Mathematical Analysis of Varicella Zoster Virus Model

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Abstract: Chicken Pox (also called Varicella) is a disease caused by a virus known as Varicella Zoster Virus (VZV) also known as human herpes virus 3 (HHV -3). Varicella Zoster Virus (VZV) is a DNA virus of the Herpes group, transmitted by direct contact with infective individuals. In this work, a deterministic mathematical model for transmission dynamics of Varicella Zoster Virus (VZV) with vaccination strategy was solved, using Adomian Decomposition Method (ADM) and Fourth-Fifth Rungekutta Felhberg Method and Approximate solutions were realized. ADM, yields analytical solution in terms of rapidly convergent infinite power series with easily computed terms. This solution was realized by applying Adomian polynomials to the nonlinear terms in the system. Similarly, fourth-fifth-order Runge-Kutta Felberg method with degree four interpolant (RK45F) was used to compute a numerical solution that was used as a reference solution to compare with the semi-analytical approximations. The main advantage of the ADM is that it yields an approximate series solution in close form with accelerated convergence. The effect of Varicella was considered in five compartments: The Susceptible, the Vaccinated, the Exposed, the Infective and the Recovered class. The Varicella Zoster virus model which is a nonlinear system can only be solved conveniently using powerful semi-analytic tool such as the ADM. Numerical simulations of the model show that, the combination of vaccination and treatment is the most effective way to combat the epidemiology of VZV in the community.

Keywords: Varicella, Zoster, Adomian Decomposition, Modeling, Sensitivity, Vaccination, Epidemiology

1. Introduction

In recent years, a lot of attention has been on the study of the Adomian decomposition method to investigate various scientific models. The ADM method is used for finding the numerical solution of higher-order deferential equations. This method which accurately computes the series solution is of great interest to applied science, engineering, physics, biology, and so forth. The method provides the solution in a rapidly convergent series with components that can be elegantly computed [1]. The work by [2], was aimed at producing approximate solutions which are obtained in rapidly convergent series with elegantly computable components by the Adomian decomposition technique. They also revealed that the Adomian decomposition method is useful for obtaining both a closed form and the explicit solution and numerical approximations of linear or nonlinear differential equations, and it is also quite straight forward to write computer codes. This method has been applied to obtain formal solution to a wide class of stochastic and deterministic problems in science and engineering involving algebraic, diffferential, integrodifferential, differential delay, integral and partial differential equations.

It is well known in the literature that the decomposition method provides the solution in a rapidly convergent series where the series may lead to the solution in a closed form if it exists.

Adomian Decomposition Method (ADM) is a technique for solving functional equations. This method was first introduced by Adomian to be used in solving stochastic and deterministic problems in basic and applied sciences. The method was observed to give analytical solution in terms of an infinite series which can be obtained without linearization, perturbation, transformation or discretization [3]. It can also be applied to non-linear differential equations both ordinary and partial linear equation and other kind of problems in science. The advantage of this method is that it provides a direct scheme for solving the problem of nonlinear epidemiological model [4].

In [5] the Adomian's scheme was used for solving differential systems for modelling the HIV immune dynamics.

The weaknesses of the thin-sheet approximation was investigated and a higher-order development allowing increasing range of convergence and preserving the nonlinear dependence of the variables was proposed [6]. In general, the decomposition method yields rapidly convergent series solutions by using a few iterations for both linear and nonlinear deterministic and stochastic equations. The advantage of this method is that it provides a direct scheme for solving the problem, i.e., without the need for linearization, perturbation, massive computation and any transformation. The convergence of this method was investigated by Cherruault and workers. A new convergence proof of Adomian's method based on properties of convergent series was proposed [7]. The convergence of Adomian's method to periodic temperature fields in heat conductors was investigated in [8]. This method has been applied to nonlinear algebraic equations, ordinary differential equations, delay differential equations, system of ordinary differential equations, partial differential equations and certain problems of linear algebra [9].

The approximation to the solution of hyperbolic equations by Adomian Decomposition Method and comparison with the method of characteristics was carried out by [10]. Numerical methods which are commonly used, needed large size of computation work and usually, round off error causes the loss of accuracy. As such, Adomian Decomposition Method has been applied to solve many functional equations and system of functional equations. They stated that Adomian Decomposition Method has proved to be very effective and results in considerable saving of computation time.

The ADM technique is based on a decomposition of a solution of a nonlinear functional equation in a series of functions. Each term of the series is obtained from a polynomial generated by a power series expansion of an analytic function. The Adomian method is very simple in an abstract formulation but the difficulty arises in computing the polynomials and in proving the convergence of the series of functions [11]. In recent years, more and more researchers have applied this method to solving nonlinear systems [13–15]. We firstly study the algorithm and convergence analysis of ADM, and then apply ADM to constructing approximate solutions for nonlinear equations with initial data, including algebraic equations, fractional ordinary differential equations and fractional partial differential equations. It is very easy to apply and can solve wide classes of nonlinear systems including algebraic equation, ordinary differential equations, partial differential equations, integral equations, integro-differential equations, and so on and so forth [12].

The Adomian decomposition method (ADM) proved to be an effective technique in dealing with nonlinear problems with initial data. By applying the ADM, one can construct approximate solutions to algebraic equations, fractional ordinary differential equations (time-fractional Riccati equations etc.), fractional partial differential equations (timefractional Kawahara equations, modified time-fractional Kawahara equations etc.), and even integro-differential

equations, differential algebraic equations and so on. In practical applications, we can take a finite sum according to the accuracy we need [16].

We would be using the Adomian Decomposition Method, which has been proven reliable to solving complex nonlinear equation to analyze the Varicella Zoster Virus model in this work.

Several studies have been carried out on Varicella Zoster Virus, which causes what we commonly know as Chicken pox. Varicella zoster virus (VZV) is a common and ubiquitous human-restricted neurotropic alphaherpesvirus of the Herpes viridae family that persists for life in the host after a primary infection (varicella or chickenpox). The site of latency is within neurons in ganglia of the peripheral somatic, autonomic, and enteric nervous systems [17]. Up to one third of infected individuals will clinically reactivate VZV in their lifetimes, usually in their elderly years when immunity is naturally senescing, or when immunity is suppressed by disease or iatrogenic cause. The most common clinical sign of reactivation is herpes zoster (HZ), manifested as a dermatome-limited, painful vesicular skin rash that causes greater morbidity than varicella with frequent complications. VZV reactivation may also underlie a variety of neurological [19], vascular [20], and gastrointestinal diseases [17] that may occur with or without rash.

A greater understanding of the events occurring during VZV latency and early reactivation may reveal targets and strategies for novel therapeutics that treat or prevent HZ. However, VZV shows a high degree of host specificity, which has precluded most small animal modeling of VZV disease. There is arguably still no in vivo model of reactivated disease. A possible exception for the growth restriction of VZV in small animals is the guinea pig, discussed below. Many insights have come from study of the VZV-related Simian Varicella Virus (SVV), which replicates in several macaque species and African green monkeys, but SVV is a distinct virus that may not completely model all aspects of VZV. There are also significant financial and ethical issues associated with primate research. The relevance of the SVV model to VZV was recently reviewed [21].

A second issue that has made VZV persistence difficult to understand is the difficulty in interpreting studies of VZV-infected human cadaveric ganglia. We are now more cognizant that a partial viral gene expression program occurs throughout the post mortem interval (PMI) [22, 23]. Early reports suggesting that VZV proteins were made during latency were subsequently complicated by staining artifacts or antibody cross-reactivity to blood group A1 antigens [24]. Very recent work indicates VZV latent transcription is highly restricted in ganglia obtained with short PMI [25].

It was revealed in [18] that the virus is spread either by direct contact with a person with active chicken pox or shingles, or by direct contact with clothes or other articles infected with vesicle fluid, saliva, nasal discharge, or by air borne spread of small droplets of infected mucous of fluid. They identified that in 2006, the Food and Drug Administration approved the zoster vaccine, a higher

concentration of the same live attenuated virus used in the primary Varicella vaccine, for persons 60 years of age or older. In their work, they revealed that the treatment for a patient with chicken pox is: reducing itches and irritation by keeping the skin cool with light clothing and tepid baths or sponging.

