ผลของการดูแลสุขลักษณะและการเลี้ยงดูบุตรด้วยนมแม่ต่อพลวัตของแบบจำลอง
ทางคณิตศาสตร์โรคปอดอักเสบสำหรับเด็กอายุต่ำกว่า 5 ขวบในประเทศไทย
The effect of children under five-year old with hygiene care and
breastfeeding on dynamics of pneumonia model for Thailand

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บทคัดย่อ

โรคปอดอักเสบเป็นโรคระบบทางเดินหายใจที่มีสาเหตุการเกิดหลักจากการติดเชื้อไวรัส ส่วนมากมักพบในเด็กมากกว่าผู้ใหญ่และเป็นสาเหตุสำคัญที่ทำให้เกิดการเจ็บป่วยขั้นรุนแรงรวมถึงการเสียชีวิตในเด็กที่มีอายุต่ำกว่า 5 ปีทั่วโลกรวมถึงในประเทศไทย ในงานวิจัยนี้ผู้ศึกษาได้สร้างแบบจำลองทางคณิตศาสตร์การระบาดของโรคปอดอักเสบ โดยศึกษาปัจจัยที่เกี่ยวข้องเพิ่มเติมคือ ภูมิต้านทานของเด็กที่ได้รับการเลี้ยงดูด้วยนมแม่ และผลจากการดูแลสุขลักษณะของเด็ก ทั้งนี้ ผู้ศึกษาได้วิเคราะห์หาเงื่อนไขของการเกิดโรคระบาดพร้อมด้วยความเสถียรของจุดวิกฤต และดัชนีการเกิดโรคระบาด โดยผลที่ได้รับจากการวิเคราะห์ความเสถียร ณ จุดวิกฤตก็คือ เพื่อไม่ให้มีการระบาดของโรคปอดอักเสบ ดัชนีการเกิดโรคระบาดจะต้องมีค่าน้อยกว่า 1 นอกจากนี้ผู้ศึกษายังได้แสดงผลเชิงตัวเลขของแบบจำลองการระบาดของโรคปอดอักเสบนี้ ซึ่งได้ผลตรงกับทฤษฎีที่ว่าการเลี้ยงดูบุตรด้วยนมแม่และใส่ใจดูแลสุขลักษณะของบุตรจะนำไปสู่ภาวะการไร้ซึ่งการระบาดของโรคปอดอักเสบ

ABSTRACT

Pneumonia is a respiratory disease that is mainly caused by virus infection. It is mostly found in children more than adults and it is one of the leading causes of serious illness and deaths among children under five years old around the world including Thailand. In this paper we develop a deterministic model of the transmission dynamics of pneumonia with the possibility of infection resistance of children with breastfeeding together with the impact of hygiene care of susceptible group. The conditions for the clearance and persistence of the pneumonia infection through the stability of the equilibria are derived. The basic reproduction number and its sensitivity are calculated. Stability of equilibrium points indicates that the basic reproduction number has to be maintained at the level less than unity in order to eliminate the disease. Sensitivity analysis of basic reproduction number and numerical simulation demonstrates that the immunity to disease due to breastfeeding and hygiene care of children should be encouraged in order to achieve a disease-free population.

1. INTRODUCTION

Pneumonia is a respiratory disease which is a severe form of acute lower respiratory infection that specifically affects the lungs. This disease is mainly caused by virus and bacterial infection. It is found in children more than adults. It can be contacted through the pathogens which may be spread through contaminated air droplets when an infected person coughs or sneezes. Evidence showed that children have a higher risk in developing pneumonia when they have weakened immune system (WHO, 2012). Children who get infected can have a range of symptoms such as severely ill with high fever, rapid breathing, cough, fever, headaches and loss appetite (WHO, 2006).

Worldwide, the cause of death among most of children under five years old in 2011 was pneumonia (WHO and UNICEF, 2006). It was occurred mostly in African and developing countries (Singh and Aneja, 2011). In Thailand, the number of pneumonia patients increases every year although there is a reduction in death of children due to this infection (Chaiya, 2015). By the data from the Bureau of Epidemiology of Thailand, the number of pneumonia patients and deaths by pneumonia in Thailand from 2011-2015 is shown in Figure 1. To prevent children from pneumonia infection, vaccination, promotion of adequate nutrition including breastfeeding and reducing indoor air pollution is essential (Jones et al., 2003; Singh and Aneja, 2011). Children who have been breastfed for at least 6 months will increase their disease resistance so breastfeeding should be done to many children as possible (Luby et al., 2005; Boccolini et al., 2011; Chotpitayasunondh, 2012; Srivastava et al., 2015). Furthermore, work by Luby et al (2005) suggested that hand washing plays a role in reducing the incidence of pneumonia (Kassa and Murthy, 2016). As for treatment, doctors used antibiotics to treat pneumonia caused by bacteria basing on the age and symptoms of patients, whereas antibiotics do not work when the cause of pneumonia is a virus. If the patients have viral pneumonia, doctor may prescribe an antiviral medicine to treat it.

