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Review

Plague disease model with weather seasonality

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ABSTRACT

The plague disease model that include the effect of seasonal weather variation in its transmission is investigated in this paper. The disease is caused by an extremely virulent bacteria *Yersinia pestis* named after a French bacteriologist Alexandre Yersin. The analysis shows that, when the periodic reproduction number (R_T) is greater than one there exist a globally asymptotically stable disease free equilibrium solution (DFS). Using fundamental existence-uniqueness theorem we were able to prove the existence of positive periodic solutions. The analysis further shows that when $R_T > 1$ then there is at least one positive periodic solution. We additionally establish the conditions for global stability of periodic solutions of the model and finally using numerical simulation we depict the behavioral dynamics of plague disease and justify the theoretical solutions.

1. Introduction

Plague disease is a serious bacterial infection caused by the gram negative bacterium called *Yersinia pestis*, it is frequently lethal and potentially epidemic re-imaging disease [36]. These bacteria are found on animals throughout the world. They mainly infect wild rodents (most notably rats) and other rodents who are the prime reservoir for the bacteria. The disease remains to be notorious and a threat to human societies throughout history, due to the extraordinary scale of death and damage it brought over the history [36].

1.1. Historical background of plague disease

At its early days the root of plague was unknown so it led to a great effects on the development of modern civilization. It caused devastating mortality of people and animals across the globe and thus contributed to a massive panic in cities and countries where it appeared. There have been three great world pandemics of plague disease; The first is the Great Plague of Justinian that occurred around AD 532 in Egypt and spread to other part of the world through the Middle East and the Mediterranean basin. Between 50% and 60% of the total population in North Africa, Europe, and central and southern Asia were lost [28]

The second plague disease pandemic, also known as the Black Death or Great Pestilence, occurred in 1334 in different cities of China and India [41]. It killed nearly 20 to 30 million people in Europe which was equivalent to more than one third of the European population at that time [31]. The third and last great plague pandemic that was labeled as a major bubonic plague pandemic occurred in Canton and Hong Kong in 1850s. It spread to different continents, and killed nearly 12 million people in India and China [34].

2. Transmission and infection

Plague disease is mostly transmitted by the bite of an infected flea. Yersinia pestis multiply themselves in the flea's stomach making millions of copies, that with time block the flea's digestive system. This gradually causes the hungry and ravenous biter flea that will attempt to feed on any warm-blooded animal it can reach. When people and animals visit places where there are cases of plague disease are at risk of being infected from flea bites. When Yersinia pestis are in lungs plague disease may be transmitted via aerosol droplets. Other ways may be through eating infected animals, direct contact and contacting the contaminated undercooked food or materials [30].

Plague disease mainly occurs in three forms which are; bubonic, septicemic and pneumonic plague [20]. The adequate contact between the flea infested with pathogens and the susceptible individual (through bite) results in primary bubonic plague or septicemic plague. In very rare cases one may get bubonic plague infection through contact with contaminated fluid or tissue. Symptoms of bubonic plague generally appear within two to seven days and includes: Fever and chills, headache, muscle pain, general weakness and seizures. One may also experience painful swollen lymph glands called buboes, which appear in the groin, armpits, neck, or site of the insect bite or scratch. The buboes are what give bubonic plague its name [6].

Septicemic plague occurs when the bacteria infect blood streams. One may be exposed to septicemic plague through physical contact

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(including sexual contact). An example is when humans being without using proper precautions, handles tissue or body fluids of an animal that is infected by plague during skinning infected animal. Animals like cats may be infected by eating infected rats. Symptoms usually start within two to seven days after exposure. Septicemic plague can lead to death before symptoms even appear. Symptoms includes: abdominal pain, diarrhea, nausea and vomiting, fever and chills, weakness, bleeding (blood may not be able to clot) and shock [29].

The other and most severe form of plague is pneumonic Plague which is transmitted through infectious droplets. When a person with plague pneumonia cough, droplets containing the plague bacteria into air, and if these bacteria-containing droplets are breathed in by another person they can cause pneumonic plague infection. Pneumonic plague symptoms may appear as quickly as one day after exposure to the bacteria and include: difficulty with breathing, chest pain, cough, fever headache, overall weakness and bloody sputum (saliva and mucus or pus from the lungs) [7].

Without control about 50% of individual with bubonic and septicemic plague and almost 100% of people with pneumonic plague die. When there is control mortality is reduced to 50 percent for all three forms of plague disease [2].

Plague disease is greatly affected by seasonal weather variation as it influences almost all components involved in plague disease transmission system [3]. In most cases seasonal fluctuation of weather condition is regarded as the primary factors that cause the recurrent of plague disease circle and is probably the factor that enlighten the reasons for variability of plague disease from small to large scales [26,27]. Weather variation dictates the infection rate of the plague disease, as it affect natural demographic behaviour of the populations involved in its dynamics [11,33].

The environmental condition varies due to seasonal fluctuation of weather parameters which naturally are subjected to fluctuation in time. Recently the issue of effect of seasonal weather variation in the dynamics of infectious disease has become the key point to many epidemiological researchers due to the fact that many infectious disease are affected by different element of weather conditions in the environment [1]. The variation in disease dynamics caused by season weather variation may be due to its ability to alter infection rate of the disease, birth and death rates and immigration rates [22,23]. Now if the desired disease dynamics is affected by these fluctuations it changes from being an autonomous disease model system to non-autonomous which is a bit tough in analyzing [5].

