Variational Iteration Method for Solving an Infectious Disease Model

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ABSTRACT

In this paper, we present a deterministic model that captures the essential dynamics of infectious diseases. Variational Iteration Method (VIM) is applied to attempt the series solution of the model. The efficiency of the VIM in solving the model is confirmed by classical fourth-order Runge-Kutta method implemented in Maple 18. The comparisons between the VIM and Runge-Kutta (RK4) solutions are made and there exists positive correlation between the results obtained by the two methods. The outcome of comparison between the VIM and RK4 validates the potential of the VIM in coping with the analysis of modern epidemics.

(Keywords: infectious disease, Variational Iteration Method, Runge-Kutta method)

INTRODUCTION

Infectious diseases are health issues characterized by a departure from sound state of health triggered by structural changes such that the normal body function is impaired, making it extremely difficult for the organs of the body to perform properly (Asor and Ugwu, 2011). Infectious diseases are also called transmissible diseases or communicable diseases due to their ability to spread from one species or person to another through a replicating agent. The spread of an infectious disease may be attributed to one or more different pathways such as contact with infected individuals. The infectious agents may also be ingested through body fluids, food, airborne inhalation, contaminated objects or through vector-borne transmission (Asor and Ugwu, 2011).

The concealed and impulsive nature of epidemic diseases has been a major source of panic and superstitions since the beginning of human civilization. The global fear accompanied the emergence of avian flu and SARS in Southeast Asia and Ebola in Africa are cases that our feeling of horror increases with our awareness of the outbreaks. One of the concerns of infectious modeling is studying the transmission of diseases in a host population, both in space and time. The first epidemiological model was credited to Daniel Bernoulli in 1760 (d'Onofrio, 2002) which was aimed at evaluating the effect of variolation on man's life expectancy. However, there was a gap in infectious modeling until the start of the 20th century with the innovating work of Hammer (Shulgin et al. 1998) on measles and malaria, respectively.

The past decade has been characterized by the fast emergence and development of a considerable theory of epidemics. The much-celebrated threshold dynamics which is one the major quantity in epidemiology was derived in 1927 by Kermack and Makendrick (Stone et al., 2000). The historic work of Kermack and Makendrick was followed by the radical work of Bartlett (Bartlett, 1960) who investigated models and data to examine the circumstances that influence disease persistence in host populations. Perhaps, the first groundbreaking book on mathematical modeling regarding the epidemiological systems was introduced by Bailey (Keeling and Grenfell, 2002) which led to the appreciation of the significance of modeling in decision making regarding the public health issues (Grossman, 1980). Given the range of epidemic diseases studied since 1950s, a remarkable class of epidemic models has been designed.

The dynamics of malaria in four ecological zones of Nigeria was studied in (Okwa, 2009). The researcher discovered that the infected mosquitoes were concentrated in the rain forest and were more active during rainy season. (Githeko and Ndegwa, 2001) designed a model to predict malaria epidemic and discovered that the rate of spread of infection is a function of the level of precipitation and temperature. (Koriko and Yusuf, 2008) also proposed a model to study the dynamics of tuberculosis and found that the spread of the disease depends not only on the population of the actively infected individuals at the initial time but also on the disease morbidity at a given time. More studies on infectious diseases are available in (Asor and Ugwu, 2011; Zaman et al., 2017; Wiwanitkit, 2017).

The aim of this paper is to employ Variational Iteration Method to solve the model introduced in (Adebisi et al, 2019) and to verify the validity of the VIM in solving the model using the computer in-built Maple 18 classical fourth-order Runge-Kutta method as the base.

MATERIALS AND METHOD

A compartmental model is adopted to analyze the transmission dynamics of disease in a human population. The model is divided into subpopulations based on the epidemiological status of individuals in the population. The susceptible population is generated from the daily recruitment of birth at the rate. It is increased as a result of loss of immunity after recovery at the rate and decreases due to vaccination and natural death at the rate, respectively.

