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A time-delayed epidemic model for Ebola disease transmission*



Isam Al-Darabsah, Yuan Yuan*

Department of Mathematics and Statistics, Memorial University of Newfoundland, St. John's, NL A1C 5S7, Canada

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ABSTRACT

In this paper, we propose a delayed mathematical model for the transmission of Ebola in humans. We consider the transmission of infection between the living humans and from infectious corpses to the living individuals in which the latent period of Ebola is incorporated. We identify the basic reproduction number R_0 for the model, prove that the disease-free equilibrium is always globally asymptotically stable when $R_0 < 1$, the disease is persistence and a unique endemic equilibrium exists when $R_0 > 1$. We show that the endemic steady state is locally asymptotically stable under certain condition and globally asymptotically stable in a special case of the model. Numerical simulations are provided to demonstrate and complement the theoretical results.

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1. Introduction

Ebola virus disease, or simply Ebola, is a disease of humans and other non-human primates (gorillas, chimpanzees and duikers) caused by Ebola virus (EBOV) which is belong to the family Filoviridae. The virus is originate in fruit bats and jump to humans through an intermediate animal, such as chimpanzees [17,20]. Ebola first appeared in 1976 in two outbreaks, one in Sudan, and the other in Democratic Republic of Congo. Since then it has resurfaced in Africa several times, for example, in 1994 in Ivory Coasty and Gabon; and in 2000 in Uganda. The outbreak in West Africa (Guinea, Liberia and Sierra Leone) in March 2014, is the largest and most complex Ebola outbreak. During this outbreak, Ebola has infected around 27,678 people, roughly 11,267 of whom have died [3]. According to Centers for Disease Control and Prevention (CDC) and World Health Organization (WHO), Ebola can be spread through human-to-human transmission via direct contact with the blood, secretions, organs or other bodily fluids (including but not limited to urine, saliva, sweat, feces, vomit, breast milk, and semen) of infected people, and with surfaces and materials (e.g., bedding and clothing) contaminated with these fluids [1,4].

The mathematical study of infectious disease dynamics is an important aspect of investigating the spread rules of infections. Compartmental models are the most frequently used to describe the epidemiology of infectious diseases, where the total population is usually divided into a finite number of discrete categories, for example, the classical SEIR model with four stages of susceptible, infected but not yet infectious, infectious and recovered is discussed in [7]. Multiple epidemiological models have been proposed to predict the spread of Ebola in West Africa [5,16,26,27]. In [5], the author used SEIR model to estimate the basic reproduction numbers of Ebola during the 2014 outbreak in West Africa. The maximum estimation of the basic reproduction number was 1.51 for Guinea, 2.53 for Sierra Leone and 1.59 for Liberia. In [16], the authors di-

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^{*} Corresponding author. Tel.: +1 709 8648795; fax: +1 709 8643010. E-mail address: yyuan.cc@gmail.com, yyuan@mun.ca (Y. Yuan).

vided the population into six compartments: susceptible, exposed, infectious, hospitalized, funeral and removed. They found that increased contact tracing, improved infection control, or a combination of the two can have a substantial impact on the number of Ebola cases. In [26], a model consisting of susceptible, exposed, infectious, contaminated deceased, isolated infectious and removed categories was proposed to indicate that isolating the infectious cases with average time less than three days between the appearance of symptoms and isolation; and the efficiently monitoring of the contact traced incubating infected cases are the most important elements for containment of Ebola within a short time. A SEIRD model (susceptible, exposed, infectious, recovered and dead but still infectious) was studied in [27]. They found robustly that inferences that do not account for post-death transmission tend to underestimate the basic reproductive number, in other words, large amounts of post-death transmission imply larger reproductive numbers.

In the real world, for many diseases such as Ebola, when adequate contact with an infectious happen, a susceptible individual becomes infected but is not yet infectious. This individual remains in the exposed class for a certain latent period before becoming infectious. Such period in disease transmission can be modeled by a delay differential equation. For example, in [6] an SEIRS epidemic model with a constant latent and immune periods is presented. In [24], the authors proposed a general mathematical model for a disease with a latent period and relapse. In [28], a disease transmission model of SEIRS type with distributed delays in latent and temporary immune periods is discussed. The authors studied the threshold property of the basic reproduction number R_0 and the dynamical properties of the disease-free/endemic equilibrium points with general and particular probability distributions in both periods.

