Abstract—Herpes zoster is a disease that manifests as a dermatological condition. The characteristic of this disease is an irritating skin rash with blisters. This is often limited to one side of body. From the data of Herpes zoster cases in Thailand, we found that age structure effects to the transmission of this disease. In this study, we construct the age structural model of Herpes zoster in Thailand. The local stability analysis of this model is given. The numerical solutions are shown to confirm the analytical results.

Keywords—Age structural model, Herpes zoster, local stability, Numerical solution.

I. INTRODUCTION

HERPES zoster is a viral disease characterized by a painful skin rash with blisters in a limited area on one side of the body (left or right), often in a stripe. The initial infection with varicella zoster virus (VZV) causes the acute, short-lived illness chickenpox which generally occurs in children and young adults. Herpes zoster is not the same disease as herpes simplex, despite the name similarity; both the varicella zoster virus and herpes simplex virus belong to the same viral subfamily Alpha herpesviridae [1]. This disease is also known as shingles or zoster, is a reactivated VZV infection of the sensory nerve ganglion and the peripheral nerve and its branches. Inflammation of the nerve axons results in a painful, burning sensation on the affected dermatome(s) being supplied by the peripheral nerve. In each year, there are about 1 million herpes zoster cases occur in the U.S. Herpes zoster more commonly occurs in white patients (35% higher incidence than in black patients), elderly patients (3 to 7 times higher incidence than in the general population). Some studies report a higher case in women (3.8 cases per 1,000 person-years and 2.6 cases per 1,000 person-years among men) [2]. Adults aged 60 years and older are at risk of infection with this disease. Recurrent of infection with this disease increases with advancing age. The highest case occurs between 50 and 80 years old. Each person has 15-20% risk of herpes zoster infection.

The symptoms are usually followed by sensations of burning pain, itching, hyperesthesia or paresthesia. The pain may be mild to extreme in the affected dermatome, with sensations that are often described as stinging, tingling, aching, numbing or throbbing, and can be interspersed with quick stabs of agonizing pain. In children, Herpes zoster is often painless, but older people are more severe than children. After 1-2 days or sometimes 3 weeks, the initial phase of patients is followed by the appearance of the skin rash. The pain and rash most commonly occurs on the torso. In Thailand, Appearance of Herpes zoster is increasing with the age. The chance of general people may be infected with Herpes zoster is 1.2-4.8/1,000 populations per year. The person who is older than 60 years old has the chance of infected about 7.2-11.8/1,000 populations per year. The trend of Thai Herpes zoster cases is increasing every year. The patient who is greater than 65 years old has the highest incidence rate [3]-[5]. Age is influence to the transmission of Herpes zoster. Older people who greater than 60 years has higher risk of Herpes zoster than children 5-10 times.

The data of Herpes zoster in Thailand are shown in Figs. 1 and 2. We will see that Herpes zoster cases in Thailand are
increasing every year. The highest incidence rate is found in the people greater than 65 years old.

In 1998, Allen and Thrasher [6] analyzed the model for Varicella and Herpes Zoster in United States considering vaccination in each age group and finding the appropriated parameters. In 2003, Schuette [7] studied the transmission of Varicella-zoster virus by using mathematical model. We found that basic reproductive number of Varicella-Zoster virus is calculated from the sum of basic reproduction of Varicella virus and Zoster virus. In the same year, Owocoey and Popoola [8] studied the relation between the Herpes zoster and HIV in Nigerian. They found that 50% of Herpes zoster cases are infected with HIV. In 2008, Zaman and et al. [9] studied SIR model by finding the equilibrium points and their stability conditions. The numerical solutions are evaluated by using Runge–Kutta method. Their results can be applied to many diseases such as varicella, mumps, etc. In 2010, Weinberg and et al. [10] studied HIV patients and normal people with Herpes zoster vaccination to study Herpes zoster infection. From the data of Herpes zoster in Thailand, we found that age structure effects to the transmission of this disease. In this study, we formulate the mathematical model of Herpes zoster in Thailand by considering age group of human.