2. The Model of Edward *et al.* (2014)

We would review a deterministic mathematical model for VZV formulated by Edward *et al.* (2014) which incorporates vaccination strategy. The total population is divided into the following epidemiological classes or subgroups: susceptible, vaccinated V, Exposed E, infectious I, recovered R. Basically, we modify the SEIR model by adding a vaccination compartment which caters for immunization.

Let us assume that the per capita birth rate π is constant, the natural fatality rate μ is time constant, there is no disease induced death, the members of the population mix homogenously (have the same interactions with one another to the same degree), and assume that on recovery, there is a permanent immunity of the rate η. Furthermore, assume that individuals can be infected through direct contact c, with an infectious individual. We let β to be the probability that a susceptible individual becomes infected by one infectious individual. We also let Λ be the constant recruitment rate. The susceptible and vaccinated individuals are recruited by both birth and immigration. A proportion ρ of the recruits are vaccinated, the remaining $1 - \rho$ are not vaccinated so they join the susceptible compartment. Proportions of newborns ϕ are vaccinated, and the remaining $1 - \phi$ newborns are not vaccinated and hence join the susceptible compartment. We consider that a proportion of the population of susceptible to receive a first dose vaccine at the rate θ_1, whereas the rest of it progress with the disease.

The primary vaccine wanes at the rate α after a fixed time t. After the first vaccine has expired, a proportion $1 - f$ of the vaccinated individuals at dose one, join the susceptible compartment at the rate α while the remaining proportion f receive a second dose at the rate θ_2, Our assumption is that the individuals who have attended the first and the second dose consecutively receive permanent immunity; otherwise they become susceptible to the disease again. The susceptible individuals enter the exposed compartment at the rate λ which is a force of infection. The exposed individuals are the ones who are infected but not infectious. After some time the exposed become infectious, they move from exposed state to infectious at the rate δ. An infected individual recover at rate η, and according to the nature of the disease; the recovered individuals are permanently immune.

The description of dynamics of VZV above can be summarized by compartmental diagram as seen in Figure 1.

3. Methods

In this chapter, we will apply the Adomian Decomposition Method (ADM) to nonlinear epidemiological model of chicken pox.

3.1. The Adomian Decomposition Method (Al-Hayani et al., 2013)

Let us consider a general functional equation

$$y - N(y) = f \qquad (1)$$

Where N is a non-linear operator, f is a known function in which the solution y satisfying equation (1) is to be found. We assume that for every f, the problem (1) has a unique solution.

The Adomian technique consists of approximating the solution of (1) as an infinite series.

$$y = \sum_{n=0}^{\infty} y_n$$

and decomposing the non-linear operator N as

$$N(y) = \sum_{n=0}^{\infty} A_n$$

where A_n, are Adomian polynomials of $y_0, y_1, \dots, y_n$ given by

$$A_n = \frac{1}{n!} \frac{d^n}{d\mu^n} \left[\sum_{n=0}^{\infty} \mu^i y_i \right] \Big|_{\mu=0}$$

For each values of n, the Adomian polynomial is computed as below:

$$A_1 = \frac{d}{d\mu} N(y_0 + y_1\mu) = [y_1 N'(y_0)]_{\mu=0} A_2$$

$$= \frac{1}{2!} \frac{d}{d\mu} [(y_1 + 2y_2\mu)N'(y_0 + y_1\mu)]_{\mu=0}$$

$$= y_2 N'(y_0) + \frac{y_1^2 N''(y_0)y_1 N'(y_0)}{2!} A_3 = A_3$$

$$= \frac{1}{3!} \frac{d}{d\mu} N\left[(y_2 + 3y_3\mu)N'(y_0 + y_1\mu) \right.$$
$$\left. + \frac{1}{2!}(y_1 + 2y_2\mu)^2 N''(y_0 + y_1\mu) \right] \Big|_{\mu=0}$$

$$= y_3 N'(y_0) + y_1 y_2 N''(y_0) + \frac{y_1^3 N'''(y_0)}{3!}$$

$$A_4 = \frac{1}{4!} \frac{d}{d\mu} \left[(y_3 + 4y_4\mu)N'(y_0 + y_1\mu) + (y_1 \right.$$
$$+ 2y_2\mu)(y_2 + 3y_3\mu)N''(y_0 + y_1\mu)$$
$$\left. + \frac{1}{3!}(y_1 + 2y_2\mu)N'''(y_0 + y_1\mu) \right] \Big|_{\mu=0}$$

$$= y_4 N'(y_0) + (y_1 y_3 + \frac{y_2^2}{2} N''(y_0) + \frac{y_1^2 y_1 N'''(y_0)}{3!}$$

$$+ \frac{y_1^4}{4!} N''''(y_0)$$

Continuing this course, we can get the other Adomian polynomials. Now going back on our equation

$$f = y - N(y),$$

$y = \sum_{n=0}^{\infty} y_n$ and

$$N(y) = \sum_{n=0}^{\infty} A_n$$

So that
$f = \sum_{n=0}^{\infty} y_n - \sum_{n=0}^{\infty} A_n$ Thus, we can identify

$$y_{n+1} = A_n(y_0, y_1, \ldots, y_n) n = 0, 1, 2 \ldots$$

We would further show the convergence of the above series by considering a system of nonlinear differential equations:

$$\frac{dy}{dt} = f(t, y), y \epsilon \mathbb{R}^d, f : \mathbb{R} \times \mathbb{R}^d \to \mathbb{R}^d,$$

with the initial condition $y(0) = y_0 \epsilon \mathbb{R}^d$. Assume that f is analytic near $y = y_0$ and $t = 0$. It is equivalent to solve the initial value problem for $\frac{dy}{dt} = f(t, y)$, $y \epsilon \mathbb{R}^d$, $f : \mathbb{R} \times \mathbb{R}^d \to \mathbb{R}^d$, and Volterra integral equation

$$y(t) = y_0 + \int_0^t f\big(s, y(s)\big) ds.$$

To set up the Adomian method, consider y in the series form:

$$y = y_0 + \sum_{n=1}^{\infty} y_n,$$

and write the nonlinear function $f(t, y)$ as the series of functions,

$$f(t, y) = \sum_{n=1}^{\infty} A_n(t, y_0, y_1, \ldots, y_n).$$

The dependence of A_n on t and y_0 may be non-polynomial. Formally, A_n is obtained by

$$A_n = \frac{1}{n!} \frac{d^n}{d\varepsilon^n} f\left(t, \sum_{i=0}^{\infty} \varepsilon^i y_i\right)\Big|_{\varepsilon=0}, n = 0, 1, 2, \ldots$$

where ε is a formal parameter Functions A_n are polynomials in $(y_1, \ldots, y_n)$, which are referred to as the Adomian polynomials.

In what follows, we shall consider a scalar differential equation and set

$$d = 1.$$

A generalization of $d \geq 2$ is possible but is technically longer.

The first four Adomian polynomials for $d = 1$ are listed as follows:

$$A_0 = f(t, y_0)$$

$$A_1 = y_1 f'(t, y_0)$$

$$A_2 = y_2 f'(t, y_0) + \frac{1}{2} y_1^2 f''(t, y_0)$$

$$A_3 = y_3 f'(t, y_0) + y_1 y_2 f''(t, y_0) + \frac{1}{6} y_1^3 f'''(t, y_0),$$

Where prime denote the partial derivatives with respect to y.

It was proven by Abbaoui and Cherruault (1994) that the Adomian polynomials A_n are defined by the explicit formulae:

$$A_n = \sum_{k=1}^n \frac{1}{k!} f^{(k)}(t, y_0)\left(\sum_{p_1 + \cdots + p_k = n} y_{p_1} \cdots y_{p_k}\right), n \geq 1$$

or, in an equivalent form, by

$$A_n = \sum |nk| = f^{(|k|)}(t, y_0) \frac{y_1^{k_1} \cdots y_n^{k_n}}{k_1! \ldots k_n!}, n \geq 1,$$

where $|k| = k_1 + \cdots + k_n$, and $|nk| = k_1 + 2k_2 + \cdots + nk_n$.