Due to its importance of this disease together with a continuously increase in number of infectious, several studies have been performed through mathematical modeling for a better understanding and predicting the disease spreading e.g. the work by Otieno et al. (2012) is a simple pneumonia model involving the carriers group, the work by Ndelwa et al. (2015) involves the treatment group of pneumonia infected individuals, the work by Kassa and Murthy (2016) is a simple pneumonia model involving the exposed individuals group and the work by Tilahun et al. (2017) involves vaccinated individuals group. By studying the models, we can predict the spreading of pneumonia in terms of number of infected individuals and the duration of the outbreak, and with the factors studied in each model, this could lead to a national policy to take effective measures to reduce or control the effect of disease. In this paper, the researchers extended a pneumonia model of Kassa and Murthy (2016) by adding the effect of hygiene care efficiency and the effect of newborns with breastfeeding for the children under five years old in Thailand. The model is studied in both theoretically and numerically. Stability analysis is carried out and the basic reproduction number is determined to gain some relevant input factors that could lead to a better control of pneumonia. Finally, the sensitivity analysis of the basic reproduction number

and the model numerical simulation are performed to seek for parameters which can reduce the possibility of infection in the community.
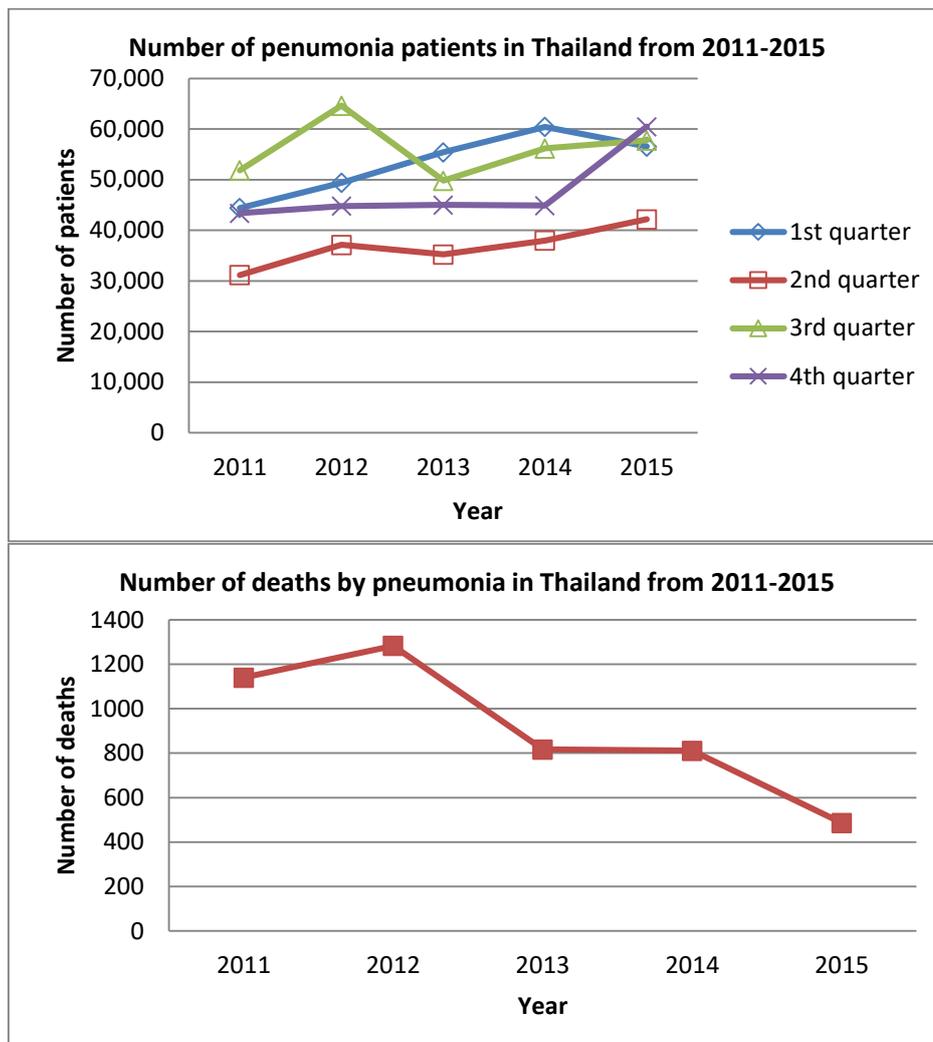

Figure 1 Data of pneumonia situation from 2011-2015 in Thailand (top) the number of pneumonia patients and (bottom) the number of deaths by pneumonia.