II. TRANSMISSION MODEL

The transmission model of Herpes Zoster by age structure is considered. We are interested in the dynamical changes of human population with the transmission of Herpes Zoster. We separate the human population into two groups. First age group represents the human less than 65 years old. Second age group represents the human greater than 65 years old. Each human age group is divided into 6 subgroups; susceptible, exposed, infected, recovered, weak immunity and re-infected groups.

We define the variables and parameters in our model as follows:

- $S_a(t)$ is the number of susceptible human of the first age group at time $t$,
- $E_a(t)$ is the number of exposed human of the first age group at time $t$,
- $I_a(t)$ is the number of infected human of the first age group at time $t$,
- $R_a(t)$ is the number of recovered human of the first age group at time $t$,
- $W_a(t)$ is the number of weak immunity human of the first age group at time $t$,
- $Z_a(t)$ is the number of re-infected human of the first age group at time $t$,
- $S_b(t)$ is the number of susceptible human of the second age group at time $t$,
- $E_b(t)$ is the number of exposed human of the second group at time $t$,
- $I_b(t)$ is the number of infected human of the second age group at time $t$,
- $R_b(t)$ is the number of recovered human of the second age group at time $t$,
- $W_b(t)$ is the number of weak immunity human of the second age group at time $t$,
- $Z_b(t)$ is the number of re-infected human of the second age group at time $t$,
- $N$ is the total human,
- $N_a$ is the total juvenile human,
- $N_b$ is the total adult human,
- $a$ is the birth rate of human,
- $c_1$ is the rate at which susceptible human change to be exposed human in the juvenile group,
- $c_2$ is the rate at which exposed human change to be infectious human in the juvenile group,
- $c_3$ is the rate at which infectious human change to be recovered human in the juvenile group,
- $c_4$ is the rate at which recovered human change to be weak immunity human in the juvenile group,
- $c_5$ is the rate at which weak immunity human change to be re-infected human in the juvenile group,
- $c_6$ is the rate at which weak immunity human change to be recovered human in the juvenile group,
- $c_7$ is the rate at which susceptible human change to be exposed human in the adult group,
- $c_8$ is the rate at which exposed human change to be infectious human in the adult group,
- $c_9$ is the rate at which infectious human change to be recovered human in the adult group,
- $c_{10}$ is the rate at which recovered human change to be weak immunity human in the adult group,
- $c_{11}$ is the rate at which weak immunity human change to be re-infected human in the adult group,
- $c_{12}$ is the rate at which weak immunity human change to be recovered human in the adult group,
- $d$ is the death rate,
- $h$ is the proportion of juvenile human.

The dynamical equations for juvenile human are given by

\[
\frac{d}{dt} S_a = a \mu h N - (c_1 + d) S_a \quad (1)
\]

\[
\frac{d}{dt} E_a = c_1 S_a - (c_2 + d) E_a \quad (2)
\]

\[
\frac{d}{dt} I_a = c_2 E_a - (c_3 + d) I_a \quad (3)
\]
\[
\frac{d}{dt} R_a = c_3 I_a + c_7 Z_a + c_9 W_a - (c_4 + d) R_a \\
\frac{d}{dt} W_a = c_4 R_a - (c_5 + c_6 + d) W_a \\
\frac{d}{dt} Z_a = c_3 W_a - (c_7 + d) Z_a \\
\text{where } N_a = S_a + E_a + I_a + R_a + W_a + Z_a. \\
\frac{d}{dt} s_a = m_h - (c_1 + d)s_a \\
\frac{d}{dt} e_a = c_3 s_a - (c_2 + d)e_a \\
\frac{d}{dt} i_a = c_7 e_a - (c_3 + d)i_a \\
\text{where } N = N_a + N_b, \quad s_a + e_a + i_a + r_a + w_a + z_a = 1 \quad \text{and} \\
s_b + e_b + i_b + r_b + w_b + z_b = 1. \\
\]