To better understand the dynamics of infectious diseases we use mathematical models, which are the powerful tools for studying the wide range of phenomena in real world [13]. In most cases mathematical epidemiology results reflect the reality and may be useful in predicting the dynamics of the disease in the particular range of time [16]. However in most epidemic models, the model parameters such as transmission rates, migration rates and birth and death rates are mostly considered to be constants regardless of the seasonal behavior of most of the infectious diseases due to weather conditions fluctuations [1]. Therefore for more realistic disease dynamics and result, we must take into account the seasonal variation of the epidemic due to weather fluctuation. In this paper we study the dynamics of non-autonomous model system of plague disease with periodic transmission rate. We therefore assume the seasonal transmission to be sinusoidal, in a form as given in (2.1).

$$\lambda(t) = \lambda_0(1 + \sigma\cos(2\pi t)) \tag{2.1}$$

where σ is the amplitude of seasonal variation in transmission also known as strength of seasonal forcing and λ_0 is the average transmission rate.

We discuss the plague disease system dynamics in terms of global stability of the disease-free equilibrium, the existence of positive periodic solutions and the stability of positive periodic solution. We further use numerical simulations to illustrate the theoretical results.

3. Model formulation

The model has four settings: Human population, rodent population, flea population and pathogens in the environment (A). The total human population is divided into six compartments: susceptible human (S_H), exposed human (E_H), bubonic plague infectives (I_{HB}), septicemic plague infectives (I_{HS}), pneumonic plague infective (I_{HP}), Recovered human (R_H) and $N_1 = S_H + E_H + I_{HB} + I_{HS} + I_{HP} + R_H$. Total rodent population is divided into five compartments: susceptible rodent (S_R); exposed rodent (E_R), bubonic plague infectives (I_{RB}), septicemic plague infectives (I_{RS}), pneumonic plague infective (I_{RP}) and $N_3 = S_R + E_R + I_{RB} + I_{RS} + I_{RP}$. The total flea population is divided into two compartment: susceptible flea (S_F), infectious flea (I_F) and $N_2 = S_F + I_F$.

Human being gets plague (Bubonic, pneumonic and septicemic) infection after they adequately interact with various infectious agents as follows: infectious human and rodent with pneumonic plague through airborne transmission at the periodic rates $\Gamma_{hph}(t)$ and $\Gamma_{rph}(t)$ respectively; infectious human and rodent with septicemic plague through direct physical contact including sexual contact at the periodic rates $\Gamma_{hsh}(t)$ and $\Gamma_{rsh}(t)$ respectively; infectious flea at a periodic rate $\Gamma_{fh}(t)$ and pathogens in the environment at the periodic rate $\omega_1(t)$ which makes the force of infection to human beings as given in (3.1)

$$G_1(t) = \frac{\Gamma_{hph}(t)I_{HP} + \Gamma_{hsh}(t)I_{HS}}{N_1} + \Gamma_{fh}(t)\frac{I_F}{N_2} + \frac{\Gamma_{rph}(t)I_{RP} + \Gamma_{rsh}(t)I_{RS}}{N_3} + \omega_1(t)A \tag{3.1}$$

Rodent also gets plague infection when they adequately contact with various infectious agents as follows: infectious rodent and human with pneumonic plague through airborne transmission at the periodic rates $\Gamma_{rpr}(t)$ and $\Gamma_{hpr}(t)$ respectively; infectious rodent and human with septicemic plague through direct physical contact including sexual contact at the periodic rates $\Gamma_{rsr}(t)$ and $\Gamma_{hsr}(t)$ respectively; infectious flea at aperiodic rate $\Gamma_{fr}(t)$ and pathogens in the environment at the periodic rate $\omega_2(t)$ which makes the force of infection to rodents as given in (3.2)

$$G_2(t) = \frac{\Gamma_{hpr}(t)I_{HP} + \Gamma_{hsr}(t)I_{HS}}{N_1} + \Gamma_{fr}(t)\frac{I_F}{N_2} + \frac{\Gamma_{rpr}(t)I_{RP} + \Gamma_{rsr}(t)I_{RS}}{N_3} + \omega_2(t)A \tag{3.2}$$

Susceptible flea may get infection when they bite human beings or rodents with bubonic or septicemic plague at the periodic rated rates $\Gamma_{hbf}(t)$ or $\Gamma_{hsf}(t)$ or $\Gamma_{rbf}(t)$ or $\Gamma_{rsf}(t)$ respectively which makes the force of infection in fleas as given in (3.3). Human being and rodents with pneumonic plague infest the environment with pathogens causing plague disease at the periodic rates $\eta_1(t)$ and $\eta_2(t)$ respectively.

$$G_3(t) = \frac{\Gamma_{hbf}(t)I_{HB} + \Gamma_{hsf}(t)I_{HS}}{N_1} + \frac{\Gamma_{rbf}(t)I_{RB} + \Gamma_{rsf}(t)I_{RS}}{N_3} \tag{3.3}$$

3.1. Variables and parameters used in the model

This section presents variables and parameters, their description and their values as used in the model. We obtain the parameters from the literature that relate to this study, the present information on plague disease and through estimation.

Fig. 1 shows the dynamics of complex interaction between human beings, rodents, fleas and pathogens in the environment that lead to plague disease transmission from one infectious individual to the susceptible individual.

Using the description of infection, variables and parameters stated in Tables 1 and 2 and the compartmental diagram in Fig. 1 we derive the system of differential equations that describe plague disease dynamics in human beings, rodents, fleas and pathogens in the environment as given in (3.4)–(3.7).