2. MODEL FORMULATION

The mathematical model of pneumonia is proposed where children are divided into five subgroups at time t: M is the number of individuals with breastfeeding, S is the number of susceptible individuals, E is the number of exposed individuals, I is the number of infected individuals and R is the number of recovered individuals, with the total population size N where $N = M + S + E + I + R$. The dynamics transmission of pneumonia associated with these five subgroups are illustrated in Figure 2. In this model, the children whom are breastfed (M) are added and are assumed to have better immune to pneumonia comparing to the susceptible children (S), however, the children from group M can transferred to susceptible individuals group with the rate of p. The susceptible individuals can be infected which is represented by the term $(1-q)\beta SI$ and are transferred to exposed individuals (E) and eventually to the infected individuals (I). In addition, the infected

individuals recover with the rate of σ and finally those who recover can possibly lose the immunity and be infected again, therefore they are transferred to the susceptible group with the rate γ.
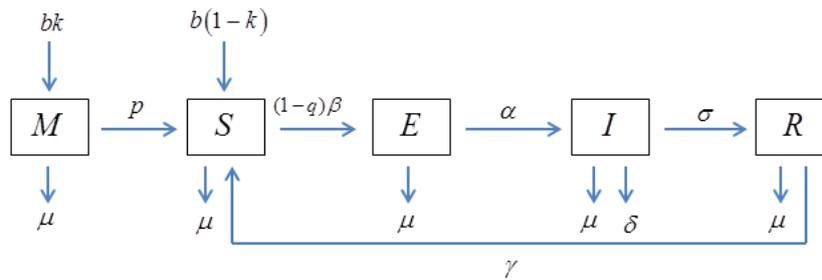

Figure 2 A schematic diagram of pneumonia dynamics.

From diagram in Figure 2, the system that governs the differential equations is given by:

$$\frac{dM}{dt} = bk - pM - \mu M \tag{2.1}$$

$$\frac{dS}{dt} = b(1-k) - (1-q)\beta SI + pM + \gamma R - \mu S \tag{2.2}$$

$$\frac{dE}{dt} = (1-q)\beta SI - \mu E - \alpha E \tag{2.3}$$

$$\frac{dI}{dt} = \alpha E - \mu I - \delta I - \sigma I \tag{2.4}$$

$$\frac{dR}{dt} = \sigma I - \gamma R - \mu R \tag{2.5}$$

with the initial conditions

$$M(0) > 0, \quad S(0) > 0, \quad E(0) > 0, \quad I(0) > 0, \quad R(0) > 0 .$$

Here, p is the resistance to immunity rate of individuals with breastfeeding to become susceptible individuals, b is the birth rate of human population, β is the transmission rate of susceptible to exposed individuals, α is the transmission rate of exposed to infected individuals, σ is the recovery rate, γ is the loss of immunity rate, q is the percentage of hygiene care efficiency, δ is the mortality rate caused by infection, k is the fraction of individuals whom are breastfed and μ is the natural mortality rate of children who are younger than 5 years old in Thailand. Note that we set $0 < q < 1$ and $0 < k < 1$.

2.1 Boundary of solution

In this section, we determine the biological feasible region of the system of equations (2.1) - (2.5).

Theorem 2.1. The feasible region of the system of equations (2.1) - (2.5) is within the region

$$\Gamma = \left\{ (M, S, E, I, R) \in \mathfrak{R}_+^5 : N \le \frac{b}{\mu} \right\}.$$

Proof. The total dynamic of children population is calculated by adding equations (2.1) - (2.5), we then obtain

$$\frac{dN}{dt} = b - (M + S + E + I + R)\mu - \delta I = b - \mu N - \delta I . \tag{2.6}$$

Consider equation (2.6) above, we obtain $\frac{dN}{dt} \le b - \mu N$.

By taking integration both sides of this inequality, we have

$$\int_0^t \frac{dN}{b-\mu N} \le \int_0^t dt.$$

And

$$b - \mu N_t \ge (b - \mu N_0)e^{-\mu t}.$$

Therefore,

$$N_t \le \frac{b}{\mu} - (\frac{b}{\mu} - N_0)e^{-\mu t}.$$

As $t \to \infty$, we obtain $N_t \to \frac{b}{\mu}$. This implies that $0 \le N_t \le \frac{b}{\mu}$. Therefore, the feasible region of the system of

equations (2.1)-(2.5) is within the region $\Gamma = \left\{ (M, S, E, I, R) \in \mathfrak{R}_+^5 : N \le \frac{b}{\mu} \right\}$. Hence, every solution with

condition to $\mathfrak{R}_+^5$ will be considered inside the region Γ, where the uniqueness of solutions, usual existence and

continuation results are satisfied.