Khehlifa and Cherrault (2000) proved a bound for Adomian polynomials by

$$|A_n| \leq \frac{(n+1)^n}{(n+1)!} M^{n+1},$$

where

$$\sup_{t \epsilon J} |f^{(k)}(t, y_0)| \leq M$$

for a given time interval $J \subset \mathbb{R}$. Substituting $y = y_0 + \sum_{n=1}^{\infty} y_n$, and

$$f(t, y) = \sum_{n=1}^{\infty} A_n (t, y_0, y_1, \ldots, y_n).$$

into

$$y(t) = y_0 + \int_0^t f\big(s, y(s)\big) ds.$$

gives a recursive equation for y_{n+1} in terms of $(y_0, y_1, \ldots, y_n)$:

$$y_{n+1}(t) = \int_0^t A_n(s, y_0(s), y_1(s), \ldots, y_n(s)) ds, n = 0, 1, 2 \ldots$$

Hence, the convergence of series $y = y_0 + \sum_{n=1}^{\infty} y_n$, has been obtained by $y_{n+1}(t) = \int_0^t A_n(s, y_0(s), y_1(s), \ldots, y_n(s)) ds, n = 0, 1, 2 \ldots$ The rapid convergence of the solution is guaranteed by [7].

3.2. The Model of Edward et al. (2014)

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dose consecutively receive permanent immunity; otherwise they become susceptible to the disease again. The susceptible individuals enter the exposed compartment at the rate λ which is a force of infection. The exposed individuals are the ones who are infected but not infectious. After some time the exposed become infectious, they move from exposed state to infectious at the rate δ. An infected individual recover at rate η, and according to the nature of the disease; the recovered individuals are permanently immune.

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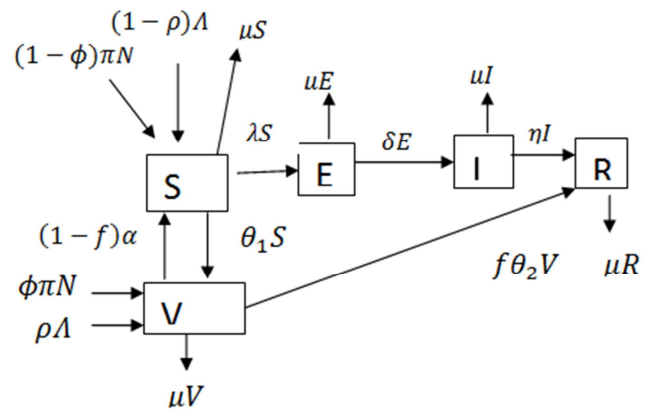

Figure 1. *The Flow Diagram for the Model of Edward et al. (2014).*

3.2.1. Model Parameters and Description of Edward et al. (2014)

Table 1. *Model Parameters and Description.*

α	The rate of waning of a vaccine
β	Probability of one infected individual to become infectious
Δ	Progression rate from latent to infectious
ϕ	Proportions of newborns who are vaccinated.
ρ	Proportions of immigrants who are vaccinated
a	Arrival rate
c	Per capita contact rate
θ_1	Fraction of individuals who receive a first dose vaccine
θ_2	Rate at which an individual receives a second dose vaccine
μ	Per capita natural mortality rate
π	Per capital birth rate
η	Recovery rate of treated infectious individuals
Λ	The recruitment rate of susceptible population
f	A fraction of population who receive second dose vaccine

3.2.2. The Model Equations of Edward et al. (2014)

The model equations using the model flow diagram in Figure 1 is:

$$\frac{dS}{dt} = (1 - \Phi)\pi N + (1 - \rho)\Lambda + (1 - f)\alpha V - (\lambda + \mu + \theta_1)S \quad (2)$$

$$\frac{dV}{dt} = \rho\Lambda + \Phi\pi N + \theta_1 S - \left((1 - f)\alpha + f\theta_2 + \mu\right)V \quad (3)$$

$$\frac{dE}{dt} = \lambda S - (\mu + \delta)E \quad (4)$$

$$\frac{dI}{dt} = \delta E - (\eta + \mu)I \quad (5)$$

$$\frac{dR}{dt} = \eta I + f\theta_2 V - \mu R \quad (6)$$

where λ is the force of infection and is given by

$$\lambda = \frac{\beta c I}{N}$$

The total population size is $N(t) = S(t) + V(t) + E(t) + I(t) + R(t)$

Adding equations $(3.12) - (3.16)$ gives

$$\frac{dN}{dt} = \Lambda + (\pi - \mu)N$$

For simplicity of analysis we normalize the equations. This can be done by scaling the population of each class by the total population.

We transform the model into proportion as follows: Let

$$s = \frac{S}{N}, v = \frac{V}{N}, e = \frac{E}{N}, i = \frac{I}{N} \text{ and } r = \frac{R}{N} \quad (7)$$

From=$\frac{S}{N}$, we have

$$sN = S$$

Differentiate the above using product rule of differentiation, we have

$$s\frac{dN}{dt} + N\frac{ds}{dt} = \frac{dS}{dt},$$

Therefore,

$$\frac{ds}{dt} = \frac{1}{N}\left[\frac{dS}{dt} - s\frac{dN}{dt}\right].$$

Substituting the values of $\frac{dS}{dt}$ and $\frac{dN}{dt}$ into the above equation, we have

$$\frac{ds}{dt}$$
$$= \frac{1}{N}\left[\begin{matrix}(1-\Phi)\pi N + (1-\rho)\Lambda + (1-f)\alpha V - (\lambda + \mu + \theta_1)S \\ -s(\Lambda + N(\pi - \mu))\end{matrix}\right]$$

$$\frac{ds}{dt}$$
$$= \left[\begin{matrix}(1-\Phi)\pi + (1-\rho)\frac{\Lambda}{N} + (1-f)\alpha\frac{V}{N} - (\lambda + \mu + \theta_1)\frac{S}{N} - s\frac{\Lambda}{N} \\ -s\frac{N}{N}(\pi - \mu)\end{matrix}\right]$$

Let $\frac{\Lambda}{N} = a$ be a constant, then

$$\frac{ds}{dt} =$$

$$[(1-\Phi)\pi + (1-\rho)a + (1-f)\alpha v - (\lambda + \mu + \theta_1)s - sa - s(\pi - \mu))]$$

Rearranging and collecting the like terms we have

$$\frac{ds}{dt} = (1-\phi)\pi + (1-\rho)a + (1-f)\alpha v - (\lambda + \theta_1 + a + \pi)s$$

Similarly, from equation of (3), we have

$$vN = V.$$

Differentiating the above using product rule, we have

$$v\frac{dN}{dt} + N\frac{dv}{dt} = \frac{dV}{dt}$$

$$\frac{dv}{dt} = \frac{1}{N}\left[\frac{dV}{dt} - v\frac{dN}{dt}\right].$$

Substituting the values of $\frac{dV}{dt}$ and $\frac{dN}{dt}$ into the above equation we have

$$\frac{dv}{dt} = \frac{1}{N}$$

$$\left[\rho\Lambda + \phi\pi N + \theta_1 s - ((1-f)\alpha + f\theta_2 + \mu)V - v\Lambda - Nv(\pi - \mu)\right]$$

$$\frac{dv}{dt} =$$

$$\left[\rho\frac{\Lambda}{N} + \phi\pi\frac{N}{N} + \theta_1\frac{S}{N} - ((1-f)\alpha + f\theta_2 + \mu)\frac{V}{N} - v\frac{\Lambda}{N} - \frac{N}{N}v(\pi - \mu)\right]$$

But $\frac{\Lambda}{N} = a$

$$\frac{dv}{dt} = \rho a + \phi\pi + \theta_1 s$$

$$-((1-f)\alpha + f\theta_2 + \mu)v - va - v(\pi - \mu)$$

Rearranging and collecting the like terms we have

$$\frac{dv}{dt} = \phi\pi + \rho a + \theta_1 s - ((1-f)\alpha + f\theta_2 + a + \pi)v$$

Similarly

$$\frac{de}{dt} = \lambda s - (\delta + a + \pi)e$$

$$\frac{di}{dt} = \delta e - (\eta + a + \pi)i$$

$$\frac{dr}{dt} = \eta i + f\theta_2 v - (a + \pi)r$$

Hence the transformed system becomes,

$$\frac{ds}{dt} = (1-\phi)\pi + (1-\rho)a + (1-f)\alpha v - (\beta ci + \theta_1 + a + \pi)s \tag{8}$$

$$\frac{dv}{dt} = \phi\pi + \rho a + \theta_1 s - ((1-f)\alpha + f\theta_2 + a + \pi)v \tag{9}$$

$$\frac{de}{dt} = \beta cis - (\delta + a + \pi)e \tag{10}$$

$$\frac{di}{dt} = \delta e - (\eta + a + \pi)i \tag{11}$$

$$\frac{dr}{dt} = \eta i + f\theta_2 v - (a + \pi)r \tag{12}$$

3.3. Analysis of the Model of Edward et al. (2014)

The model (8) – (12) is analyzed qualitatively to get insights into its dynamical features which give better understanding of the impact of immunization on the epidemiology of varicella zoster virus.