The dynamical equations for adult human are given by
\[
\frac{d}{dt} S_b = (1 - a) \mu_b N - (e_1 + d) S_b \\
\frac{d}{dt} E_b = e_3 S_b - (c_2 + d) E_b \\
\frac{d}{dt} I_b = c_2 E_b - (c_3 + d) I_b \\
\frac{d}{dt} R_b = c_3 I_b + c_7 Z_b + c_9 W_b - (c_4 + d) R_b \\
\frac{d}{dt} W_b = c_4 R_b - (c_5 + c_6 + d) W_b \\
\frac{d}{dt} Z_b = c_3 W_b - (c_7 + d) Z_b \\
\text{where } N_b = S_b + E_b + I_b + R_b + W_b + Z_b. \\
\]

We suppose that the total human population and the human population for each group have constant sizes. Thus the rate of change for each human group equals to zero; i.e.
\[
\frac{d}{dt} N = 0, \quad \frac{d}{dt} N_a = 0, \quad \frac{d}{dt} N_b = 0. \\
\]

Then we obtain
\[
d = \mu_b, \quad N_a = aN, \quad N_b = (1 - a)N. \\
\]

We normalize our dynamical equations by letting
\[
s_a = S_a / N_a, \quad e_a = E_a / N_a, \quad i_a = I_a / N_a, \\
r_a = R_a / N_a, \quad w_a = W_a / N_a, \quad z_a = Z_a / N_a, \\
s_b = S_b / N_b, \quad e_b = E_b / N_b, \quad i_b = I_b / N_b, \\
r_b = R_b / N_b, \quad w_b = W_b / N_b, \quad z_b = Z_b / N_b. \\
\]
then the reduced equations become
\[
(\frac{d}{dt} s_a = \frac{a \mu_b N}{(c_1 + \mu_b)N_a}, \\
\frac{d}{dt} e_a = \frac{ac_3 \mu_b N}{(c_1 + \mu_b)(c_2 + \mu_b)N_a}, \\
\frac{d}{dt} i_a = \frac{ac_7 \mu_b N}{(c_1 + \mu_b)(c_3 + \mu_b)N_a}. \\
\]
\[(\begin{array}{c}
(\xi + c_6 + \mu_b) (-q_4 b_7) (c_7 + \mu_b) (c_3 + \mu_b) + c_1 (c_2 - c_3 + c_7)
+ c_4 (c_5 + c_7 + \mu_b) N_b)
\end{array})
\]

\[r_a^* = \frac{-c_7 (c_3 + \mu_b)) N_b + c_1 (c_2 - c_3 + c_7)}{(q + \mu_b)(c_2 + \mu_b) ((q + c_6 + \mu_b) (q + \mu_b))}
\]

\[= \frac{c_5 (c_2 + \mu_b) (c_3 + \mu_b) + c_1 (c_2 - c_3 + c_7)}{(c_1 + \mu_b)(c_2 + \mu_b)(c_3 + \mu_b)(c_5 + c_6 + \mu_b)(c_7 + \mu_b)}
\]

\[s_b^* = \frac{(l - a) \mu_b N}{(e_1 + \mu_b) N_b}
\]

\[e_b^* = \frac{(l - a) e_2 \mu_b N}{(e_1 + \mu_b)(e_2 + \mu_b)(e_3 + \mu_b) N_a}
\]

\[i_b^* = \frac{(l - a) e_2 \mu_b N}{(e_1 + \mu_b)(e_2 + \mu_b)(e_3 + \mu_b) N_a}
\]

\[w_b^* = \frac{(l - a) e_2 \mu_b N}{(e_1 + \mu_b)(e_2 + \mu_b)(e_3 + \mu_b) N_b}
\]

\[\lambda_1 = -c_1 - \mu_b,
\]

\[\lambda_2 = -c_2 - \mu_b,
\]

\[\lambda_3 = -c_3 - \mu_b,
\]

\[\lambda_{4,5} = \frac{1}{2}(-c_4 - c_5 - c_6 - c_7 - 2\mu_b)
\]

