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An exact global solution for the classical *SIRS* epidemic model

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ABSTRACT

In this paper we propose an analytical approach to obtain an exact global solution for the classical *SIRS* epidemic mathematical model. The approach is based on modal expansion infinite series. These mode series are shown to provide a reliable and accurate analytical solution for the classical *SIRS* epidemic model. It is shown that for real initial conditions the modal expansion series present a convergent behaviour. These proposed modal expansion series do not rely on the well-known orthogonality relation. The validity and reliability of the proposed analytical approach is tested by its application in the *SIRS* epidemic model with various parameter values.

1. Introduction

Mathematical models have become important tools in studying the spread and control of infectious diseases. The most cited work referred to as the pioneering research in mathematical epidemiology is the one developed in 1926 by Kermack and McKendrick. In their work, they obtained the epidemic threshold result that the density of susceptibles must exceed a critical value in order for an epidemic outbreak to occur [1,2]. However, other historical antecedents include the smallpox model formulated and solved by Daniel Bernoulli in 1760 in order to evaluate the effectiveness of variolation of healthy people with the smallpox virus, and the discrete time model proposed in 1906 by Hamer formulated in his attempt to understand the recurrence of measles epidemics, which may have been the first model to assume that the incidence (number of new cases per unit time) depends on the product of the densities of the susceptibles and infectives [2]. In addition, Ross developed differential equation models for malaria as a host–vector disease in 1911, and he won the second Nobel prize in medicine. For more details readers can see the first edition (1957) of Bailey's book, which is an important book that helped the development of mathematical epidemiology, and the review made by Hethcote in 2000 [2].

The most recent epidemiological models have involved aspects such as passive immunity, stages of infection, vertical transmission, disease vectors, macroparasitic loads, age structure, social and sexual mixing groups, spatial spread, vaccination, quarantine, and chemotherapy [2].

Mathematical models and computer simulations are useful experimental tools for building and testing theories, assessing quantitative conjectures, determining sensitivities to changes in parameter values, and estimating key parameters from data. Understanding the transmission characteristics of infectious diseases in communities, regions, and countries can lead to better approaches to decrease the transmission of these diseases [2]. Mathematical models are used in planning, evaluating, and optimizing various detection, prevention, therapy, and control programs. Epidemiology modeling can contribute to the design and analysis of epidemiological surveys, suggest crucial data that should be collected, identify trends, make general forecasts, and estimate the uncertainty in forecasts [2]. Many of these models are based upon systems of ordinary

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differential equations (ODEs). In these models commonly the variables represent subpopulations of susceptibles (S), infected (I), recovered (R), latent (E), transmitted disease vectors, and so forth. Thus, the ODE system describes the dynamics of the different classes of subpopulations in the model [3–7]. In this way, acronyms for epidemiology models are often based on the flow patterns between the compartments such as *SI*, *SIS*, *SIRS*, *SEIR*, and *SEIRS*. All these and most of the current models are extensions of the *SIR* model elaborated by Kermack and McKendrick in 1927 [2]. Closed-form expressions for these variables in the mathematical models only are known for the *SI* epidemic model and closely related variants [1]. On the other hand, several numerical methods has been applied to solve epidemic models [8–12].

One of the major difficulties of the analytical epidemic approach has been the rapid growth of the mathematical complexity of the models used to describe the various aspects of phenomena in sufficient detail and the difficulty in solving them in an analytical form. Hence, their practical use in specific cases is limited. The main difficulties arise from the presence of the nonlinear term *SI* which comes from the law of mass of action [13]. However, is our aim in this paper to develop an exact solution for the classical *SIRS* epidemic model that may be extended to more complex systems. The *SIRS* model and related variants have been used to model several topics such as respiratory syncytial virus, hepatitis, influenza, and others [2,14–18].

Our procedure to solve the problem is as follows: (i) We rewrite the system of equations for the functions $I(t)$, $S(t)$ and $R(t)$ corresponding respectively to the infected, susceptible and recovered individuals as a single integro–differential equation for $I(t)$. (ii) Exploiting the fact that all nonlinear terms in this equation are quadratic in $I(t)$ and $I(t)$ times the integral of $I(t)$ over time we propose a modal expansion series:

$$I(t) = \sum_{k=0}^{\infty} \mathcal{A}_k \mathrm{e}^{-k\omega t}, \quad \omega > 0, \tag{1}$$

where the frequencies and the coefficients $\mathcal{A}_{ks}$, $k = 1, 2, \ldots$, are determined by a recurrence relation in terms of $\mathcal{A}_0$ and the parameters of the model. The series in Eq. (1) can be seen as a particular case of general transseries which runs by Borel summation conditions [19].

This paper is organized as follows. In Section 2 the *SIRS* epidemiological mathematical model and its underlying assumptions are presented. We also show how to reduce the system of three differential equations to a single nonlinear integro–differential equation for $I(t)$. In Section 3, an exact global solution for the classical *SIRS* epidemic model is obtained. In order to support our theoretical results, numerical comparisons are shown in Section 4. Finally, in Section 5, a discussion and conclusions are presented.

2. *SIRS* epidemic mathematical model

In this section the *SIRS* epidemic mathematical model is presented. The *SIRS* model is based on a system of first-order ordinary differential equations and has been used in the modeling of several infectious diseases where the parameters need to be estimated by medical data [2]. In this model the variables represent subpopulations of susceptibles (S), infected (I) and recovered (R). Thus, the model describes the dynamics of the different classes [3,4]. The classical *SIRS* model (Susceptibles, Infected, Recovered and Susceptibles) is given analytically by the following form:

$$\begin{aligned}
&\dot{S}(t) = \mu - \mu S(t) - \beta S(t) I(t) + \gamma R(t), \quad S(0) = S_0 > 0\\
&\dot{I}(t) = \beta S(t) I(t) - \nu I(t) - \mu I(t), \quad I(0) = I_0 > 0\\
&\dot{R}(t) = \nu I(t) - \gamma R(t) - \mu R(t), \quad R(0) = R_0 > 0.
\end{aligned} \tag{2}$$

Some hypotheses assumed in this model are:

(1) The population is divided in three disjoint classes: Susceptibles $S(t)$, who are all individuals that do not have the virus; Infected $I(t)$, who are all individuals with the virus who are able to transmit the illness; and Recovered $R(t)$, who are all the healthy individuals with a temporary immunity.
(2) The birth rate $\mu > 0$ and death rate are assumed equal. This means that $\dot{S}(t) + \dot{I}(t) + \dot{R}(t) = 0$. Thus the total population is constant, and without loss of generality $S(t) + I(t) + R(t) = 1$.
(3) The coefficient β between classes $S(t)$ and $I(t)$ is called the transmission rate.
(4) The per capita rate of leaving the infective class $I(t)$ is called ν and the per capita rate of leaving the recovered class $R(t)$ is γ.
(5) Initial conditions are $S(t = 0) = S_0$, $I(t = 0) = I_0$ and $R(t = 0) = R_0$.

In order to present model (2) in a more compact form we define

$$\alpha = \nu + \mu \quad \text{and} \quad \delta = \gamma + \mu.$$

Thus model (2) can be rewritten as follows:

$$\begin{aligned}
&\dot{S}(t) = \mu - \mu S(t) - \beta S(t) I(t) + \gamma R(t), \quad S(0) = S_0 > 0\\
&\dot{I}(t) = \beta S(t) I(t) - \alpha I(t), \quad I(0) = I_0 > 0\\
&\dot{R}(t) = \nu I(t) - \delta R(t), \quad R(0) = R_0 > 0.
\end{aligned} \tag{3}$$