3.3.1. Positivity and Boundedness

In order to retain the biological validity of the model, we must prove that solutions to the system of differential equations are positive and bounded for all values of time. For example, concluding that a population is negative is not biologically feasible. Furthermore, the populations must remain finite since the human body can only be composed of a finite number of cells. In addition, boundedness and positivity illustrate that once infected, it is possible that the population of the virus will continue to exist beneath the detectable threshold without doing significant damage (Roemer, 2013). The next step in analyzing our model will be to prove positivity and boundedness for the system of differential equations. We will

do so by proving the following theorems.

3.3.2. Lemma 1 (Positivity)

Let $t_0 > 0$. In the model, if the initial conditions satisfy $S(0) > 0$, $V(0) > 0$, $E(0) > 0$, $I(0) > 0$, $R(0) > 0$ then for all $t \in [0, t_0]$, $S(t)$, $V(t), E(t), I(t)$, $R(t)$ will remain positive in $\mathbb{R}_+^4$.

$$\frac{dS}{dt} = (1 - \Phi)\pi N + (1 - \rho)\Lambda + (1 - f)\alpha V - (\lambda + \mu + \theta_1)S \geq -(\lambda + \mu + \theta_1)S$$

$$\frac{dV}{dt} = \rho\Lambda + \Phi\pi N + \theta_1 S - \big((1 - f)\alpha + f\theta_2 + \mu\big)V \geq -\big((1 - f)\alpha + f\theta_2 + \mu\big)V$$

$$\frac{dE}{dt} = \lambda S - (\mu + \delta)E \geq -(\mu + \delta)E$$

$$\frac{dI}{dt} = \delta E - (\eta + \mu)I \geq -(\eta + \mu)I$$

$$\frac{dR}{dt} = \eta I + f\theta_2 V - \mu R \geq -\mu R$$

Through basic differential equation methods we can resolve the inequalities and produce:

$$S(t) \geq e^{-(\lambda+\mu+\theta_1)t} \geq 0 \qquad (13)$$

$$V(t) \geq e^{-((1-f)\alpha+f\theta_2+\mu)t} \geq 0 \qquad (14)$$

$$E(t) \geq e^{-(\mu+\delta)t} \geq 0 \qquad (15)$$

$$I(t) \geq e^{-(\eta+\mu)t} \geq 0 \qquad (16)$$

$$R(t) \geq e^{-\mu t} \geq 0 \qquad (17)$$

Thus, for all $t \in [0, t_0]$, $S(t), V(t), E(t), I(t), R(t)$ will be positive and remain in $\mathbb{R}_+^4$.

3.3.3. Lemma 2 (Boundedness)

There exists an $S_M, V_M, E_M, I_M, R_M > 0$ such that for $S(t)$,

$$\lim_{t\to\infty} \sup(S + V + E + I + R)(t) \leq \lim_{t\to\infty} \sup\left(\frac{\Lambda}{\min\{\pi,\mu\}} + c_0 e^{-\min\{\pi,\mu\}t}\right) = \frac{\Lambda}{\min\{\pi,\mu\}} \qquad (20)$$

So choose

$$S_M = V_M = E_M = I_M = R_M = \frac{\Lambda}{\min\{\pi,\mu\}} \qquad (21)$$

Thus, $(S + V + E + I + R)(t)$ is bounded, so $S(t)$, $V(t)$, $E(t), I(t)$ and $R(t)$ are all bounded since $S(t), V(t), E(t), I(t), R(t) \leq (S + V + E + I + R)(t)$.

So, $S(t) \leq S_M$, $V(t) \leq V_M$, $E(t) \leq E_M$, $I(t) \leq I_M$, $R(t) \leq R_M$ for all $t \in [0, t_0]$.

3.3.4. Existence and Uniqueness of Solutions

Prior to conducting an in–depth analysis of the model, it is crucial to show that the solutions to the initial-value problem exist and are unique.

Theorem 1 (Existence) Let $t_0 > 0$. In the model, if the initial conditions satisfy $S(0) > 0$, $V(0) > 0$, $E(0) > 0$, $I(0) > 0, R(0) > 0$ then for all $t \in \mathbb{R} S(t), V(t), E(t), I(t), R(t)$ will exist in $\mathbb{R}_+^4$.

Proof: In the case of our model we have:

Proof: Positivity

We must prove that for all $t \in [0, t_0], S(t), V(t), E(t), I(t), R(t)$ will be positive in $\mathbb{R}_+^4$. We know that all of the parameters used in the system are positive. Thus, we can place lower bounds on each of the equations given in the model. Thus,

$V(t)$, $E(t)$, $I(t)$, $R(t)\lim_{t\to\infty} \sup(S(t)) \leq S_M$, $\lim_{t\to\infty} \sup(V(t)) \leq V_M, \lim_{t\to\infty} \sup(E(t)) \leq E_M,$ $\lim_{t\to\infty} \sup(I(t)) \leq I_M$, $\lim_{t\to\infty} \sup(R(t)) \leq R_M$ for all $t \in [0, t_0]$.

Proof: Boundedness

We must prove that for all $t \in [0, t_0]$, $S(t)$, $V(t)$, $E(t)$, $I(t)$, $R(t)$ will be bounded. We know that all of the constants used in the system are positive.

$$\frac{dS}{dt} + \frac{dV}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = \Lambda + (\pi - \mu)N$$

Since all of the constants are positive,

$$\frac{d(S+V+E+I+R)}{dt} \leq \Lambda + \min\{\pi, \mu\}(S + V + E + I + R)(t) \quad (18)$$

which implies,

$$(S + V + E + I + R)(t) \leq \frac{\Lambda}{\min\{\pi,\mu\}} + c_0 e^{-\min\{\pi,\mu\}t} \qquad (19)$$

Taking the lim sup of both sides,

$$x = \begin{bmatrix} S(t) \\ V(t) \\ E(t) \\ I(t) \\ R(t) \end{bmatrix}$$

and

$$f(x) = \begin{bmatrix} (1 - \Phi)\pi N + (1 - \rho)\Lambda + (1 - f)\alpha V - (\lambda + \mu + \theta_1)S \\ \rho\Lambda + \Phi\pi N + \theta_1 S - \big((1 - f)\alpha + f\theta_2 + \mu\big)V \\ \lambda S - (\mu + \delta)E \\ \delta E - (\eta + \mu)I \\ \eta I + f\theta_2 V - \mu R \end{bmatrix} \quad (22)$$

Note that f has a continuous derivative on $\mathbb{R}^4$ and thus, f is locally Lipschitz in $\mathbb{R}^4$. Hence, by the Fundamental Existence and Uniqueness Theorem as well as the lemmas proved on positivity and boundedness of solutions, we know that there exists a unique, positive, and bounded solution to

the ordinary differential equations given in (8) – (12).

