\right)(1-\rho)E(t) - \mu E(t)\right\}$$
$$S^{\alpha_3}L\{D^{\alpha_3}I_r(t)\} = L\left\{\left(\frac{1}{\eta}\right)\rho E(t) - r_1 I_r(t) - (\mu + \mu_0)I_r(t)\right\} \quad (8)$$
$$S^{\alpha_4}L\{D^{\alpha_3}I_s(t)\} = L\left\{\left(\frac{1}{\eta}\right)(1-\rho)E(t) - r_2 I_s(t) - (\mu + \mu_0)I_s(t)\right\}$$
$$S^{\alpha_5}L\{D^{\alpha_4}R(t)\} = L\{r_1 I_r(t) + r_2 I_s - \mu R(t)\}$$

We have the system (8) applying the initial conditions and the inverse Laplace transform. $S(t) = S(0) = L^{-1}\{\Lambda - \beta S(tt)(I_r(t) + I_s(t)) - \mu S(t)\}$

$$E(t) = E(0) = L^{-1}\left\{\beta S(t)(I_r(t) + I_s(t)) - \left(\frac{1}{\eta}\right)\rho E(t) - \left(\frac{1}{\eta}\right)(1-\rho)E(t) - \mu E(t)\right\}$$
$$I_r(t) = I_r(0) = L^{-1}\left\{\left(\frac{1}{\eta}\right)\rho E(t) - r_1 I_r(t) - (\mu + \mu_0)I_r(t)\right\} \quad (9)$$
$$I_s(t) = I_s(0) = L^{-1}\left\{\left(\frac{1}{\eta}\right)(1-\rho)E(t) - r_2 I_s(t) - (\mu + \mu_0)I_s(t)\right\}$$
$$R(t) = R(t) = L^{-1}\{r_1 I_r(t) + r_2 I_s - \mu R(t)\}$$

Using the values of the initial condition in (9), we get

$$S(t) = N_1 = L^{-1}\{\Lambda - \beta S(tt)(I_r(t) + I_s(t)) - \mu S(t)\}$$
$$E(t) = N_2 = L^{-1}\left\{\beta S(t)(I_r(t) + I_s(t)) - \left(\frac{1}{\eta}\right)\rho E(t) - \left(\frac{1}{\eta}\right)(1-\rho)E(t) - \mu E(t)\right\}$$
$$I_r(t) = N_3 = L^{-1}\left\{\left(\frac{1}{\eta}\right)\rho E(t) - r_1 I_r(t) - (\mu + \mu_0)I_r(t)\right\} \quad (10)$$
$$I_s(t) = N_4 = L^{-1}\left\{\left(\frac{1}{\eta}\right)(1-\rho)E(t) - r_2 I_s(t) - (\mu + \mu_0)I_s(t)\right\}$$
$$R(t) = N_5 = L^{-1}\{r_1 I_r(t) + r_2 I_s - \mu R(t)\}$$

Adopt that the results, $S(t), E(t), I_r(t), I_s(t), R(t)$ in the form of an infinite series, are given by

$$S(t) = \sum_{n=0}^{\infty} S_n(t)$$
$$E(t) = \sum_{n=0}^{\infty} E_n(t) \quad (11)$$
$$I_r(t) = \sum_{n=0}^{\infty} I_{r\,n}(t)$$
$$I_s(t) = \sum_{n=0}^{\infty} I_{s\,n}(t)$$
$$R(t) = \sum_{n=0}^{\infty} R_n(t)$$

While the nonlinear term involved in the model are $S(t)E(t), S(t)I_r(t), S(t)I_s(t)$and are decomposed as follows

$$S(t)I_r(t) = \sum_{n=0}^{\infty} B_n$$
$$S(t)I_s(t) = \sum_{n=0}^{\infty} C_n \quad (12)$$

where $B_n and C_n$are the Adomian polynomials defined as

$$B_n = \frac{1}{\Gamma(n+1)}\frac{d^n}{dt^n}\left[\sum_{k=0}^{\infty}\lambda^k S_k \sum_{k=0}^{\infty}\lambda^k I_{r_k}\right]|\lambda = 0$$
$$C_n = \frac{1}{\Gamma(n+1)}\frac{d^n}{dt^n}\left[\sum_{k=0}^{\infty}\lambda^k S_k \sum_{k=0}^{\infty}\lambda^k I_{s_k}\right]|\lambda = 0 \quad (13)$$

