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Stability analysis for the spread of health care-associated infection (HCAI) with and without self infection and control

Asrul Sani
Mathematics Department, Faculty of Mathematics and Sciences, Halu Oleo University, Kendari, Indonesia
saniasrul2001@yahoo.com

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Abstract

Patients during the course of their medical treatment in a hospital often contract healthcare-associated infections (HCAI) or hospital-acquired infections (HAI). Such infection has seriously become concerned in hospital management since many nosocomial infections have caused health care expenses increasing due to lengthened hospital stay and morbidity. In this study, we develop a mathematical model describing the spread of HCAI and discuss the dynamic behaviour of its solution. There are four type models developed; i.e., with cross infection only and self-cross infection, both are studied under with and without control. All models have a disease-free equilbrium and a positive endemic equilibrium.We derive a threshold condition for each model in which above the threshold the presence of a HCAI is able to spread in the unit care, otherwise, if below the threshold condition the infection is died out.The threshold condition is defined as the basic reproductive number. Numerical experiments show how the dynamics of HCAI is changing as several model parameters below and above threshold condition for each model.

Keywords: Mathematical Model, Nosocomial Infection, HCAI, hospital, stability.

Introduction

Health Care-Associated Infections, known as nosocomial infections, have become a serious problem on a community as they relate to patient safety. During medical treatment in healthcare facilities, the patients in particular those with weakened immune systems can easily get infections. Nosocomial infections spread to the susceptible patient in the clinical setting by various means. They can originate from the outside environment, contaminated equipment, bed linens, air droplets, another infected patient or from staff that may be infected. However, in some cases, it is difficult to determie the source of the infection. There are various types of HCAIs; for instance, associated bloodstream infections, associated urinary tract infections, surgical site infections, hospital-onset Clostridium difficile (C. difficile) infections, hospital-onset methicillin-resistant Staphylococcus aureus (MRSA) infections, hospital-onset clostridium difficile infections and device-associated infections[13,19].

Nosocomial infections have spread in the world with the highest cases occur in the poor and developing countries. WHO reported that 8,7% from 55 hospitals in 14 countries in Europe, 11,8% and 10% of cases in Middle Asian and in Southeast Asia and Pacific, respectively, still show the nosocomial infection[21]. In the developed countries such as US, the survey reported that it was estimated 722,000 HAIs in US acute care hospitals in 2011. Additionally, about 75,000 patients with HCAIs died during their hospitalizations[4]. In 2015, the US Centers for Disease Control and Prevention estimates that HCAIs in American hospitals account for approximately 1.7 million infections and 99,000 associated deaths each year and more than half of all HCAIs occur outside of the intensive care unit[5]. Meanwhile, it is approximately 4,1 million patients in the EU to acquire a HCAI each year. The number of deaths occurring as a direct consequence of these infections is estimated to be at least 37,000 and these infections are thought to contribute to an additional 110,000 deaths each year. Although the magnitude of HCAIs in many developing countries is not clearly understood, it has been estimated that it affects from 5% to 15% of hospitalized patients in regular wards[22] and more than half of patients admitted to ICUs[19].

The most frequent infections are urinary tract infections, followed by respiratory tract infections, infections after surgery, bloodstream infections, and others (including diarrhoea due to Clostridium difficile)[11]. The major causes of HCAIs are due to resistant or multiresistant bacteria such as Meticillin-resistant Staphylococcus aureus (MRSA), vancomycin-resistent enterococci, multidrug-resistant Mycobacterium tuberculosis, etc. Resistant bacteria emerge under the selective pressure of antibiotics and become a healthcare problem whenever they are able to spread and cause infections[14].

In worldwide, considerablee attention is focused on the prevention of the emergence and transmission of resistant bacteria. Approximately 20–30% of HCAIs are considered to be preventable by intensive hygiene and control programmes[21]. Steps can be taken to control and prevent HAIs in a variety of

settings; healthcare facilities, care teams, individual doctors and nurses.

Mathematical models are increasingly being used in epidemiology to understand the dynamics of the disease spread in a community[3, 17]. This will give insight health worker to make infection control policy decisions. Few mathematical models have been introduced to study nosocomial infection, see for example[1-2, 6, 8-9, 13-15, 18] and very few models on HCAI from low or middle-income countries have been published[10]. In this paper, we introduce and discuss several simple models of HCAI by considering self-cross infection and the presence of a control.

