A Simple Deterministic Model for the Spread of Leptospirosis in Thailand

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Abstract—In this work, we consider a deterministic model for the transmission of leptospirosis which is currently spreading in the Thai population. The SIR model which incorporates the features of this disease is applied to the epidemiological data in Thailand. It is seen that the numerical solutions of the SIR equations are in good agreement with real empirical data. Further improvements are discussed.

Keywords—Leptospirosis, SIR Model, Deterministic model, Thailand.

I. INTRODUCTION

EPTOSPIROSIS, a worldwide zoonotic disease, is an ⊿acute febrile illness caused by pathogenic spirochete of the genus Leptospira [1,2]. The disease is considered to be a major public health problem worldwide. The illness resulting from the disease ranges from a mild flu-like illness to a severe or fatal disease involving renal and/or liver failures and hemorrhages (referred as Weil's syndrome) [3]. Most outbreaks tend to be seasonal in nature and are often linked to environment factors, to animals and to agricultural and occupational cycles like cultivating rice in marshy land. Mammals such as rats and cattle are commonly involved in exposure to contaminated tissues or urine [1,2,4]. Outbreaks of leptospirosis occur mainly after flooding, leading to its become an occupational hazard for sanitary and agricultural workers as well as being recreational hazard for humans [5]. Some pathogenic leptospires have been found to be associated with domesticated animals. For example, serovar canicola (L. canicola) has adapted itself to canines. This has led it to

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become common in many human communities. Epidemiologic investigation of leptospirosis is often hampered by the difficulty of making a definitive microbiologic diagnosis. Isolation of leptospira from clinical samples provides a definitive diagnosis. However, the value of this technique is limited because prolonged incubation periods are needed before the bacteria's are detectable.

In Thailand, leptospirosis is becoming a major concern of the public health officials and control strategies are being developed. It is a notifiable disease, and reported cases are investigated by the Division of Epidemiology, Ministry of Public Health. The number of leptospirosis cases reported in 1999 was 6,080 (incidence 9.89/100,000/year) [6]. The number reported in 2000 was 14,286 (incidence 23.13/100,000/year) [7] and in 2001, the number was 10,217 (incidence 16.45/100,000/year) [8]. It occurs mainly in the rainy season, with an increase in cases beginning in August, reaching a peak in October, and beginning to fall in November.

In this paper, we model the spread of the leptospirosis using the susceptible-infective-removed (SIR) model that has been used to describe the transmission dynamics of many infectious diseases. Modification of the SIR model must be made it applicable to a particular disease [9].

II. MODEL

The SIR model was initially proposed by Kermack and Mckendrick [10]. Common to most SIR models is division of the players into the human population and the vector population, which in the case of leptosprosis are rats. The human population is then divided into three groups; S_H^* – susceptible human, I_H^* – infected human, and R_H^* – removed human. The vector population is divide into two subgroups, S_A^* – susceptible vector, and I_A^* – infected vector. In many of the SIR models, the following assumptions are made:

a. The total size N of human population is constant.

b. The natural death constant rate λ_H is taken to be the same for all population subgroups.

c. The individuals are unaffected by age or disease status so that the vital statistics of all individuals are the same. Thus the life expectancy is the same for everyone and is $1/\lambda_H$.

d. Deaths are balanced by births (birth rate being μ_H). This leads to condition 'a.'

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e. All newborn are considered not to be immunized and so become vulnerable instantly.

f. There is spatially homogeneous mixing among vector and human populations.

In addition to these common assumptions, there are a few assumptions particular to the spread of leptosprosis and a few other diseases:

i.Only infected vectors can be infected human. This means that an infected human can

not infected another human.

ii. Infected humans can not infect the susceptible vectors.

iii.Once infected, a susceptible vector (S_A^*) becomes

instantly infectious vector (I_A^*) with no incubation time needed for the infectious agents (leptospira) to develop.

iv. The infected human can be cured by the antibiotic medicines and they become immune at a rate (r_1) .

v. Immune individuals become susceptible (S_H^*) again at a constant rate r_2 .

vi.The rate of transmission of leptosprosis from an infected vector to a susceptiable human varys with the amount of rain fall according to some Gaussian distribution dependence.

The diagram representing the dynamics of transmission of Leptospirosis is shown in Fig. 1. Based on the common assumptions given by (a.) to (f.) and the special assumptions given by (i.) to (vi.), we have the following equations;

$$\frac{dS_{H}}{dt} = \mu_{H}N_{H} - \lambda_{H}S_{H}^{*} - \gamma_{H}^{*}I_{A}^{*}S_{H}^{*} + r_{2}R_{H}^{*}$$
(1)

$$\frac{dI_{H}}{dt} = \gamma_{H}^{*} I_{A}^{*} S_{H}^{*} - \lambda_{H} I_{H}^{*} - r_{1} I_{H}^{*}$$
(2)

$$\frac{dR_{H}^{*}}{dt} = r_{1} I_{H}^{*} - \lambda_{H} R_{H}^{*} - r_{2} R_{H}^{*}$$
(3)

Here $S_H^*(t) + I_H^*(t) + R_H^*(t) \equiv N_H^*(t) = N_H^*(t_0)$ denotes the number of the total population, which is kept constant. The positive constant γ_H is the average number of contacts per infective individual per month.