3.3.5. Disease Free Equilibrium (DFE), P_0

The disease free equilibrium of the model system (8) – (12) is obtained by setting $\frac{dv}{dt} = \frac{ds}{dt} = \frac{de}{dt} = \frac{di}{dt} = \frac{dr}{dt} = 0$ and P_0 of the model system (8) – (12) exist and given by:

$$P_0(s^*, v^*, e^*, i^*, r^*) = (s_0^*, v_0^*, 0, 0, r_0^*)$$

$$-(\lambda + \theta_1 + a + \pi)s + (1 - f)\alpha v + (1 - \phi)\pi + (1 - \rho)a = 0$$

$$(\theta_1 s - ((1 - f)\alpha + f\theta_2 + a + \pi)v + \phi\pi + \rho a = 0$$

$$f\theta_2 v - (a + \pi)r = 0$$

which implies:

$$s_0^* = \frac{(1 - f)\alpha(a + \pi) + (f\theta_2 + a + \pi)[(1 - \phi)\pi + (1 - \rho)a]}{(a + \pi)(1 - f)\alpha + (f\theta_2 + a + \pi)(\theta_1 + a + \pi)}$$

$$v_0^* = \frac{(a + \pi)(\theta_1 + \phi\pi + \rho a)}{\theta_1(f\theta_2 + a + \pi) + (a + \pi)[(1 - f)\alpha + f\theta_2 + a + \pi]}$$

$$r_0^* = \frac{f\theta_2(\theta_1 + \phi\pi + \rho a)}{\theta_1(f\theta_2 + a + \pi) + (a + \pi)[(1 - f)\alpha + f\theta_2 + a + \pi]}$$

Thus the Disease Free Equilibrium (DFE) point denoted by P_0 of the model system (3.18) – (3.22) exists and is given by: $P_0(s^*, v^*, e^*, i^*, r^*) = (s_0^*, v_0^*, 0, 0, r_0^*)$

3.3.6. The Basic Reproduction Number, R_0

Poletti *et al.* (2013) defined the basic reproduction number denoted by R_0, as the average number of secondary infections caused by an infectious individual during his or herentire periodof infectiousness. The basic reproduction number is an important non-dimensional quantity in epidemiology as it sets the threshold in the study of a disease both for predicting its outbreak and for evaluating its control strategies. Thus, whether a disease becomes persistent or dies out in a community depends on the value of the reproduction number, R_0.

Furthermore, stability of equilibrium can be analyzed using R_0. If $R_0 < 0$ it means that every infectious individual will cause less than one secondary infection and hence the disease will die out and when $R_0 > 1$, every infectious individual will cause more than one secondary infection and hence the disease will invade the population. A large number of R_0 may indicate the possibility of a major epidemic. For the case of a model with a single infected class, R_0 is simply product of the

In case there is no disease; $e = i = 0$ the sum of susceptible and vaccinated populations is equal to total population.

That is to say $s_0^* + v_0^* + r_0^* = 1$

Consequently, system (3.18) - (3.22) reduces to:

infection rate and the mean duration of the infection.

In more complicated epidemics we compute the basic reproduction number, R_0 using the next generation operator approach by Van den and Watmough (2002).

From the system Equations (8) – (12) we define $\mathcal{F}_i$ and $\mathcal{V}_i$ as

$$\mathcal{F}_i = \begin{bmatrix} \beta cis \\ 0 \end{bmatrix}, \tag{23}$$

$$\mathcal{V}_i = \begin{bmatrix} (\delta + a + \pi)e \\ \delta e - (\eta + a + \pi)i \end{bmatrix} \tag{24}$$

We differentiate F_i with respect to e and i to get

$$\mathcal{F} = \begin{bmatrix} 0 & \beta cs \\ 0 & 0 \end{bmatrix} \tag{25}$$

We differentiate V_i with respect to e and i and get

$$\mathcal{V}\mathcal{V} = \begin{bmatrix} (\delta + a + \pi) & 0 \\ -\delta & (\eta + a + \pi) \end{bmatrix} \tag{26}$$

We find the inverse of V and get

$$\mathcal{V}^{-1} = \begin{bmatrix} \frac{1}{(\delta+a+\pi)} & 0 \\ (\delta + a + \pi)(\eta + a + \pi) & \frac{1}{(\eta+a+\pi)} \end{bmatrix} \tag{27}$$

$$\mathcal{F}\mathcal{V}^{-1} = \begin{bmatrix} \frac{\partial \mathcal{F}_i(E_0)}{\partial x_j} \end{bmatrix} \begin{bmatrix} \frac{\partial \mathcal{V}_i(E_0)}{\partial x_j} \end{bmatrix}^{-1} \begin{bmatrix} 0 & \beta cs_0^* \\ 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{(\delta+a+\pi)} & 0 \\ (\delta + a + \pi)(\eta + a + \pi) & \frac{1}{(\eta+a+\pi)} \end{bmatrix} = \begin{bmatrix} \frac{\beta cs_0^*\delta}{(\delta+a+\pi)(\eta+a+\pi)} & \frac{\beta cs_0^*}{\eta+a+\pi} \\ 0 & 0 \end{bmatrix} \tag{28}$$

The eigen values, λ of this equation above can be computed from the characteristic equation: $|\mathcal{F}\mathcal{V}^{-1} - \lambda I| = 0$, and we see that from our matrix that

$$\lambda_1 = \frac{\beta cs_0^*\delta}{(\delta+a+\pi)(\eta+a+\pi)} \text{ and } \lambda_2 = 0$$

Obviously, λ_1 is the dorminant eigenvalue and becomes

equal to R_e of the model.

Therefore, if we substitute s_0^* from equation (22) above into λ_1, we get effective reproduction number denoted by R_e in the equation (14) below

$$R_e = \frac{\beta c \delta \{(1-f)\alpha(\phi\pi+\rho a)+((1-f)\alpha+f\theta_2+a+\pi)(1-\phi)\pi+(1-\rho)a]\}}{\{\theta_1(f\theta_2+a+\pi)+(a+\pi)[(1-f)\alpha+f\theta_2+a+\pi]\}(\delta+a+\pi)(\eta+a+\pi)} \quad (29)$$

where there is no any control strategy, then $\theta_1 = \theta_2 = \phi = \rho = 0$

and hence $f = 0, \alpha = 0$, so we get basic reproduction number

$$R_0 = \frac{\beta c \delta}{(\delta+a+\pi)(\eta+a+\pi)} \quad (30)$$

3.3.7. Global Stability of Disease Free Equilibrium State (DFE)

In this section, we analyze the global stability of disease–free steady state. Here we use the Next Generation Matrix Approach by Castillo-Chavez et al., (2002). Now we state two conditions which guarantee the global stability of the disease free state. We rewrite the model system (8) – (12) as

$$\frac{dX}{dt} = F(X,Z),$$

$$\frac{dZ}{dt} = G(X,Z), G(X,0) = 0, \quad (31)$$

where $X = (S)$ and $Z = (E, I)$, with $X \epsilon \mathbb{R}$ denotes the number of uninfected individuals and $X \epsilon \mathbb{R}^2$ denoting (its components) the number of infected individuals including latent and infectious. The disease-free equilibrium is now denoted by $Q_0 = (X^0, 0)$. The following conditions (H_1) and (H_2) must be met to guarantee a local asymptotic stability:

(H_1) for $\frac{dX}{dt} = F(X^0, 0), X^0$ is globally asymptotically stable (g.a.s),

$G(X,Z) = AZ - G(X,Z), H_2$ where $G(X,Z) \geq 0$ for

$$(X,Z) \epsilon \Omega \quad (32)$$

Where $A = D_2 G(X^0, 0)$ is an M-matrix (the off-diagonal elements of A are non-negative) and Ω is the region where the model is practically possible. Then the following lemma holds:

Lemma 3

The fixed point $Q_0 = (X^0, 0)$ is globally asymptotic stable (g.a.s) equilibrium of system (8)-(12) provided that $R_e < 1$ (l.a.s) and that the assumptions (H_1) and (H_2) are satisfied. We state the following theorem:

Theorem 1

Suppose $R_e < 1$. The disease free equilibrium P_0 is globally asymptotically stable.

Proof:

The system equation (8)-(12) can be expressed in the form of equation (16) and thus we get

$$X=(s, r), Z=(e, i),$$

$$A = \begin{bmatrix} -(\delta+a+\pi) & \beta s^* \\ \delta & -(\eta+a+\pi) \end{bmatrix}$$

$$G = \begin{bmatrix} -(\delta+a+\pi) & \beta s \\ \delta & -(\eta+a+\pi) \end{bmatrix}$$

We need to show that (H_2) holds in the system equation (8) - (12).

$$G(X,Z) = AZ - G(X,Z),$$

$$\Rightarrow G(X,Z) = AZ - G(X,Z)$$

$$G = \begin{bmatrix} -(\delta+a+\pi) & \beta s^* \\ \delta & -(\eta+a+\pi) \end{bmatrix}$$

$$\begin{bmatrix} e \\ i \end{bmatrix} - \begin{bmatrix} -(\delta+a+\pi) & \beta s \\ \delta & -(\eta+a+\pi) \end{bmatrix} \begin{bmatrix} e \\ i \end{bmatrix}$$

$$= \begin{bmatrix} 0 & \beta(s^*-s) \\ 0 & 0 \end{bmatrix} \begin{bmatrix} e \\ i \end{bmatrix}$$

$$= \begin{bmatrix} \beta(s^*-s)i \\ 0 \end{bmatrix}$$

Since $s^* \geq s$ then $\hat{G} \geq 0$ therefore (H_1) and (H_2) are satisfied. Hence the disease free equilibrium point is globally asymptotically stable.