Mathematical Model

A population in a healthcare facility is simply divided into two sub population; patients and health workers. Futhermore, both sub population are divided into two groups of individuals; susceptible patients ($\boldsymbol{S_p}$), infected patients ($\boldsymbol{I_p}$), susceptible health workers ($\boldsymbol{S_w}$) and infected health workers ($\boldsymbol{I_w}$).

Assumption: To develop a mathematical model describing the spread of HCAIs in a hospital, we set the following assumptions: i. The total number of patients in hospital is assumed to be fixed at $\boldsymbol{N_p}$ for all time course. ii. Patients entering a healtcare unit or hospital are assumed to be free from HCAI. iii. Both susceptible and infected patients will leave hospital at rate $\boldsymbol{\mu}$ and all are replaced with susceptible patients. iv. Susceptible patients will get infected from both infected patients and infected health workers at rate $\boldsymbol{\beta_{pp}}$ and $\boldsymbol{\beta_{wp}}$, respectively. v. The number of health workers is also assumed to be constant at $\boldsymbol{N_w}$ for all time course. vi. Health workers during working hours in a health care unit will get infected and then spread the infection to patients at rate $\boldsymbol{\gamma_{pw}}$. vii. There is no infection from an infected health worker to a susceptible health worker. viii. The infected workers might prevent the disease transmission to patients, e.g., washing hand, etc., at rate $\boldsymbol{\phi}$. ix. Both susceptible and infected workers will leavea health care unit at rate $\boldsymbol{\sigma}$ and then replaced with susceptible workers.

Mechanism: The mechanism of HCAI spread is described as in the following scheme.

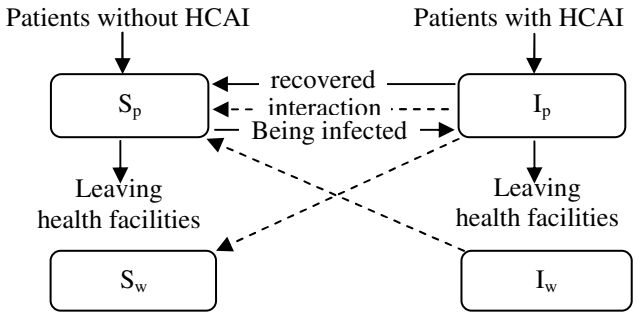

Figure-1: The mechanism of HCAI spread.

The system is govern by the following ODEs:

$$\begin{aligned}\frac{dS_p}{dt} &= \mu N_p - \beta_{pp} S_p I_p - \beta_{wp} S_p I_w - \mu S_p \\ \frac{dI_p}{dt} &= \beta_{pp} S_p I_p + \beta_{wp} S_p I_w - \mu I_p \\ \frac{dS_w}{dt} &= \sigma N_w - \gamma_{pw} S_w I_p + \phi I_w - \sigma S_w \\ \frac{dI_w}{dt} &= \gamma_{pw} S_w I_p - \phi I_w - \sigma I_w\end{aligned} \quad (1)$$

Since N_p and N_w are constant, then S_p and S_w can be calculated as $S_p = N_p - I_p$ and $S_w = N_w - I_w$. Thus, the system (1) reduces into.

$$\begin{aligned}\frac{dI_p}{dt} &= \beta_{pp}(N_p - I_p)I_p + \beta_{wp}(N_p - I_p)I_w - \mu I_p \\ \frac{dI_w}{dt} &= \gamma_{pw}(N_w - I_w)I_p - \phi I_w - \sigma I_w\end{aligned} \quad (2)$$

Model without control: Case 1.1. Cross Infection (Patient to Health worker Infection). In this case, we assume that no control $\phi = 0$, no patient to patient transmission $\beta_{pp} = 0$ but only worker to patient transmission $\beta_{wp} > 0$ and patient to worker transmission $\gamma_{pw} > 0$.

Case 1.2. Self-cross Infection (Patient to Patient and to Health workers): As in Case 1.1., we assume no control $\phi = 0$, however, there are worker to patient to worker as well as patient to patient transmission, i.e., $\beta_{pp},\ \beta_{wp}, \gamma_{pw} > 0$.

Model with Control: Case 2.1. Cross Infection (Patient to Health worker Infection): In this case a control is considered $\phi > 0$. In addition, we assume there is no patient to patient transmission $\beta_{pp} = 0$ but only patient to worker transmission, i.e., $\beta_{wp} > 0$ and worker to patient transmission, i.e. $\beta_{wp} > 0$, $\gamma_{pw} > 0$.