In West Africa it is common to contact with the bodies and fluids of persons who have died, where family and community members often touch and wash the body of the deceased in preparation for funerals [14,15]. Since Ebola virus can survive for several days at room temperature in body fluids [2], one of the main infection pathways is through preparation of corpses for burial. In this paper, we propose a model that incorporates both the transmission of infection between the living humans and from the infected corpses to the living individuals with a constant latent period. From the viewpoint of dynamics, we discuss the existence and stability of equilibrium points and give numerical simulations to show the theoretical results and explore the dynamical behavior of the disease under varied environments. The main difference of this work, from the literature e.g., [5,16,26,27], is that those models are all given by ordinary differential equations which neglected the effect of latent period. However, the latent period has a profound effect on the generation time, and hence epidemic growth (see e.g., [13]). Moreover, although there are some contain analysis on the basic reproduction number R_0 previously, which offer some interesting insights into Ebola transmission in humans and show some numeric results, in this work, in addition to the mathematical derivation of R_0 , we determine the local and global dynamics of the model analytically with respect to the basic reproduction number R_0 .

The rest of the manuscript is organized as follows. In Section 2, we present the model and discuss its well-posedness by verifying the non-negativity and boundedness of the solutions with reasonable initial data. In Section 3, we calculate the basic reproduction number R_0 ; discuss the global stability of the disease-free equilibrium when $R_0 < 1$; explore the existence of a unique endemic equilibrium when $R_0 > 1$ and show the local stability under certain condition. In Section 4, we prove the persistence of infection when $R_0 > 1$ and study the global stability of the endemic equilibrium in a special case of the model by considering a limiting system of the model and then using the Lyapunov functional and LaSalle invariance principle. In Section 5, numerical simulations are given to demonstrate the theoretical results and explore the disease transmission with the variation of seasonality. Finally, conclusion remarks are drawn in Section 6.

2. Mathematical model and the well-posedness property

Motivated by the model in [27], we consider the transmission of infection between the living humans and from the infected corpses to the living individuals in which the latent period of Ebola is incorporated.

We consider the size of the population N(t) is divided into susceptible, exposed (infected but not yet infectious), infectious, recovered individuals and infected corpses who are nonetheless infectious, with class sizes denoted by S(t), E(t), I(t), R(t) and D(t), respectively. Let τ be the latent period, c the probability of transmission of infection from an infectious human to a susceptible individual when a contact occurs, and d the probability of transmission of infection from an infectious corpse to a susceptible individual. Therefore, the total number of new infected individuals at time t is

$$c\frac{S(t)I(t)}{N(t)}+d\frac{S(t)D(t)}{N(t)}.$$

If μ is the natural death rate, then the probability that an individual survives in the latent period $[t-\tau,t]$ is $e^{-\mu\tau}$. Hence, the total number of individuals surviving in the latent period τ and becoming infectious at time t is

$$c\frac{S(t-\tau)I(t-\tau)}{N(t-\tau)}e^{-\mu\tau}+d\frac{S(t-\tau)D(t-\tau)}{N(t-\tau)}e^{-\mu\tau}.$$

Thus, we have the following delayed model:

$$\begin{split} \frac{dS(t)}{dt} &= \Lambda - c \frac{S(t)I(t)}{N(t)} - d \frac{S(t)D(t)}{N(t)} - \mu S(t), \\ \frac{dE(t)}{dt} &= c \frac{S(t)I(t)}{N(t)} + d \frac{S(t)D(t)}{N(t)} - c \frac{S(t-\tau)I(t-\tau)}{N(t-\tau)} e^{-\mu\tau} - d \frac{S(t-\tau)D(t-\tau)}{N(t-\tau)} e^{-\mu\tau} - \mu E(t), \end{split}$$

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