4. Results

4.1. Application of Adomian Decomposition Method to the Model Equations of Edward et al. (2014)

The model (3.18) – (3.22) as shown below:

$$\frac{ds}{dt} = (1-\phi)\pi + (1-\rho)a + (1-f)\alpha v - (\beta c i + \theta_1 + a + \pi)s$$

$$\frac{dv}{dt} = \phi\pi + \rho a + \theta_1 s - ((1-f)\alpha + f\theta_2 + a + \pi)v$$

$$\frac{de}{dt} = \beta cis - (\delta + a + \pi)e$$

$$\frac{di}{dt} = \delta e - (\eta + a + \pi)i$$

$\frac{dr}{dt} = \eta i + f\theta_2 v - (a + \pi)r$ will be considered in finding the solution of the system of the model equations using ADM.

To apply the Adomian Decomposition Method to the equations above, we will find the canonical transformation of each equation. The equivalent canonical transform of this system gives

$$s(t) = s(0) + (1 - \phi)\pi t + (1 - \rho)at + (1 - f)\alpha \int_0^t v \, dt - \beta c \int_0^t sidt - (\theta_1 + a + \pi) \int_0^t sdt \tag{33}$$

$$v(t) = v(0) + \phi\pi t + \rho at + \theta_1 \int_0^t s \, dt - \left((1 - f)\alpha + f\theta_2 + a + \pi\right) \int_0^t v \, dt \tag{34}$$

$$e(t) = e(0) + \beta c \int_0^t si \, dt - (\delta + a + \pi) \int_0^t e \, dt \tag{35}$$

$$i(t) = i(0) + \delta \int_0^t e \, dt - (\eta + a + \pi) \int_0^t i \, dt \tag{36}$$

$$r(t) = r(0) + \eta \int_0^t i \, dt + f\theta_2 \int_0^t v \, dt - (a + \pi) \int_0^t r \, dt \tag{37}$$

In Adomian Decomposition Method, the equations above are considered to be the sum of the following series:

$$s = \sum_{n=0}^{\infty} s_n, v = \sum_{n=0}^{\infty} v_n, e = \sum_{n=0}^{\infty} e_n, i = \sum_{n=0}^{\infty} i_n, r = \sum_{n=0}^{\infty} r_n \tag{38}$$

Then we approximate the nonlinear terms in the system as shown below

$$si = \sum_{n=0}^{\infty} A_n(s_0, \dots, s_n, i_0, \dots, i_n) \tag{39}$$

where

$$A_n = \frac{1}{n!} \left[\frac{d^n (\sum_{k=0}^{\infty} s_k \lambda^k)(\sum_{k=0}^{\infty} I_k \lambda^k)}{d\lambda^n} \right]_{\lambda=0} \tag{40}$$

The nonlinear function A_n is called the Adomian polynomial. Substituting we get:

$$\sum_{n=0}^{\infty} s_n = s(0) + (1 - \phi)\pi t + (1 - \rho)at + (1 - f)\alpha \int_0^t \sum_{n=0}^{\infty} v_n \, dt - \beta c \int_0^t \sum_{n=0}^{\infty} A_n - (\theta_1 + a + \pi) \int_0^t \sum_{n=0}^{\infty} s_n \, dt$$

$$\sum_{n=0}^{\infty} v_n = v(0) + \phi\pi t + \rho at + \theta_1 \int_0^t \sum_{n=0}^{\infty} s_n \, dt - \left((1 - f)\alpha + f\theta_2 + a + \pi\right) \int_0^t \sum_{n=0}^{\infty} v_n \, dt$$

$$\sum_{n=0}^{\infty} e_n = e(0) + \beta c \int_0^t \sum_{n=0}^{\infty} A_n \, dt - (\delta + a + \pi) \int_0^t \sum_{n=0}^{\infty} e_n \, dt \quad \sum_{n=0}^{\infty} i_n = i(0) + \delta \int_0^t \sum_{n=0}^{\infty} e_n \, dt - (\eta + a + \pi) \int_0^t \sum_{n=0}^{\infty} i_n \, dt$$

$$\sum_{n=0}^{\infty} r_n = r(0) + \eta \int_0^t \sum_{n=0}^{\infty} i_n \, dt + f\theta_2 \int_0^t \sum_{n=0}^{\infty} v_n \, dt - (a + \pi) \int_0^t \sum_{n=0}^{\infty} r_n \, dt$$

From the equation above, we define the following scheme:

$$s_0 = s(0) + (1 - \phi)\pi t + (1 - \rho)at,$$

$$v_0 = v(0) + \phi\pi + \rho at,$$

$$e_0 = e(0) i_0 = i(0) r_0 = r(0) \text{ and} \tag{41}$$

$$s_{n+1} = (1 - f)\alpha \int_0^t v_n \, dt - \beta c \int_0^t A_n - (\theta_1 + a + \pi) \int_0^t s_n \, dt \tag{42}$$

$$v_{n+1} = \theta_1 \int_0^t s_n \, dt - \left((1 - f)\alpha + f\theta_2 + a + \pi\right) \int_0^t v_n \tag{43}$$

$$e_{n+1} = \beta c \int_0^t A_n \, dt - (\delta + a + \pi) \int_0^t e_n \, dt \tag{44}$$

$$i_{n+1} = \delta \int_0^t e_n \, dt - (\eta + a + \pi) \int_0^t i_n \, dt \tag{45}$$

$$r_{n+1} = \eta \int_0^t i_n \, dt + f\theta_2 \int_0^t v_n \, dt - (a + \pi) \int_0^t r_n \, dt \tag{46}$$

we generate the Adomian polynomials as follows:

$$A_n = \frac{1}{n!} \left[\frac{d^n}{d\lambda^n} \left(\sum_{k=0}^\infty s_k \lambda^k \right) \left(\sum_{k=0}^\infty i_k \lambda^k \right) \right]_{\lambda=0} \tag{47}$$

at $n = 0$,

$$A_0 = s_0 i_0$$

$$n = 1,$$

$$A_1 = \frac{d}{d\lambda}(s_0 + s_1\lambda)(i_0 + i_1\lambda)$$

$$A_1 = s_1(i_0 + i_1) + i_0(s_0 + s_1\lambda) = s_1 i_0 + s_0 i_1$$

$$n = 2,$$

$$A_2 = \frac{1}{2}\frac{d}{d\lambda}[(s_1 + 2s_2\lambda)(i_0 + i_1\lambda)(i_1 + 2i_2\lambda)(s_0 + s_1\lambda)]$$

$$A_2 = s_2 i_0 + s_1 i_1 + s_0 i_2$$

$$n = 3,$$

$$A_3 = \frac{1}{3}\frac{d}{d\lambda}(s_2 + 3s_3\lambda)(i_0 + i_1\lambda) + (i_1 + 2i_2\lambda)(s_1 + 2s_2\lambda) + (i_2 + 3i_3\lambda)(s_0 + s_1\lambda)$$

$$A_3 = s_3 i_0 + s_2 i_1 + s_1 i_2 + s_0 i_3.$$

$$A_4 = s_4 i_0 + s_3 i_1 + s_2 i_2 + s_1 i_3 + s_0 i_4.$$

The first Adomian Polynomials are

$$A_0 = s_0 i$$

$$A_1 = s_1 i_0 + s_0 i_1$$

$$A_2 = s_2 i_0 + s_1 i_1 + s_0 i_2$$

$$A_3 = s_3 i_0 + s_2 i_1 + s_1 i_2 + s_0 i_3$$

$$A_4 = s_4 i_0 + s_3 i_1 + s_2 i_2 + s_1 i_3 + s_0 i_4$$

$$A_5 = s_5 i_0 + s_4 i_1 + s_3 i_2 + s_2 i_3 + s_1 i_4 + s_0 i_5$$

$$A_6 = s_6 i_0 + s_5 i_1 + s_4 i_2 + s_3 i_3 + s_2 i_2 + s_1 i_3 + s_0 i_6$$

Now we can go ahead and solve for the system of the model equations
(8) – (12) as shown below.
Computation for **s**-Series
Consider