The first three polynomials are given by

$B_0 = S_0(t)I_{r_0}(t)$,
$B_1 = S_0(t)I_{r1}(t) + S_1(t)I_r(t)$
$B_2 = 2S_0(t)I_{r2}(t) + 2S_1(t)I_{r1}(t) + 2S_2(t)I_{s0}(t)$

$C_0 = S_0(t)I_{s_0}(t),$
$C_1 = S_0(t)I_{s1}(t) + S_1(t)I_s(t)$
$C_2 = 2S_0(t)I_{s2}(t) + 2S_1(t)I_{s1}(t) + 2S_2(t)I_{s0}(t)$ (14)

Using (11), (13) in model (9), yields

$$L\left\{\sum_{n=0}^{\infty} S(t)\right\} = \frac{S_0}{s} + \left[\frac{1}{s^{\alpha}} L\{\Lambda - \beta S(t)\left(I_r(t) + I_s(t)\right) - \mu S(t)\}\right]$$
$$L\left\{\sum_{n=0}^{\infty} E(t)\right\} = \frac{E_0}{s} + \left[\frac{1}{s^{\alpha}} L\left\{\beta S(t)\left(I_r(t) + I_s(t)\right) - \left(\frac{1}{\eta}\right)\rho E(t) - \left(\frac{1}{\eta}\right)(1-\rho)E(t) - \mu E(t)\right\}\right]$$
$$L\{\textstyle\sum_{n=0}^{\infty} I_r(t)\} = \frac{I_{r0}}{s} + \left[\frac{1}{s^{\alpha}} L\left\{\left(\frac{1}{\eta}\right)\rho E(t) - r_1 I_r(t) - (\mu + \mu_0) I_r(t)\right\}\right] \quad (15)$$
$$L\left\{\sum_{n=0}^{\infty} I_s(t)\right\} = \frac{I_{s0}}{s} + \left[\frac{1}{s^{\alpha}} L\left\{\left(\frac{1}{\eta}\right)(1-\rho)E(t) - r_2 I_s(t) - (\mu + \mu_0) I_s(t)\right\}\right]$$
$$L\{\textstyle\sum_{n=0}^{\infty} R(t)\} = \frac{R_0}{s} + \left[\frac{1}{s^{\alpha}} L\{r_1 I_r(t) + r_2 I_s - \mu R(t)\}\right]$$

Iterative algorithms are produced by synchronizing both sides of (15).

Differential Transform Method

The system (3) has a subsequent recurrence relation with respect to time t.

$$S(k+1) = \frac{1}{k+1}\left[\Lambda\partial(k) - \beta\sum_{i=0}^{k} S(l)I_r(k-l) - \beta\sum_{i=0}^{k} S(t)I_s(k-l) - \mu S(k)\right]$$
$$E(k+1) = \frac{1}{k+1}\left[\beta\sum_{i=0}^{k} S(l)I_r(k-l) + \beta\sum_{i=0}^{k} S(l)I_s(k-l) - \left(\frac{1}{\eta}\right)\rho E(k) - \left(\frac{1}{\eta}\right)(1-\rho)E(k) - \mu E(k)\right]$$
$$I_r(k+1) = \frac{1}{k+1}\left[\left(\frac{1}{\eta}\right)\rho E(k) - r_1 I_r(k) - (\mu + \mu_0) I_r(k)\right] \quad (16)$$
$$I_s(k+1) = \frac{1}{k+1}\left[\left(\frac{1}{\eta}\right)(1-\rho)E(k) - r_2 I_s(k) - (\mu + \mu_0) I_s(k)\right]$$
$$R_c(k+1) = \frac{1}{k+1}\left[r_1 I_r(k) + r_2 I_s - \mu R(k)\right]$$

The inverse differential transform $S(k)$is defined as When t_0 is set to zero, the given function $y(x)$ is denoted by a finite series, and the above equation can be written as$S(t) = \sum_{k=0}^{2} S(k)t^i$