The infectious class is generated at the rate when there is interaction between the susceptible and the infectious individuals. The infectious class however reduces through the natural death rate and through the recovery from infection and the disease induced death at the rates and respectively. Furthermore, the Recovery subclass is generated from vaccinated susceptible subpopulation and recovered infected individuals at the rate and respectively. They are reduced due to loss of immunity from recovery and natural death at the rate and respectively. To indicate this mathematically, we have:

$$\frac{dS}{dt} = \beta - \alpha SI - (\rho + \mu)S + \sigma R \qquad \textbf{(1)}$$

$$\frac{dS}{dt} = \beta - \alpha SI - (\rho + \mu)S + \sigma R \qquad \textbf{(2)}$$

$$\frac{dR}{dt} = \gamma I - (\mu + \sigma)R + \rho S \qquad \textbf{(3)}$$

VARIATIONAL ITERATION METHOD

To illustrate the basic idea of variational iteration method, Abbasbandy and Shivanian, (1999), Abdou and Soliman (2005), and Akinboro et al. (2014) gave the analysis of VIM as follows: Given the general differential equation of the form:

$$Ny + Ly = g(x) \qquad \textbf{(4)}$$

Where *N* is a non-linear operator, *L* is a linear operator and *g(x)* is a non-homogenous term of the differential equations. The construction of correctional function for the equation is given as:

$$y_{n+1}(x) = y_n(x) + \int_0^x \lambda\{Ly_n(s) + N\tilde{y}_n(s) - g(s)\}ds \qquad \textbf{(5)}$$

Where λ is a Lagragian multiplier which can be expressed as:

$$\lambda(\eta) = \frac{(-1)^n}{(n-1)!}(\eta - t)^{n-1} \qquad \textbf{(6)}$$

where n is the highest order of the differential equation.

Subject to the initial conditions S_0=3000, I_0=200, R_0=100.

Solution of the Model Using Variational Iteration Method

We present the analysis of the system of equations governing the model using variational iteration method. Following the same approach as in Momani and Abuasad (2006), we obtain the correctional function as:

$$S_{n+1}(t) = S_n(t) - \int_0^t \{S_{n'}(x) - \beta + \alpha\tilde{S}_n(x)\tilde{I}(x) + (\rho+\mu)\tilde{S}_n(x)\}dx$$

$$I_{n+1}(t) = I_n(t) - \int_0^t \{I_{n'}(x) - \alpha\tilde{S}_n(x)\tilde{I}(x) + (\delta+\mu+\gamma)\tilde{I}_n(x)\}dx$$

$$R_{n+1}(t) = R_n(t) - \int_0^t \{R_{n'}(x) - \gamma\tilde{I}_n(x) + (\mu+\delta)\tilde{R}_n(x) + \rho\tilde{S}_n(x)\}dx$$

Subject to the initial conditions $S(0) = 300$, $I(0) = 200$, $R(0) = 100$. Using the initial conditions and the parameter values in the table and with the help of Maple 18, we obtain the iterated values for each compartment: :

$$S(t) = \sum_{n=0}^{k} S(k)t^k = 3000 + 9.9982944001E5t - 2.849606446E5t^2 + 21242.56063t^3$$
$$-3.024139142E7t^4 + 6.000217282E6t^5 - 5.542837547E9t^6 + \ldots$$

$$I(t) = \sum_{n=0}^{k} I(k)t^k = 200 + 57.4000t + 1.099894753E5t^2 + 10668.28948t^3$$
$$+3.023938248E7t^4 - 4.143376832E6t^5 + 5.542515808E9t^6 + \ldots$$

$$R(t) = \sum_{n=0}^{k} R(k)t^k = 100 + 98.5600t + 1.649709422E5t^2 - 32320.80299t^3$$
$$+1976.338874t^4 - 1.935462713E6t^5 + 3.309772042E5t^6 + \ldots$$

The VIM is demonstrated against the Maple 18 fourth order Runge-Kutta procedure to investigate the degree of accuracy of the method (i.e. VIM). Figure 1, Figure 2, and Figure 3 show the combined plots of the solutions of $S(t)$, $I(t)$ and $R(t)$ by the VI M and RK4.

RESULTS AND DISCUSSION

Numerical simulation which illustrates the analytical results for the model is demonstrated. This is achieved by using some set of values given in the Table 1 whose sources are mainly from the literature as well as assumptions. The VIM is demonstrated against maple in-built fourth order Runge-Kutta procedure for the solution of the model. Figure 1 to Figure 3 show the combined plots of the solutions of $S(t)$, and $R(t)$ by VIM and RK4.

Table 1: Parameters Values for the Model.