Case 2.2. Self-cross Infection (Patient to Patient and to Health workers): As in Case 2.1., there is a control $\phi > 0$. Moreover, there are patient to patient transmission $\beta_p > 0$, worker to patient transmission $\beta_{wp} > 0$ and patient to worker transmission $\gamma_{pw} > 0$.

Analysis

We analyze the dynamics of HCAI spread for Model I (Modal without control) and Model II (Modal with control) with the case 1.1-2.1 in the constant population.

Model I (without Control): Case 1.1. (Cross infection or patient-health workers)

For $\phi = 0,\ \beta_{pp} = 0$ and $\beta_{wp} > 0$ and $\gamma_{pw} > 0$, the system (2) has two equilibrium points; namely,

$E_1^*(I_p^*, I_w^*) = E_1^*(0,0)$, (3a)
- a disease-free equilibrium (DFE)
and
$E_2^*(I_p^*, I_w^*) = E_2^*\left(\frac{\beta_{wp}\gamma_{pw}N_pN_w-\mu\sigma}{\gamma_{pw}(\beta_{wp}N_w+\mu)}, \frac{\beta_{wp}\gamma_{pw}N_pN_w-\mu\sigma}{\beta_{wp}(\gamma_{pw}N_p+\sigma)}\right)$, (3b)
- an endemic equilibrium (EE).

Theorem-1: Suppose $R_{01} = \frac{\beta_{wp}\gamma_{pw}N_pN_w}{\mu\sigma}$.
i. If $R_{01} < 1$, the equilibrium E_1^* (3) is locally asymptotically stable (l.a.s). ii. Otherwise, if $R_{01} > 1$, the equilibrium E_1^* becomes unstable and the equilibrium E_2^* (4) is l.a.s.

Proof
The Jacobian matrix of (2) is given by
$$J = \begin{pmatrix} -\beta_{wp}I_w^* - \mu & \beta_{wp}(N_p - I_p^*) \\ \gamma_{pw}(N_w - I_w^*) & -\gamma_{pw}I_p^* - \sigma \end{pmatrix} \quad (4)$$

i. For DFE (3a), the Jacobian matrix (4) evaluated at this point reads
$$J_1 = \begin{pmatrix} -\mu & \beta_{wp}N_p \\ \gamma_{pw}N_w & -\sigma \end{pmatrix} \quad (5)$$

Characteristic equation of (6) is given by

$\Phi(\lambda) = a_0 + a_1\lambda + \lambda^2$

Where:
$a_0 = \mu\sigma - \beta_{wp}\gamma_{pw}N_pN_w$
$a_1 = \mu + \sigma$

Based on the Routh-Hourwitz criteria, the polinom $\Phi(\lambda)$ will have all negative real parts of its roots if $a_0, a_1 > 0$. Since $a_1 > 0$, it is enough to check the condition $a_0 > 0$. For $R_{01} < 1$, we get the following result

$$a_0 = \mu\sigma - \beta_{wp}\gamma_{pw}N_pN_w = \mu\sigma\left(1 - \frac{\beta_{wp}\gamma_{pw}N_pN_w}{\mu\sigma}\right) = \mu\sigma(1 - R_{01}) > 0$$

Thus, E_1^* is l.a.s if $R_{01} < 1$. Otherwise, if $R_{01} > 1$ then E_1^* is unstable.

ii. The Jacobian matrix (4) evaluated at E_2^* (3b) reads

$$J_2 = \begin{pmatrix} -\beta_{wp}\left(\frac{\Theta-\Lambda}{\Omega}\right) - \mu & \beta_{wp}\left(N_p - \frac{\Theta-\Lambda}{\Gamma}\right) \\ \gamma_{pw}\left(N_w - \frac{\Theta-\Lambda}{\Omega}\right) & -\gamma_{pw}\left(\frac{\Theta-\Lambda}{\Gamma}\right) - \sigma \end{pmatrix} \quad (6)$$

Where:
$\Theta = \beta_{wp}\gamma_{pw}N_pN_w$,
$\Lambda = \mu\sigma$,
$\Omega = \gamma_{pw}(\beta_{wp}N_w + \mu)$,
$\Gamma = \beta_{wp}(\gamma_{pw}N_p + \sigma)$