$$s_{n+1} = (1-f)\alpha \int_0^t v_n \, dt - \beta c \int_0^t si - (\theta_1 + a + \pi) \int_0^t s_n \, dt$$

at $n = 0$,

$$s_1 = (1-f)\alpha \int_0^t v_0 \, dt - \beta c \int_0^t A_0 - (\theta_1 + a + \pi) \int_0^t s_0 \, dt$$

$$s_1 = \{(1-f)\alpha v_0 - (\beta c s_0 i_0 + \theta_1 + a + \pi)\}t$$

$$= (1 - f)\alpha v_0 t - \beta c v_0 t - (\theta_1 + a + \pi)s_0 t$$

At $n = 1$,

$$s_2 = (1 - f)\alpha \int_0^t v_1 \, dt - \beta c \int_0^t A_1 \, dt - (\theta_1 + a + \pi) \int_0^t s_1 \, dt$$

$$s_2 = (1 - f)\alpha \int_0^t \{\theta_1 s_0 - ((1 - f)\alpha + f\theta_2 + a + \pi)v_0\}t \, dt - \beta c \int_0^t (s_1 i_0 + s_0 i_1) \, dt - (\theta_1 + a + \pi) \int_0^t s_1 \, dt$$

The result is gotten by substituting the values of s_1, A_1, v_1 in the equation above and solving.

Therefore

$$\sum_{n=0}^{\infty} s_n = s_0 + s_1 + s_2 + s_3 + \cdots$$

is the approximate solution for s.

Approximate Solution for $S(t)$

$$s(t) = \sum_{n=0}^{\infty} s_n$$

$$s(t) = s_0 + s_1 + s_2 + s_3 + \cdots$$

Thus, the summation of this series gives us an approximate solution.

This is therefore the third order approximate solution for $s(t)$. This can be obtained to the nth term but for the purpose of this research, we have to truncate at the third term. The same will apply to the other computations

4.2. Computing Approximate Solutions by Introducing Parameter Values

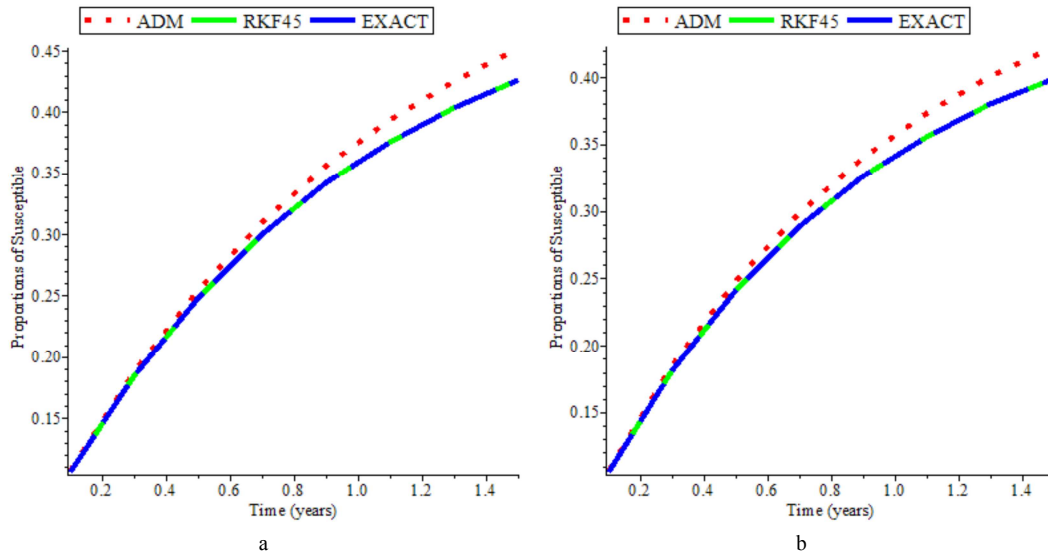In this section, we will make use of estimated parameter values to derive approximate solutions. To enhance our work, we use tables for various cases. Note that the values used in this work are the estimated values from Edward *et al.* (2014). These parameters are clearly shown for various cases of ϕ, θ_1 and θ_2.

Recall that ϕ is the proportions of newborns that are vaccinated while θ_1 and θ_2 are the fraction of individuals who receive a first dose vaccine and the rate at which an individual receives a second dose vaccine respectively. $\phi = 0$, signifies no fraction of the population of new born was vaccinated. $\phi = 0.5$ shows half of the population of new born was vaccinated while $\phi = 1$ shows that all the population of new born were vaccinated. This also explains the significance of each fraction for θ_1 and θ_2 as the case may be.
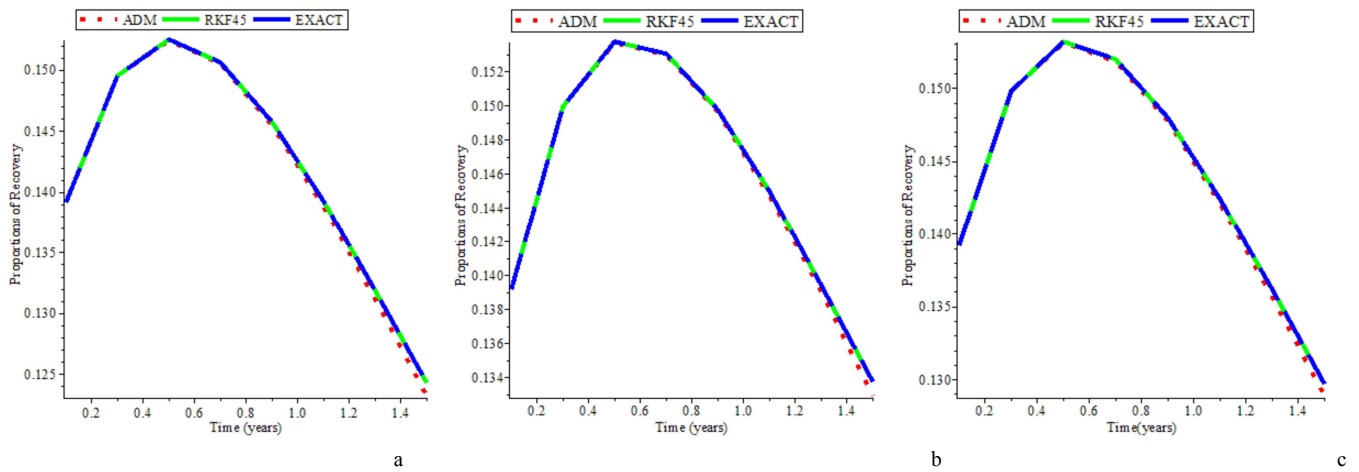
4.3. Graphical Representation of ADM, RKF45 and Exact Solution from the Various Tables