2.2 Equilibrium point

Within this model, we determine the equilibrium point relating to M, S, E, I and R. By setting the

right hand side of the system of equations (2.1)-(2.5) to be zero, we obtain two main equilibrium points:

1. the disease-free equilibrium point

$$E_0 = (M_0^*, S_0^*, E_0^*, I_0^*, R_0^*) = \left(\frac{bk}{p+\mu}, \frac{b(1-k)(p+\mu)+pbk}{\mu(p+\mu)}, 0, 0, 0 \right) \text{ and }$$

2. the endemic equilibrium point $E_1 = (M_1^*, S_1^*, E_1^*, I_1^*, R_1^*)$ where

$$M_1^* = \frac{bk}{p+\mu}, \quad S_1^* = \frac{(\mu+\delta+\sigma)(\mu+\alpha)}{(1-q)\alpha\beta}, \quad E_1^* = \frac{(\mu+\delta+\sigma)I_1^*}{\alpha},$$

$$I_1^* = \frac{b(1-k)(1-q)\alpha\beta(\gamma+\mu) + pM_1^*(1-q)\alpha\beta(\gamma+\mu) - \mu(\gamma+\mu)(\mu+\delta+\sigma)(\mu+\alpha)}{(1-q)\beta[(\gamma+\mu)(\mu+\delta+\sigma)(\mu+\alpha) - \alpha\gamma\sigma]} \text{ and }$$

$$R_1^* = \frac{\sigma I_1^*}{\gamma+\mu}.$$

2.3 Basic reproduction number (R_0)

The basic reproduction number (R_0) is the average number of secondary infections generated by a

single infection, it is one of the important threshold quantities to determine whether or not an infectious disease

will spread among population (Diekmann et al., 1990; Heffernan et al., 2005). In this section, the basic

reproduction number is calculated by using next generation matrix method (van den Driessche and Watmough,

2002).

Let $X = (E, I)^T$, $\mathbf{F}(X)$ be the matrix of new infections and $\mathbf{V}(X)$ be the matrix of transfer between

components for the infective equations. From the system of equations (2.1)-(2.5), we have

$$\mathbf{F}(X) = \begin{bmatrix} (1-q)\beta SI \\ 0 \end{bmatrix} \text{ and } \mathbf{V}(X) = \begin{bmatrix} (\mu+\alpha)E \\ -\alpha E + (\mu+\delta+\sigma)I \end{bmatrix}.$$

The Jacobian matrices of $\mathbf{F}(X)$ and $\mathbf{V}(X)$ are

$$F(X) = \begin{bmatrix} 0 & (1-q)\beta S \\ 0 & 0 \end{bmatrix} \text{ and } V(X) = \begin{bmatrix} \mu+\alpha & 0 \\ -\alpha & \mu+\delta+\sigma \end{bmatrix}.$$

By substituting disease-free equilibrium point $E_0 = \left(\dfrac{bk}{p+\mu}, \dfrac{b(1-k)(p+\mu)+pbk}{\mu(p+\mu)}, 0,0,0\right)$ in the Jacobian

matrices above, we get

$$F(E_0) = \begin{bmatrix} 0 & \dfrac{(1-q)\beta(b(1-k)(p+\mu)+pbk)}{\mu(p+\mu)} \\ 0 & 0 \end{bmatrix} \text{ and } V(E_0) = \begin{bmatrix} \mu+\alpha & 0 \\ -\alpha & \mu+\delta+\sigma \end{bmatrix}.$$

The next generation matrix is

$$FV^{-1} = \begin{bmatrix} \dfrac{(1-q)\alpha\beta(b(1-k)(p+\mu)+pbk)}{\mu(\mu+\alpha)(\mu+\delta+\sigma)(p+\mu)} & \dfrac{(1-q)\beta(b(1-k)(p+\mu)+pbk)}{\mu(\mu+\delta+\sigma)(p+\mu)} \\ 0 & 0 \end{bmatrix}.$$

The spectral radius of FV^{-1} is $\rho(FV^{-1}) = \dfrac{\alpha\beta(1-q)[b(1-k)(p+\mu)+pbk]}{\mu(\mu+\alpha)(\mu+\delta+\sigma)(p+\mu)}$. Therefore, the basic

reproduction number of the system of equations (2.1)-(2.5) is $R_0 = \dfrac{\alpha\beta(1-q)[b(1-k)(p+\mu)+pbk]}{\mu(\mu+\alpha)(\mu+\delta+\sigma)(p+\mu)}$.

2.4 Local stability analysis

The local stability of this model is studied by constructing Jacobian matrix of the system of equations (2.1) - (2.5) as follows:

$$J(M,S,E,I,R) = \begin{bmatrix} -p-\mu & 0 & 0 & 0 & 0 \\ p & -(1-q)\beta I-\mu & 0 & -(1-q)\beta S & \gamma \\ 0 & (1-q)\beta I & -\mu-\alpha & (1-q)\beta S & 0 \\ 0 & 0 & \alpha & -\mu-\delta-\sigma & 0 \\ 0 & 0 & 0 & \sigma & -\gamma-\mu \end{bmatrix}. \qquad (2.7)$$

Theorem 2.2. (local stability at E_0) The disease-free equilibrium point, E_0 is locally asymptotically stable if $R_0 < 1$, and unstable If $R_0 > 1$.