For the vector populations, we have

$$\frac{dS_A^*}{dt} = \mu_A S_A^* - \lambda_A S_A^* - \gamma_A^* S_A^* I_A^*$$
⁽⁴⁾

$$\frac{dI_A^*}{dt} = \mu_A I_A^* - \lambda_A I_A^* + \gamma_A^* S_A^* I_A^*$$
(5)

 μ_A and λ_A are the birth and death rate respectively. We have assumed that all newborns have the same status as their parents.

We normalize the variables in (1)-(5), based on the assumption that the number of population is constant, by

letting

$$S_{H} = S_{H}^{*} / N_{H}, I_{H} = I_{H}^{*} / N_{H}, R_{H} = R_{H}^{*} / N_{H},$$

$$S_{A} = S_{A}^{*} / N_{A}, I_{A} = I_{A}^{*} / N_{A}, \gamma_{H} = \gamma_{H}^{*} N_{A}, \text{ and}$$

$$\gamma_{A} = \gamma_{A}^{*} N_{A} \text{ then we obtain}$$

$$\frac{dS_H}{dt} = \mu_H - \lambda_H S_H - \gamma_H I_A S_H + r_2 R_H \tag{6}$$

$$\frac{dI_{H}}{dt} = \gamma_{H}I_{A}S_{H} - \lambda_{H}I_{H} - r_{1}I_{H}$$
⁽⁷⁾

$$\frac{dR_H}{dt} = r_1 I_H - \lambda_H R_H - r_2 R_H \tag{8}$$

$$\frac{dS_A}{dt} = \mu_A S_A - \lambda_A S_A - \gamma_A S_A I_A \tag{9}$$

$$\frac{dI_A}{dt} = \mu_A I_A - \lambda_A I_A + \gamma_A S_A I_A \tag{10}$$

Because of constraints $S_H + I_H + R_H = 1$ and $S_A + I_A = 1$, the above system of equations can be simplified into the following three equations.

$$\frac{dI_{H}}{dt} = \gamma_{H} I_{A} (1 - I_{H} - R_{H}) - I_{H} (\mu_{H} + r_{1})$$
(11)

$$\frac{dR_{H}}{dt} = r_{1} I_{H} - R_{H} (\mu_{H} + r_{2})$$
(12)

$$\frac{dI_A}{dt} = \gamma_A I_A (1 - I_A) \tag{13}$$

The steady-state solutions are determined by setting

$$dI_H / dt = 0, \ dR_H / dt = 0$$

 $dI_A / dt = 0$; the nontrivial solution is $\overline{E}_2 = (I'_H, R'_H, I'_A)$, where

$$I'_{H} = \frac{\gamma_{H}}{\gamma_{H} + \frac{\gamma_{H}r_{1}}{\mu_{H} + r_{2}} + (\mu_{H} + r_{1})}$$
$$R'_{H} = \left[\frac{\gamma_{H}}{\gamma_{H} + \frac{\gamma_{H}r_{1}}{\mu_{H} + r_{2}} + (\mu_{H} + r_{1})}\right]\frac{r_{1}}{\mu_{H} + r_{2}}$$

and

$$I'_{A} = 1$$

III. NUMERICAL RESULTS AND DISCUSSION

The model described by Eqs. (11)-(13) can be integrated numerically using a fourth-order Runge-Katta method. Time step of 2.2 (corresponding to 6 days in real time) is used. Since we know the numbers of infected human with leptospirosis disease in 2000 and 2001, reported by the Ministry of Public Health of Thailand, we should be able to reproduce data using simulation methods to find the values of some of the parameters appearing in the three equations. We pick the first point to be May in order to match the beginning of the raining season and other points to be one month until we reach the end of April. In term of the infected people, we normalize the number of the patients in every month by the total number of them in each year for representation on the graph. We do the same to the data for the amount of the rain as reported by Department of Meteorology in Fig.2. We fit the graphs using a Gaussian fitting to the 12 data points. The Gaussian fitting equation used is

$$y = y_0 + \frac{A}{w\sqrt{\pi/2}}e^{-\frac{2(x-x_c)^2}{w^2}}$$

In this study, we will see that behavior of the epidemic at the local levels is the same as that at the national level (Thailand as a whole). We have picked Phrae province, located in the north of Thailand, and Nakhon Ratchasima province, located in the north-east of Thailand, to study. These two provinces are isolated from each other and rats are hardly moving from Phrae to Nakhon Ratchasima so they are nothing in common. Moreover, both of them are in the list of the ten provinces having the highest rate of infection (See Fig.3.).