Characteristic equation (6) of matrix J_2 is
$\Phi(\lambda) = a_0 + a_1\lambda + a_2\lambda^2$

Where:
$a_0 = \Omega\Gamma(\Theta - \Lambda)$,
$a_1 = (\beta_{wp}\Gamma + \gamma_{pw}\Omega)(\Theta - \Lambda) + \Omega\Gamma(\mu + \sigma)$,
$a_2 = \Omega\Gamma$

Based on the Routh-Hourwitz criteria, the polinom $\Phi(\lambda)$ will have all negative real parts of its roots if $a_0, a_1, a_2 > 0$. We can see that $a_2 > 0$. Thus, we need to show that $a_0, a_1 > 0$. For $R_{01} > 1$, we get

$$a_0 = \Omega\Gamma(\Theta - \Lambda) = \Omega\Gamma\Lambda\left(\frac{\Theta}{\Lambda} - 1\right) = \Omega\Gamma\Lambda(R_{01} - 1) > 0$$
$$a_1 = (\beta_{wp}\Gamma + \gamma_{pw}\Omega)(\Theta - \Lambda) + \Omega\Gamma > (\beta_{wp}\Gamma + \gamma_{pw}\Omega)(\Theta - \Lambda) = (\beta_{wp}\Gamma + \gamma_{pw}\Omega)\Lambda(R_{01} - 1) > 0$$

Thus, E_2^* becomes l.a.s. if $R_{01} > 1$. This completes the proof of Theorem 1.

Case 2.2. (Self-cross infection or patient-patient-worker infection)

For $\phi = 0$, $\beta_{pp} > 0$, $\beta_{wp} > 0$ and $\gamma_{pw} > 0$, the system (2) has two equilibrium points; namely,

$E_3^*(I_p^*, I_w^*) = E_1^*(0,0)$, (7a)
- a disease-free equilibrium (DFE)

and

$E_4^*(I_p^*, I_w^*) = \left(\frac{(\beta_{wp}\gamma_{pw}N_w+\beta_{pp}\sigma)S_p^*-\mu\sigma}{\gamma_{pw}(\mu-\beta_{pp}S_p^*)}, \frac{(\beta_{wp}\gamma_{pw}N_w+\beta_{pp}\sigma)S_p^*-\mu\sigma}{\gamma_{pw}\beta_{wp}S_p^*}\right)$, (7b)
- an endemic equilibrium (EE)

with
$\frac{N_p}{R_{02}} < S_p^* < \frac{\mu}{\beta_{pp}}$

and S_p^* is the positive solution of the following equation
$a_2(S_p^*)^2 + a_1S_p^* + a_0 = 0$

Where:
$a_2 = \beta_{pp}\gamma_{pw} > 0$,
$a_1 = -\gamma_{pw}(\mu + \beta_{pp}N_p + \beta_{wp}N_w) - \beta_p\sigma < 0$,
$a_0 = \mu(\sigma + \gamma_{pw}N_p) > 0$

Theorem 2: Suppose $R_{02} = R_{01} + \frac{\beta_{pp}N_p}{\mu}$ with R_{01} as defined in Theorem 1. i. If $R_{02} < 1$, the equilibrium $E_3^*(0,0)$ of the system

(4) is l.a.s. ii. Otherwise, if $R_{02} > 1$ the equilibrium E_3^* becomes unstable and the equilibrium E_4^* is l.a.s.

Proof

The Jacobian matrix of (2) is given by

$$J = \begin{pmatrix} \beta_{pp}(N_p - 2I_p^*) - \beta_{wp}I_w^* - \mu & \beta_{wp}(N_p - I_p^*) \\ \gamma_{pw}(N_w - I_w^*) & -\gamma_{pw}I_p^* - \sigma \end{pmatrix} \quad (8)$$

i. For $E_3^*(0,0)$, the characteristic equation of (8) is given by

$\lambda^2 + a_1\lambda + a_0 = 0$

with

$a_1 = \mu + \sigma - \beta_{pp}N_p$

$a_0 = \mu\sigma - \beta_{wp}\gamma_{pw}N_wN_p - \beta_{pp}\sigma N_p$

For $R_{02} = R_{01} + \frac{\beta_{pp}N_p}{\mu} < 1$, we have $\beta_{wp}\gamma_{pw}N_wN_p + \beta_{pp}\sigma N_p < \mu\sigma$. Then, we obtain $a_1, a_0 > 0$ as follows