The function is derived by solving the above equation (16) for$S(k+1), E(k+1), I_r(k+1), I_s(k+1), and R(k+1)$ up order 2 and $S(k), E(k), I_r(k), I_s(k) and R(k)$respectively.

$$\begin{cases} S(t) = \sum_{k=0}^{2} S(k)t^k \\ E(t) = \sum_{k=0}^{2} E(k)t^k \\ I_r(t) = \sum_{k=0}^{2} I_r(k)t^k \\ I_s(t) = \sum_{k=0}^{2} I_s(k)t^k \\ R(t) = \sum_{k=0}^{2} R(k)t^k \end{cases} \quad (17)$$

Numerical Results

The plots below depict the population of each compartment at various levels of $\alpha_i (i = 1,2,3,4)$

Table 1: State variables in the RSV model described

Parameter	Description	Values
S	Susceptible humans	600
E	Exposed humans to RSV	250
I_r	Infected humans to RSV	100
I_r	Infected humans to RSV	100
R	Recovered humans to RSV	50

Table 2: Parameters in the RSV model is described.

Par.	Description	Est. Value	References
Λ	The recruitment rate of humans	0.21	Gökdoğan, Yildirim & Merdan (2011)
β	Force of infection	0.1-0.19	Gökdoğan, Yildirim & Merdan (2011)
μ	Natural death of humans	0.21	Gökdoğan, Yildirim & Merdan (2011)
μ_0	Death due to infectivity	0.05	Assumed
η	Incubation rate of virus in human	0.1-0.19	Gökdoğan, Yildirim & Merdan (2011)
ρ	Probability of regulated infected human	0.01-0.0009	Gökdoğan, Yildirim & Merdan (2011)
$(1-\rho)$	Probability of super spreading infected human	0.01-0.9	Gökdoğan, Yildirim & Merdan (2011)
r_1	Recovery of regular infected humans	0.01-0.9	Gökdoğan, Yildirim & Merdan (2011)
r_2	Recovery of super spreading infected humans		Gökdoğan, Yildirim & Merdan (2011)