Using the special condition (vi), we obtain the changes in the transmission rate of the leptosprosis to humans for each month. A one month scaling was used since the Leptospira requires one month to develop. A brute force method was used to determine the initial values of parameters such as the number of infected human, the number or removed human, and the number of infected vectors. The values of certain parameters of the model such as the birth rate, the death rate, and the population size, were obtained from the demographic data of each area. The life expectancy of a human $(1/\lambda_{\rm H})$ is about 60 years old and so the mortality rate of the human ($\lambda_{\rm H}$) is $1/(365 \times 60)$ per day. The life span under natural conditions of the vectors (rats) is one and a half year. Therefore, the death rate of the vectors (λ_v) is 1/(1.5 x 365) per day. The recovery rate of an infectious human, or immunity (r_1) , is 1/15 per day. The rate of loss of immunity (r_2) is taken to be 1/360 per day. The transmission rate (γ_H or γ_A) depends on We have only considered the possible many factors. dependence on the amount of rain fall. We have assumed that the transmission rate of leptosprosis from an infected vector to a susceptible vector to be a constant.

Fig. 4 shows the temporal evolution of I(t) in 2000 and 2001 in the Phrae province and Nakhon Ratchasima. The star data represents the actural data and the closed circle lines represent the values obtained by solving equations (11)-(13). We then use the data of on the total number of infected people in Thailand during the years of study and the rain fall all over the country in the year, we will obtain the figure shown in Fig. 5.

IV. CONCLUSION

In this paper, we have seen that number of cases of

leptosprosis infections in two provinces, Phrae province and Nakhon Ratchasima province, and throughout Thailand obtained by solving a SIR model of the transmission of this diseases in 2000 and 2001 are in good agreement with the actural incidence rates if the transmission rate of leptosprosis from the vectors to the human is taken to be correlated to the rain fall. Why this happens is still a matter of conjecture. In forthcoming works we expect to generalize the model becoming more realistic model including the time delay of the

TABLE I Top ten rates per 100000 persons of leptospirosis disease in Thailand in 2000

Provinces In	nfected rate per 100000 persons
1. Buri Ramx	207.90
2.Nong Bua Lam Phu	138.17
3.Loei	135.94
4.Surin	84.72
5.Khon Kaen	75.80
6.Phrae	71.07
7.Nakhon	57.95
Ratchasima	
8.Chaiyaphum	51.23
9.Kalasin	44.51
10.Maha Sarakham	41.75
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Ratchasima	
10.Amnat Charoen	41.75

From: Annual epidemiological surveillance report 2001, Bureau of epidemiology department of disease control Ministry of Public Health disease diffusion that may provide some enlightenment.

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APPENDIX

Fig. 1 Flowchat of the dynamics of transmission of Leptospirosis.

Fig. 2 Gaussian fitting of the rain from May to April. The rainfall for 2000 is in a) with $Chi^2 = 0.00102$, $R^2 = 0.6353$, $y_0 = 0.04232 \pm 0.02033$, $x_c = 6.77107 \pm 1.08999$, $w = 5.51086 \pm 2.88476$, and $A = 0.61819 \pm 0.38179$. The rainfall for 2001 is in b) with $Chi^2 = 0.00124$, $R^2 = 0.63694$, $y_0 = 0.03538 \pm 0.02324$, $x_c = 7.30693 \pm 0.81597$, $w = 5.30834 \pm 2.40361$, and $A = 0.67135 \pm 0.3721$.

Fig. 3 Thailand map showing Phrae and Nakhon Ratchasima provinces, in black color. The simulated data obtained from the model were compared to the real data for these two provinces. From http://www.thailand-yellowpages.com/Thai/info/map.html

Fig. 4 The temporal evolution of infected human where the star indicates the actual data and the circles correspond to the simulated results. a) Phrae province in 2000, b) Phrae province in 2001, c) Nakhon Ratchasima in 2000, and d) Nakhon Ratchasima in 2001. All use the same time steps, 2.2 with $\gamma_A = 0.2$, $r_1 = 1/15$, $r_2 = 1/360$, and with γ_H taken to be dependent on the rainfall during the month

Fig. 5. The temporal evolution of infected human in a) 2000 and b) 2001 where the stars indicate real data while the circles indicate the simulation result. Here, time step = 2.2, $\gamma_A = 0.2$, $r_1 = 1/15$, $r_2 = 1/360$, and γ_H varies with the amount of rain fall during the month.



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