$$a_1 = \mu + \sigma - \beta_{pp}N_p = \frac{1}{\sigma}(\mu\sigma + \sigma^2 - \beta_{pp}\sigma N_p)$$
$$> \frac{1}{\sigma}(\beta_{wp}\gamma_{pw}N_wN_p + \beta_{pp}\sigma N_p + \sigma^2 - \beta_{pp}\sigma N_p)$$
$$= \frac{1}{\sigma}(\beta_{wp}\gamma_{pw}N_wN_p + \sigma^2) > 0$$
$$a_0 = \mu\sigma - \beta_{pw}\gamma_{wp}N_wN_p - \beta_{pp}\sigma N_p$$
$$= \mu\sigma(1 - R_{02}) > 0$$

Thus, based on the Routh-Hourwitz criteria, the polinom $\Phi(\lambda)$ has all egative real parts of its roots. The proof of (i) is completed.

ii. For $E_4^*(I_p^*, I_w^*)$ in (7b), the characteristic equation of (8) is given by

$\lambda^2 + a_1\lambda + a_0 = 0$

with

$$a_1 = (\gamma_{pw} + 2\beta_{pp})I_p^* + \beta_{wp}I_w^* + \mu - \beta_{pp}N_p$$
$$a_0 = (\beta_{wp}\gamma_{pw}N_w - \beta_{pp}\sigma)(N_p - I_p^*) + \beta_{pp}(2\gamma_{pw}I_p^* + \sigma)I_p^*$$
$$+\gamma_{pw}(\mu - \beta_{pp}N_p)I_p^* + \beta_{wp}(\gamma_{pw}N_w + \sigma)I_w^* + \mu\sigma$$

Next, we show that $a_1, a_0 > 0$. Let $N_p = S_p^* + I_p^*$. We have $\mu - \beta_{pp}S_p^* > 0$ for positive endemic equilibrium E_4^*. Then, we obtain

$$a_1 = (\gamma_{pw} + 2\beta_{pp})I_p^* + \beta_{wp}I_w^* + \mu - \beta_{pp}(S_p^* + I_p^*)$$
$$= (\gamma_{pw} + \beta_{pp})I_p^* + \beta_{wp}I_w^* + \mu - \beta_{pp}S_p^* > 0$$
$$a_0 = (\beta_{wp}\gamma_{pw}N_w - \beta_{pp}\sigma)S_p^* + \beta_p(2\gamma_{pw}I_p^* + \sigma)I_p^*$$
$$+\gamma_{pw}\left(\mu - \beta_{pp}(S_p^* + I_p^*)\right)I_p^* + \beta_{wp}(\gamma_{pw}N_w + \sigma)I_w^* + \mu\sigma$$
$$= \beta_{wp}\gamma_{pw}N_wS_p^* + \sigma(\mu - \beta_{pp}S_p^*) + \beta_{pp}(\gamma_{pw}I_p^* + \sigma)I_p^*$$
$$+\gamma_{pw}(\mu - \beta_{pp}S_p^*)I_p^* + \beta_{wp}(\gamma_{pw}N_w + \sigma)I_w^* > 0$$

Therefore, based on the Routh-Hourwitz criteria, the polinom $\Phi(\lambda)$ will have all egative real parts of its roots. This completes Theorem 2.

Case 2.1 (cross or patient-worker infection with control)

For $\phi > 0, \beta_{pp} = 0, \beta_{wp} > 0$ and $\gamma_{pw} > 0$, the system (2) has two equilibrium points; namely,

$E_5^*(I_p^*, I_w^*) = E_5^*(0,0)$, (9a)

- a disease-free equilibrium (DFE)

and

$E_6^*(I_p^*, I_w^*) = E_6^*\left(\frac{\Delta}{\gamma_{pw}(\beta_{wp}N_w + \mu)}, \frac{\Delta}{\beta_{wp}(\gamma_{pw}N_p + \sigma + \phi)}\right)$, (9b)

- an endemic equilibrium (EE).

Where: $\Delta = \beta_{wp}\gamma_{pw}N_pN_w - \mu(\sigma + \phi)$

Theorem-3: Let $R_{03} = \frac{\beta_{wp}\gamma_{pw}N_pN_w}{\mu(\sigma+\phi)}$.

i. If $R_{01} < 1$, the DFE (9a) is l.a.s. ii. Otherwise, if $R_{01} > 1$, the equilibrium E_5^* becomes unstable and the EE in (9b) is l.a.s.