Proof. The Jacobian matrix about the disease-free equilibrium point E_0 is determined by substituting E_0 in (2.7), thus we obtain

$$J(E_0) = \begin{bmatrix} -p-\mu & 0 & 0 & 0 & 0 \\ p & -\mu & 0 & -(1-q)\beta S_0^* & \gamma \\ 0 & 0 & -\mu-\alpha & (1-q)\beta S_0^* & 0 \\ 0 & 0 & \alpha & -\mu-\delta-\sigma & 0 \\ 0 & 0 & 0 & \sigma & -\gamma-\mu \end{bmatrix}. \qquad (2.8)$$

The characteristic equation of the Jacobian matrix (2.8) is

$$(-p-\mu-\lambda)(-\mu-\lambda)(-\gamma-\mu-\lambda)[\lambda^2+(2\mu+\alpha+\delta+\sigma)\lambda+(\mu+\alpha)(\mu+\delta+\sigma)-(1-q)\alpha\beta S_0^*]=0$$
$$(2.9)$$

From the characteristic equation (2.9), the first three eigenvalues obtained are $\lambda_1 = -p-\mu < 0, \lambda_2 = -\mu < 0$ and $\lambda_3 = -\gamma-\mu < 0$ which are all negative. Next, consider equation

$$\lambda^2+(2\mu+\alpha+\delta+\sigma)\lambda+(\mu+\alpha)(\mu+\delta+\sigma)-(1-q)\alpha\beta S_0^* = 0$$

where

$$a_1 = 2\mu + \alpha + \delta + \sigma > 0 \quad \text{and}$$

$$a_2 = (\mu + \alpha)(\mu + \delta + \sigma) - (1-q)\alpha\beta S_0^*$$

$$= (\mu + \alpha)(\mu + \delta + \sigma)(1 - R_0), \text{ thus } a_2 > 0 \text{ when } R_0 < 1.$$

This is satisfied with the Routh-Hurwitz criteria for $n = 2$ (where n is the highest degree of characteristic equation) i.e. $a_1 > 0$ and $a_2 > 0$ when $R_0 < 1$.

Therefore, the disease-free equilibrium point E_0 is locally asymptotically stable if $R_0 < 1$. If $R_0 > 1$, the characteristic equation (2.9) would have some positive eigenvalues, resulting in an unstable. This completes the proof.

Theorem 2.3. (local stability at E_1) For $R_0 > 1$, the endemic equilibrium point, E_1 exists and is locally asymptotically stable if and only if the coefficients of the characteristic equation satisfy with the Routh-Hurwitz criteria for $n = 4$.

Proof. The endemic equilibrium point exists when $I_1^* > 0$ and by section 2.2. i.e. when

$$b(1-k)(1-q)\alpha\beta(\gamma + \mu) + p\frac{bk}{(p+\mu)}(1-q)\alpha\beta(\gamma + \mu) > \mu(\gamma + \mu)(\mu + \delta + \sigma)(\mu + \alpha).$$

Then,
$$b(1-k)(1-q)\alpha\beta + p\frac{bk}{(p+\mu)}(1-q)\alpha\beta > \mu(\mu + \delta + \sigma)(\mu + \alpha),$$

$$\alpha\beta(1-q)[b(1-k)(p+\mu) + pbk] > \mu(\mu + \delta + \sigma)(\mu + \alpha)(p+\mu)$$

$$\frac{\alpha\beta(1-q)[b(1-k)(p+\mu) + pbk]}{\mu(\mu + \delta + \sigma)(\mu + \alpha)(p+\mu)} > 1 \text{ , i.e. when } R_0 > 1.$$

Therefore, the endemic equilibrium point exists when $R_0 > 1$.

Next, the Jacobian matrix about the endemic equilibrium point E_1 is given by

$$J(E_1) = \begin{bmatrix} -p - \mu & 0 & 0 & 0 & 0 \\ p & -(1-q)\beta I_1^* - \mu & 0 & -(1-q)\beta S_1^* & \gamma \\ 0 & (1-q)\beta I_1^* & -\mu - \alpha & (1-q)\beta S_1^* & 0 \\ 0 & 0 & \alpha & -\mu - \delta - \sigma & 0 \\ 0 & 0 & 0 & \sigma & -\gamma - \mu \end{bmatrix}. \quad (2.10)$$

The characteristic equation of Jacobian matrix (2.10) can be written in the form

$$\lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4 = 0,$$

where

$$a_1 = (1-q)\beta I_1^* + \mu + B + C + D,$$

$$a_2 = (1-q)B\beta I_1^* + (1-q)C\beta I_1^* + (1-q)D\beta I_1^* + B\mu + C\mu + D\mu + BC + BD,$$

$$a_3 = (1-q)BC\beta I_1^* + (1-q)BD\beta I_1^* + (1-q)CD\beta I_1^* + BC\mu + BD\mu,$$

$$a_4 = (1-q)\beta I_1^*[BCD - \alpha\gamma\sigma],$$

where $B = \gamma + \mu, C = \mu + \alpha$ and $D = \mu + \delta + \sigma$.   (2.11)

From the above characteristic equation (2 . 1 1), for $R_0 > 1$ the endemic equilibrium point E_1 will be locally asymptotically stable when the coefficients of $\lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4 = 0$ satisfy with the Routh-Hurwitz criteria for $n = 4$ i.e. $a_1 > 0, a_3 > 0, a_4 > 0, a_1a_2a_3 > a_3^2 + a_1^2a_4$. It can be seen that in equation (2.11), $a_1 > 0$

and $a_3 > 0$ because every term is positive while $a_4 > 0$ because by calculation $BCD > \alpha\gamma\sigma$, hence only the last condition is left. Therefore, the endemic equilibrium point is locally asymptotically stable if and only if $a_1 a_2 a_3 > a_3^2 + a_1^2 a_4$. This completes the proof.