We shall consider the graphical behavior of ADM, RKF45 and the exact solution for various cases of ϕ, θ_1 and θ_2 respectively to enable us analyze the effect of the vaccination introduced into the system both at birth and adulthood.
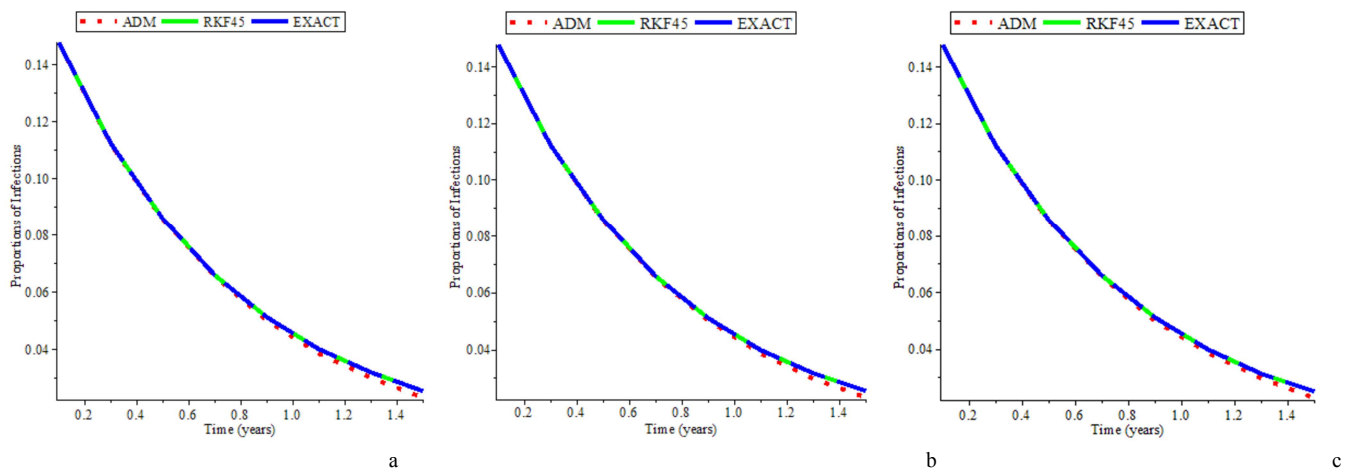
First and second dose vaccination was introduced at adult stage to ascertain the behavior of the disease after close monitoring.
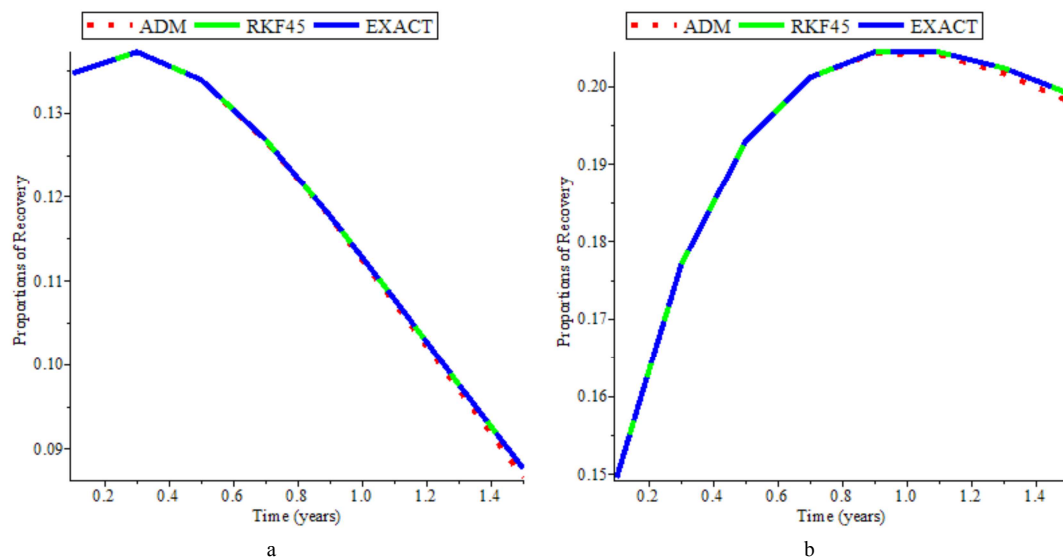

Figure 2. (a, b): Comparison plot of Susceptible Population in an Outbreak varing the proportion of adults who take dose 2 ($\theta_2 = 0, 1.0$) considering Table 11 and 13 using ADM, RKF45 Method.

Figure 3. *(a, b, c): Comparison plot of Recovery Population in an Outbreak varing the proportion of adults who take dose 1 (θ_1=0, 0.5, 1.0) considering Table 8, 9 and 10 using ADM, RKF45 Method.*

Figure 4. *(a, b, c): Comparison plot of Infective Population in an Outbreak varing the proportion of adults who take dose 1 (θ_1=0, 0.5, 1.0) considering Table 8, 9 and 10 using using ADM, RKF45 Method.*

Figure 5. *Comparison plot of Recovery Population in an Outbreak varing the proportion of adults who take dose 2 (θ_2=0, 1.0) considering Table 11, and 13 using ADM, RKF45 Method.*

These graphs will also help us see the efficacy of the ADM in comparison to the conventional Rungekutta Method for future application in solving nonlinear equations.

5. Discussion

5.1. Discussion on Proportion of Individuals Who Received First Dose of Vaccine

The first dose of vaccination yielded significant effect as the number of susceptible and infective drop immediately the whole population of adults were given the first dose (in the case of $\theta_1 = 1$). It can be seen from Figure 4 that increase in vaccination proportion of susceptible adults, tend to reduce the proportion of susceptible and as a result reduction in number of sick humans and hence Chicken Pox diminishes. This decline of the proportion of susceptible and infective changed as the first dose began to wane. We had the entire population becoming infected leading to a resurface of the disease. This trend if not controlled on time could lead to more outbreak of Chicken Pox that may become endemic. This call for the introduction of the second dose vaccination θ_2.

5.2. Discussion on Rate at Which Individuals Received the Second Dose of Vaccine

It can be seen from Figure 5 that the more increase in the coverage of dose two among the humans, the more the decrease in the proportion of susceptible but in a small amount. This is due to the fact that susceptible humans are not directly related to individual who receive second dose. Susceptible humans will receive dose two only if they had already received dose one otherwise they do not receive dose two. Such a condition is what made a slight decrease in susceptible population even when more individuals are vaccinated in dose two because such people might be newborns or recruited hence not or less affecting population of susceptible. So in general this practice has less but significant impact in reducing the disease.

5.3. Discussion on Recovery Rate of Population when Vaccinated

It can be seen from Figure 6 that the number of recovered individuals increases with increase in the vaccination coverage of newborns. It can also be seen that when no newborns are vaccinated, the proportion of recovered population decline after a slight increase which might be due to natural immunity of the sick ones. This decline in the proportion of recovery is Perhaps due to loss of immunity which wanes with time. In the Figure 6, the graph with the grey line shows that with a 50% of newborns being vaccinated, there is a significant increase in the population of recovered humans. However as time increases we note a slight decline in the recovered humans, this agrees with our intuition that the first vaccine wanes with time and this calls for the next boosting up vaccine coverage. The top most graph, with the light shade of black of Figure 4 shows the maximum proportion of recovered humans when the vaccination coverage is 100%. The graph is seen increasing and remains almost constant after reaching the maximum point. This suggests that when vaccination coverage is optimal then the disease can be eradicated from the community.

5.4. Comparison Between ADM and RKF45 Solution

The Adomian decomposition method determine solutions in a close form by using initial conditions. These solutions are continuous while Runge-Kutta method gave us the solutions at fixed points. This feature of the ADM makes it more efficient in solving various problems.

The Adomian decomposition method also avoid the difficulties and massive computational work that we encountered in the conventional method where more tedious computations are involved.

Both Method revealed that the rate of decline of the recovery population is faster for the nonvaccinated population. The entire graphical representation for both methods shows that the convergence of the Adomian decomposition method (ADM) is faster than the Runge-Kutta-Fehlberg method (RKF45). This confirmed and proved the argument by Babolian *et al.* (2004) of the Adomian decomposition method being a method that provides solution in a rapidly convergent series with components that can be elegantly computed.

6. Conclusion and Recommendations

6.1. Conclusion

We have reviewed the VZV model of Edward *et al.* (2014), and also applied the ADM to it. We have also computed approximate solutions of this model using the ADM. The results of this method are compared to that of Fourth-Fifth-order Runge-Kutta-Fehlberg method (RKF45) in table 5 which affirms the reliability and accuracy of the Adomian decomposition method. It also revealed its reduction in the size of computation domain. This gives the method a wider applicability. The Adomian decomposition method also shows a high degree of accuracy, and in most cases, accurate with lower step sizes.

Numerical solutions of the model have shown that the combination of vaccination and treatment is the most effective way to combat the epidemiology of VZV in the community.

6.2. Recommendation

Based on the findings in the research study, to minimize VZV transmission in a population, this study recommends that, the combination of vaccination and treatment should be implemented. This is due to the fact that, vaccination reduces the likelihood of an individual to be infected while treatment of latently infected people reduces the progression rate to infectious stage and also, treatment of infectious people will stop them from transmitting the disease.

Hence, we equally recommend that further research should be made on the application of the Adomian decomposition method to the system of VZV model with the combination of vaccination and treatment as the strategy.

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