The Plots of the LADM of the RSV Model

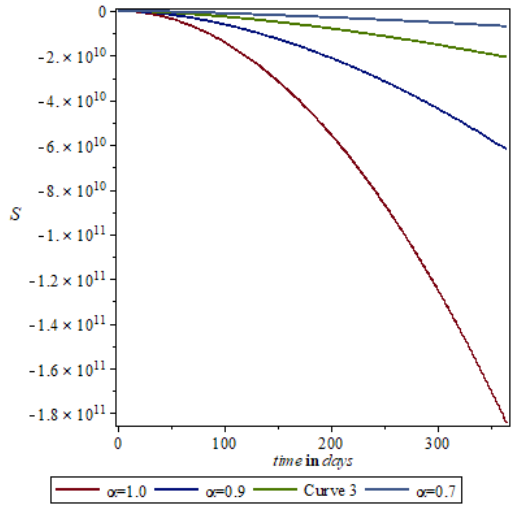

Figure 1: Demonstrates the behavior of the susceptible humans

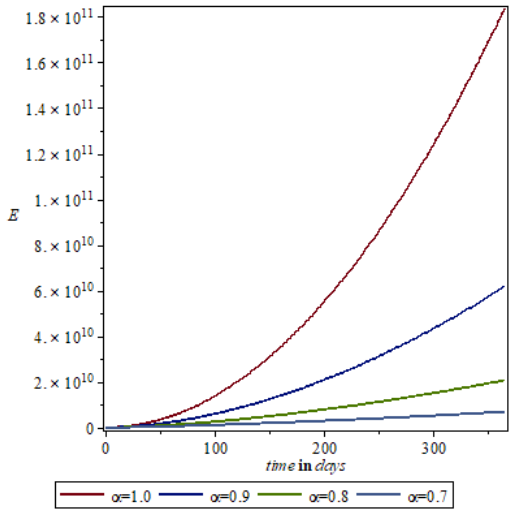

Figure 2: Demonstrates the behavior of the exposed humans

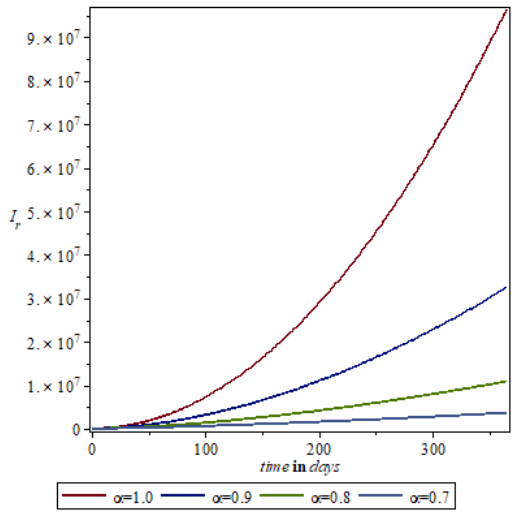

Figure 3: Demonstrates the behavior of the infected humans with regular spread

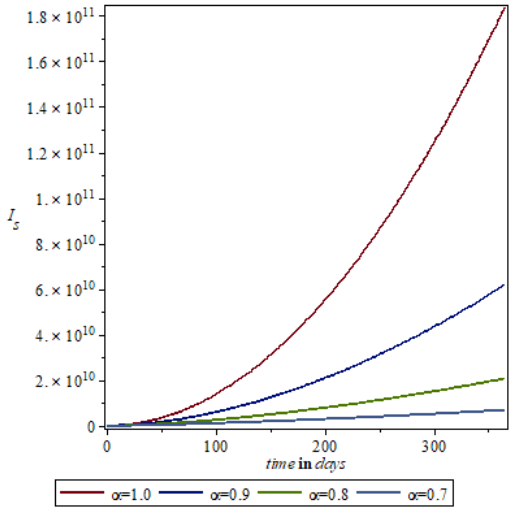

Figure 4: Demonstrates the behavior of the infected humans with regular spread

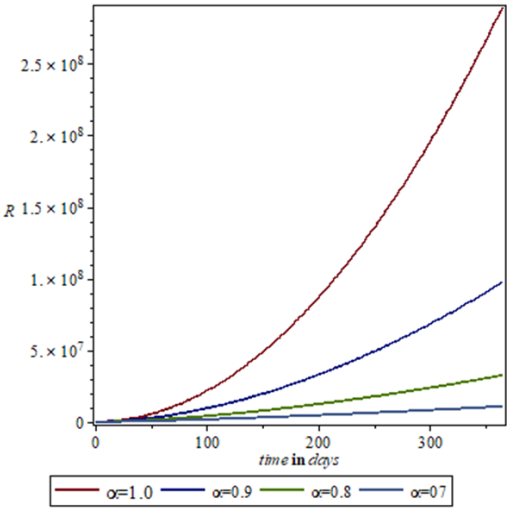

Figure 5 Demonstrates the behavior of the recoverd humans.