2.5 Global stability of disease-free equilibrium point

The Lyapunov's second method is applied to find the condition of E_1 to have global stability.

Theorem 2.4. (global stability at E_0) If $\dfrac{(1-q)\beta b}{\mu(\mu+\delta)} < 1$, then E_0 is globally asymptotically stable in Γ.

Proof. Let the Lyapunov function be as follows:

$$L = E + I + R.$$

We have $\dfrac{dL}{dt} = [(1-q)\beta S - \mu - \delta]I - \mu E - (\gamma + \mu)R.$

Since $S \le \dfrac{b}{\mu}$, therefore $\dfrac{dL}{dt} \le [\dfrac{(1-q)\beta b}{\mu} - \mu - \delta]I - \mu E - (\gamma + \mu)R.$

Hence, $\dfrac{dL}{dt} < 0$ when $\dfrac{(1-q)\beta b}{\mu} < \mu + \delta.$

Therefore, the disease-free equilibrium point E_0 is globally asymptotically stable when $\dfrac{(1-q)\beta b}{\mu(\mu+\delta)} < 1.$

2.6 Sensitivity analysis

Sensitivity indices allow us to measure the relative change in a variable when a parameter changes. The normalized forward sensitivity index of a variable to a parameter is the ratio of the relative change in the variable to the relative change in the parameter. If the variable is a differentiable function of the parameter, the sensitivity index is then defined using partial derivatives (Ngoteya and Gyekye, 2015).

Definition 2.1. The normalized forward-sensitivity index of R_0 that depends differentiably on a parameter, p, is defined as (Ngoteya and Gyekye, 2015):

$$\Upsilon_p^{R_0} = \frac{\partial R_0}{\partial p} \times \frac{p}{R_0}. \tag{2.12}$$

In particular in our model, the sensitivity indices of the basic reproduction number, R_0, with respect to the model parameters are examined. Using (2.12), we obtain:

$$\Upsilon_\alpha^{R_0} = \frac{\partial R_0}{\partial \alpha} \cdot \frac{\alpha}{R_0} = 1 - \frac{\alpha}{\mu + \alpha},$$

$$\Upsilon_\beta^{R_0} = \frac{\partial R_0}{\partial \beta} \cdot \frac{\beta}{R_0} = 1,$$

$$\Upsilon_b^{R_0} = \frac{\partial R_0}{\partial b} \cdot \frac{b}{R_0} = 1,$$

$$\Upsilon_k^{R_0} = \frac{\partial R_0}{\partial k} \cdot \frac{k}{R_0} = -\frac{\mu k}{p + \mu(1-k)},$$

$$\Upsilon_p^{R_0} = \frac{\partial R_0}{\partial p} \cdot \frac{p}{R_0} = \frac{\mu p k}{(p+\mu)(p+\mu+\mu k)},$$

$$\Upsilon_q^{R_0} = \frac{\partial R_0}{\partial q} \cdot \frac{q}{R_0} = -\frac{q}{(1-q)},$$

$$\Upsilon_\delta^{R_0} = \frac{\partial R_0}{\partial \delta} \cdot \frac{\delta}{R_0} = -\frac{\delta}{\mu+\delta+\sigma},$$

$$\Upsilon_\sigma^{R_0} = \frac{\partial R_0}{\partial \sigma} \cdot \frac{\sigma}{R_0} = -\frac{\sigma}{\mu+\delta+\sigma},$$

$$\Upsilon_\mu^{R_0} = \frac{\partial R_0}{\partial \mu} \cdot \frac{\mu}{R_0} = \frac{\mu(\mu+\alpha)(\mu+\delta+\sigma)(p+\mu)b(1-k)-[b(1-k)(p+\mu)+pbk]K}{(\mu+\alpha)(\mu+\delta+\sigma)(p+\mu)[b(1-k)(p+\mu)+pbk]},$$

when $K = \left\{ \mu[\mu(4\mu+3\delta+3\sigma)+\alpha(3\mu+\alpha\delta+2\sigma)+p(3\mu+2\delta+2\alpha)]+(\alpha\delta+\alpha\sigma)p \right\}$.