\[\pm \sqrt{c_4^2 + (c_5 + c_6 - c_7)^2 - 2c_4(c_5 - c_6 + c_7)}
\]

\[\lambda_6 = -e_1 - \mu_b,
\]

\[\lambda_7 = -e_2 - \mu_b,
\]

\[\lambda_8 = -e_3 - \mu_b,
\]

\[\lambda_{9,10} = \frac{1}{2}(-e_4 - e_5 - e_6 - e_7 - 2\mu_b)
\]

\[\pm \sqrt{e_4^2 + (e_5 + e_6 - e_7)^2 - 2e_4(e_5 - e_6 + e_7)}
\]

We can see that all eigenvalues have negative real parts for

\[R_0^a > 1 \text{ and } R_0^b > 1 \text{ where}
\]

\[R_0^a = \frac{c_4 (c_4 + 2c_6) + (c_5 + c_6)^2 + c_7^2}{2c_4 c_5 + 2(c_4 + c_5 + c_6) c_7}
\]

and

\[R_0^b = \frac{c_4 (c_4 + 2c_6) + (c_5 + c_6)^2 + c_7^2}{2c_4 c_5 + 2(c_4 + c_5 + c_6) c_7}
\]

Therefore, the positive state is local stable for \(R_0^a > 1 \text{ and } R_0^b > 1\).

IV. NUMERICAL SOLUTIONS

The numerical solutions of our model are shown with the parameters defined as follows: \(\mu_b = 1/(365*70)\); \(a = 0.4\), \(c_1 = 0.6\), \(c_2 = 1/10\), \(c_3 = 1/14\), \(c_4 = 1/(365*2)\), \(c_5 = 1/(365*2)\), \(c_6 = 1/14\), \(c_7 = 1/14\), \(e_1 = 0.6\), \(e_2 := 1/10\); \(e_3 := 1/14\); \(e_4 = 1/365\), \(e_5 := 1/365\), \(e_6 = 1/14\), \(e_7 = 1/10\). Each parameters are obtained from simulation. \(R_0^a = 1.01\) and \(R_0^b = 1.03\). Steady state solution is given by (0.000043, 0.00026, 0.00037, 0.433348, 0.015999, 0.000065, 0.000391, 0.000547, 0.980208, 0.0184349).

From our simulation, we can see that the solutions converge to the non-trivial solution for \(R_0^a > 1 \text{ and } R_0^b > 1\) corresponding to the analytical solutions.

III. ANALYTICAL SOLUTIONS

The local stable of each steady state is determined by the sign of eigenvalues for each steady state. If all eigenvalues have negative real parts, then that steady state is local stable [11]. The eigenvalues are the results of the following characteristic equation:

\[\det(J_e - \lambda I_{10}) = 0\]
V. DISCUSSION AND CONCLUSION

Fig. 3 Numerical solutions of our dynamical equations

Fig. 4 Time series solutions for the different basic reproductive number (a) $R_0^a = 1.01$ and $R_0^b = 1.03$ (b) $R_0^a = 1.1$ and $R_0^b = 1.2$
From our analysis, we found the condition for local stability. We found that if $R_0^a > 1$ and $R_0^b > 1$, then the positive steady state is locally stable, where

$$R_0^a = \frac{e_4 (e_4 + 2e_6) + (e_5 + e_6)^2 + e_7^2}{2c_4 e_5 + 2(c_4 + e_5 + e_6) e_7}$$

and

$$R_0^b = \frac{e_4 (e_4 + 2e_6) + (e_5 + e_6)^2 + e_7^2}{2c_4 e_5 + 2(c_4 + e_5 + e_6) e_7}.$$

We defined $R_0^a$ and $R_0^b$ as the basic reproductive number of this disease. It represents the average number of secondary cases reproduced from the primary cases [12]-[15]. From Fig. 4, we can see that the different basic reproductive number can produce the different outbreak time and highest cases. The results of this study should introduce the way for reducing the transmission of this disease.

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