Description	Parameter	Initial Value	Source
Rate of loss of immunity after recovery	σ	0.44	Assumed
Rate of recovery from infection	γ	0.01	Mushayaba (2011)
Disease induced death rate	δ	0.013	Adetunde (2008)
Natural death rate	μ	0.02	Assumed
Contact rate	α	0.0011	Assumed
Vaccination rate	ρ	0.33	Lauria et al (2009)
Recruitment rate	β	10^6	Assumed

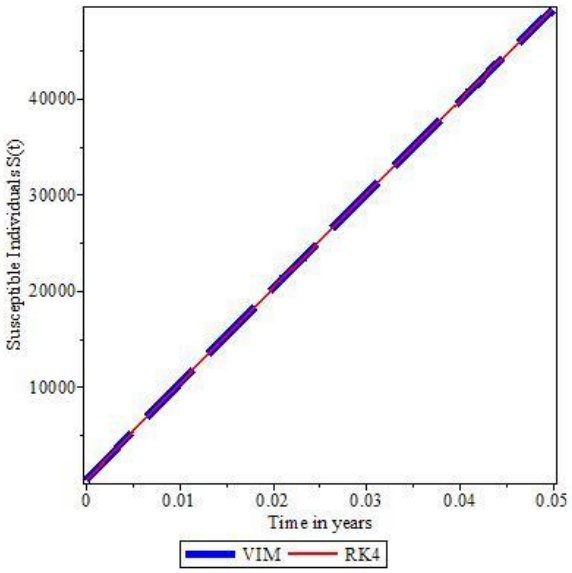

Figure 1: Solution of Susceptible Population by VIM and RK4.

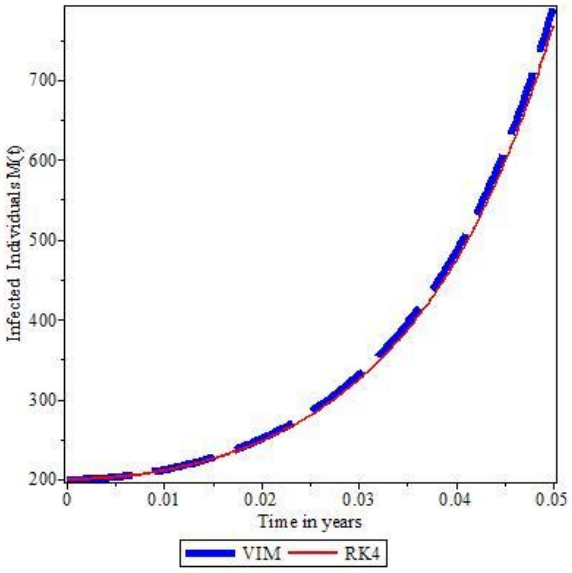

Figure 2: Solution of Infected Population by VIM and RK4.

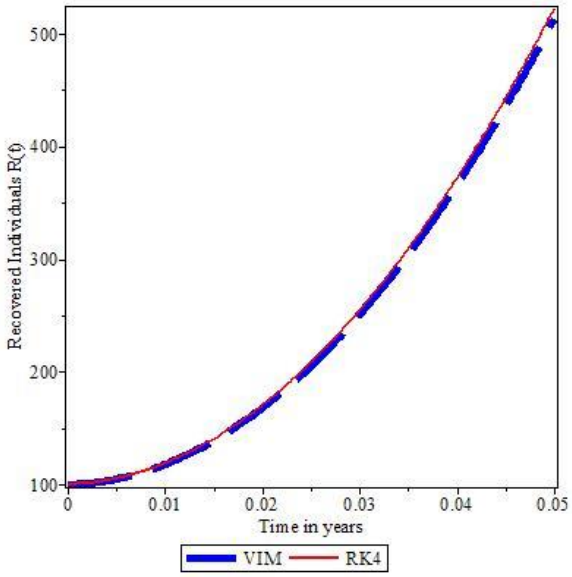

Figure 3: Solution of Recovered Population by VIM and RK4.

Figures 1, 2, and 3 show the outcome of the comparison between the solutions of the proposed model by using the VIM and RK4. The figures demonstrate the existence of positive correlation between the solutions of the two methods as the two curves follow the same pattern and behavior in each case. The superb convergence of the solutions of the VIM with that of RK4 indicates that the VIM obtained the reliable and accurate results for the model.

CONCLUSION

A deterministic model formulated by (Adebisi et al., 2019) is solved by VIM to analyze the transmission dynamics of infectious disease. Taking the initial conditions from the literature as well as assumption, the VIM is applied straight away to obtain the solutions of the model without linearization or perturbation. The reliability and accuracy of the VIM is examined when the results obtained by it are compared with the results obtained by using the Runge-Kutta method; the outcome of which is displayed graphically. We also obtained similar result when of (Adebisi et al, 2019) .Going by the outcome of our simulations, we conclude that the VIM is a powerful technique which can be used to obtain the approximate accurate series solutions for the epidemic models designed in terms of ordinary differential equations.

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