The value of sensitivity indices are given in Table 1, where they are calculated by using parameters values from Table 2. This information permits us to aware the strength of the model prediction with respect to parameter values. Furthermore, it allows us to acknowledge where to focus the control strategies. The positive sign of sensitivity index of R_0 (i.e. with respect to α, β, b, p) shows that a decrease in these values will lead to a decrease in R_0 and eventually to the elimination of the disease in the community. On the contrary, the negative sign of sensitivity index of R_0 (i.e. with respect to $\sigma, \delta, \mu, q, k$) shows that an increase in these values will lead to a reduction in R_0.

Table 1 Numerical values of sensitivity indices of R_0

Parameter	Index at Parameter Value	Sign
α	+0.0467	positive
σ	-0.2342	negative
δ	-0.7169	negative
β	+1.000	positive
μ	-0.3377	negative
q	-0.3333	negative
b	+1.000	positive
k	-0.3289	negative
p	+0.1002	positive

3. NUMERICAL SIMULATION

In this section, the system of equations (2.1) - (2.5) is solved numerically. The parameters within this model are chosen appropriately and are shown in Table 2. Numerical results of both exposed and infected individuals when the percentage of hygiene care efficiency (q) varies (0.25, 0.50, 0.75 and 0.90) and the resistance to immunity rate of individuals with breastfeeding to become susceptible individuals (p) varies (0.01, 0.05, 0.10 and 0.30) are shown in Figure 3 and Figure 4, respectively.

Table 2 Parameter values used in numerical study

| Parameter	Description	Value	Reference
b	The birth rate of human population of Thailand	0.4330 per week	National Statistical Office, Thailand (2013)
μ	The natural mortality rate of children who are younger than 5 years old in Thailand	0.0245 per week	National Statistical Office, Thailand (2013)
p	The resistance to immunity rate of individuals with breastfeeding to become susceptible individuals	0.0250 per week	Variable
β	The transmission rate of susceptible to exposed individuals	0.0250 per week	Estimate
α	The transmission rate of exposed to infected individuals	0.5000 per week	Kassa and Murthy, 2016
σ	The recovery rate	0.1176 per week	Kassa and Murthy, 2016
γ	The loss of immunity rate	0.5000 per week	Estimate
δ	The mortality rate caused by infection	0.3600 per week	National Statistical Office, Thailand (2013)
k	The fraction of individuals whom are breastfed	0.5000 per week	Estimate
q	The percentage of hygiene care efficiency	0.2500 per week	Variable

(a)   (b)
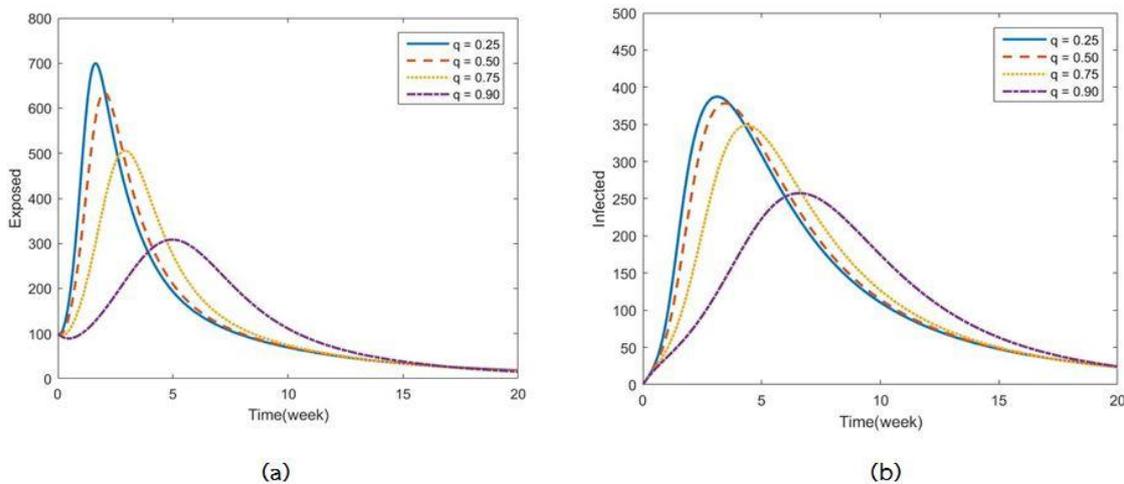
Figure 3 Numerical solutions of system of equations (2.1) - (2.5) obtained using parameters:

$$b = 0.433, \mu = 0.0245, \beta = 0.025, \alpha = 0.5, \sigma = 0.1176, \delta = 0.360, \gamma = 0.5, k = 0.5$$

and $p = 0.025$, where (a) is the dynamics of exposed individuals and (b) is the dynamics of infected individuals when q varies.