The Plots of the LADM VS DTM of the RSV Model

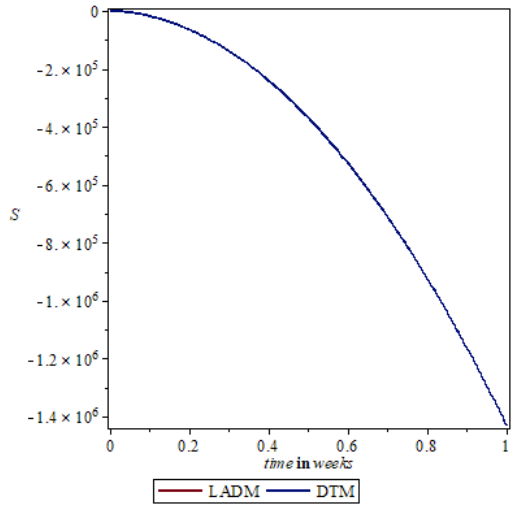

Figure 6: Plots exhibiting LADM and DTM dynamics of the susceptible humans

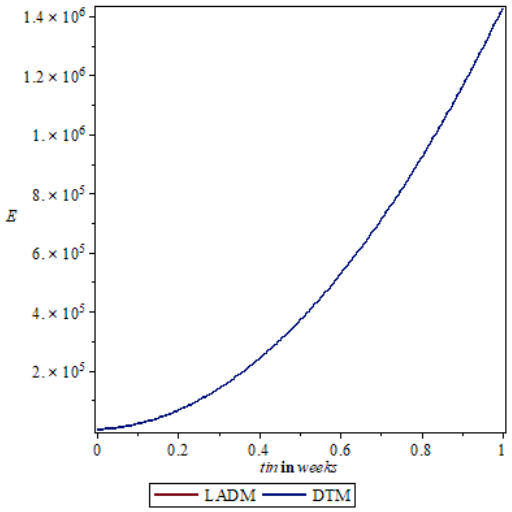

Figure 7: Plots exhibiting LADM and DTM dynamics of the exposed humans

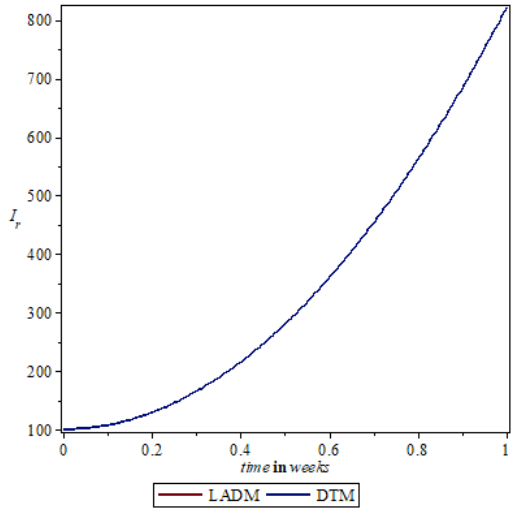

Figure 8 Plots exhibiting LADM and DTM dynamics of the regular infected humans

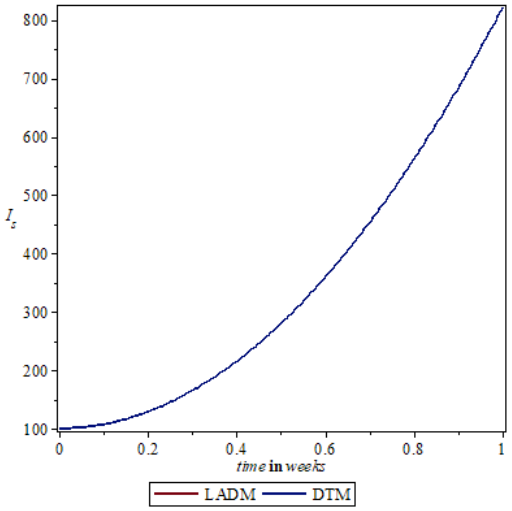

Figure 9 Plots exhibiting LADM and DTM dynamics of the super spread infected humans

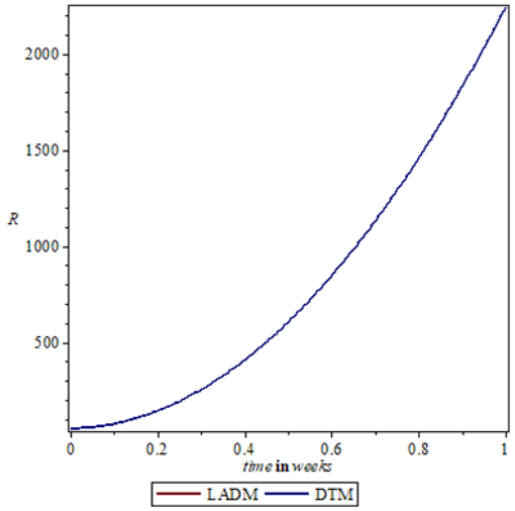

Figure 10: Plots exhibiting LADM and DTM dynamics of the recovered humans

CONCLUSION

This work developed a fractional order model of the respiratory syncytial virus, or RSV. The Caputo fractional derivative operator of the order was used to build the model scheme of non-integer differential equations. The Laplace-Adomian Decomposition Method was used to discover an estimated solution to the system of nonlinear fractional differential equations. It was discovered that the solutions to fractional differential equations are infinite series. Figures 1–10 demonstrate how the model's proposed series solution soon converges to its precise value. The acquired results are applicable to the typical instance.

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