Figure 3 shows the changes in dynamics of the exposed and the infected individuals when the percentage of hygiene care efficiency (q) varies. Figure 3 (a) shows that when q increases, the number of exposed individuals decreases, respectively and the peak height is reduced. The peak height is dropped by approximately 400 individuals when q increases from 25% to 90%. In addition, time spent for the peak to occur is longer when the value of q is higher i.e. time for the peak to occur at $q = 0.25$ is about two weeks whereas it is about five weeks at $q = 0.90$. This result demonstrates the shift to the right of exposed peak. Figure 3 (b) shows similar pattern of dynamics to Figure 3 (a) i.e. when q increases, the peak height of infected individuals

decreases, respectively and the time for the peak to occur is longer, resulting in the shift to the right of infected peak.
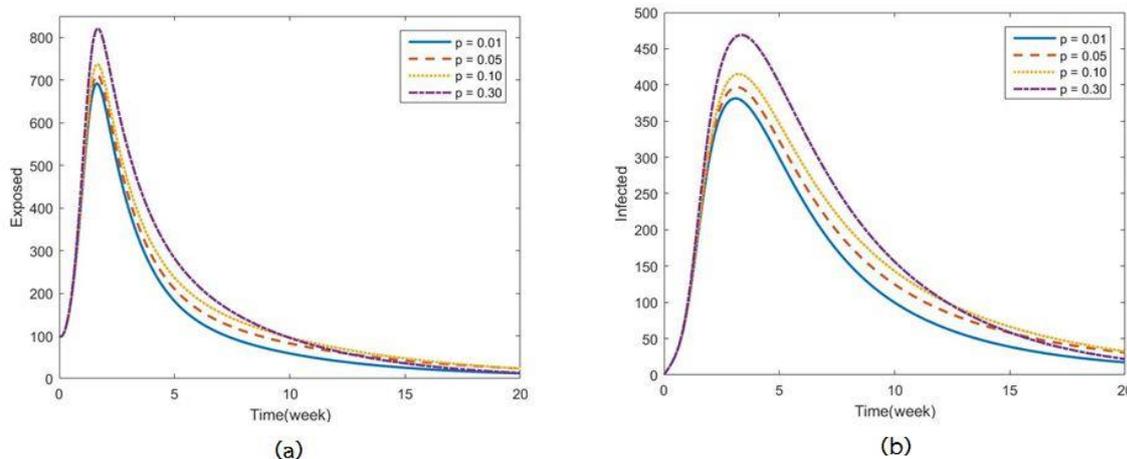

Figure 4   Numerical solutions of system of equations (2.1)-(2.5) obtained using parameters: $b = 0.433, \mu = 0.0245, \beta = 0.025, \alpha = 0.5, \sigma = 0.1176, \delta = 0.360, \gamma = 0.5, k = 0.5$ and $q = 0.25$, where (a) is the dynamics of exposed individuals and (b) is the dynamics of infected individuals when p varies

The result in Figure 4 demonstrates that when p increases, the peak height of exposed individuals and infected individuals increases, respectively, although there is no change in the time for the peak to occur. Further, the peak of both E and I at lower value of p is sharper than those of higher p. This result can be interpreted that when the resistance to immunity rate of individuals with breastfeeding to become susceptible individuals increases, it leads to an increase in both E and I with the longer period of outbreak.

Therefore, with above results the hygiene care efficiency has given significant impact in reducing the number of both exposed and infected individuals together with slowing the outbreak time of epidemic. Further, our results confirmed that children with breastfeeding lead to have less infection. Hence, both hygiene care and breastfeeding to children are essential and should be encouraged.

4. CONCLUSIONS

In this paper, we study the dynamics of pneumonia for under five-year old children in Thailand by developing mathematical model involving the effect of both hygiene care and children with breastfeeding. There are two main equilibrium points within this model, the disease-free and the endemic ones. The basic reproduction number is $R_0 = \dfrac{\alpha\beta(1-q)[b(1-k)(p+\mu)+pbk]}{\mu(\mu+\alpha)(\mu+\delta+\sigma)(p+\mu)}$ and it becomes a threshold condition for determining the stability of the model. If $R_0 < 1$, the disease-free equilibrium point is locally asymptotically stable, whereas if $R_0 > 1$, it is unstable. Furthermore, the disease-free equilibrium point is globally asymptotically stable when $\dfrac{(1-q)\beta b}{\mu(\mu+\delta)} < 1$. This means that when $R_0 < 1$, only the systems with the initial values of population

around disease-free equilibrium point would lead to the situation when the disease dies out, however, in the case when $\dfrac{(1-q)\beta b}{\mu(\mu+\delta)}<1$, the systems with any initial values of population would always reach the absence of epidemic situation. For $R_0>1$, the endemic equilibrium point exists and is locally asymptotically stable when the coefficients of characteristic equation (2.11) satisfy the Routh-Hurwitz criteria for $n=4$. The numerical simulations in this study show some impact of the percentage of hygiene care efficiency (q) and the resistance to immunity rate of individuals with breastfeeding to become susceptible individuals (p) to the number of both exposed and infected individuals. Our sensitivity analysis shows that by reducing the value of α, β, b and p and by increasing the value of σ, δ, μ, q and k will lead to a reduction in R_0. From this work, it is recommended that all newborns should be breastfed and all children should have a hygiene care as much as possible in order to effectively control the pneumonia disease.

5. ACKNOWLEDGEMENTS

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