Proof

The Jacobian matrix of (2) becomes

$$J = \begin{pmatrix} -\beta_{wp}I_w^* - \mu & \beta_{wp}(N_p - I_p^*) \\ \gamma_{pw}(N_w - I_w^*) & -\gamma_{pw}I_p^* - \sigma - \phi \end{pmatrix} \quad (10)$$

i. For DFE(9a), the Jacobian matrix (10) reads

$$J_1 = \begin{pmatrix} -\mu & \beta_{wp}N_p \\ \gamma_{pw}N_w & -\sigma + \phi \end{pmatrix} \quad (11)$$

Characteristic equation of (10) is given by

$\Phi(\lambda) = a_0 + a_1\lambda + \lambda^2$

Where:

$a_1 = \mu + \sigma + \phi$

$a_0 = \mu(\sigma + \phi) - \beta_{wp}\gamma_{pw}N_pN_w$

Based on the Routh-Hourwitz criteria, the polinom $\Phi(\lambda)$ will have all negative real parts of its roots if $a_0, a_1 > 0$. We can see $a_1 = \mu + \sigma + \phi > 0$. For $R_{03} < 1$, we obtain

$$a_0 = \mu(\sigma + \phi) - \beta_{wp}\gamma_{pw}N_pN_w = \frac{1 - R_{03}}{\mu(\sigma + \phi)} > 0$$

Thus, E_5^* is l.a.s. Otherwise, if $R_{03} > 1$ then $a_0 < 0$ and E_5^* becomes unstable.

ii. The Jacobian matrix (10) of E_6^* reads

$$J_2 = \begin{pmatrix} -\beta_{wp}\left(\frac{\Theta-\Lambda}{\Omega}\right) - \mu & \beta_{wp}\left(N_p - \frac{\Theta-\Lambda}{\Gamma}\right) \\ \gamma_{pw}\left(N_w - \frac{\Theta-\Lambda}{\Omega}\right) & -\gamma_{pw}\left(\frac{\Theta-\Lambda}{\Gamma}\right) - \sigma - \phi \end{pmatrix} \quad (12)$$

Where:

$\Theta = \beta_{wp}\gamma_{pw}N_pN_w$,

$\Lambda = \mu(\sigma + \phi)$,

$\Omega = \gamma_{pw}(\beta_{wp}N_w + \mu)$,

$\Gamma = \beta_{wp}(\gamma_{pw}N_p + \sigma + \phi)$

Characteristic equation of matrix $J_2$is
$\Phi(\lambda) = a_0 + a_1\lambda + a_2\lambda^2$
Where:
$a_2 = \Omega\Gamma$
$a_1 = \gamma_{pw}\Gamma\Lambda(\mathrm{R}_{03} - 1) + \beta_{wp}\Omega\Lambda(\mathrm{R}_{03} - 1)$
$\quad +\Omega\Gamma(\mu + \sigma + \phi)$
$a_0 = \Omega\Gamma\Lambda(\mathrm{R}_{03} - 1),$

Based on the Routh-Hourwitz criteria, the polinom $\Phi(\lambda)$ will have all negative real parts of its roots if $a_0, a_1, a_2 > 0$. We can see that $a_2 > 0$. If $R_{03} - 1 > 0$ then it results in $a_0, a_1 > 0$. Thus, E_6^* becomes l.a.s. if $R_{03} > 1$. This completes the proof of Theorem 3.

Case 2.2 (Self-cross or patient-patient-workers infection)

In this case,$\phi, \beta_{pp}, \beta_{wp} > 0$.The system (2) has two equilibrium points; namely,

$$E_7^*(I_p^*, I_w^*) = E_7^*(0,0), \quad (13a)$$
- a disease-free equilibrium (DFE)
and
$$E_8^*(I_p^*, I_w^*) = \left(\frac{\Psi}{\gamma_{pw}(\mu - \beta_{pp}S_p^*)}, \frac{\Psi}{\gamma_{pw}\beta_{wp}S_p^*}\right), \quad (13b)$$
- an endemic equilibrium (EE)

with
$\Psi = \big(\beta_{wp}\gamma_{pw}N_w + \beta_{pp}(\sigma + \phi)\big)S_p^* - \mu(\sigma + \phi).$

In addition, S_p^* satisfies
$$\frac{N_p}{R_{04}} < S_p^* < \frac{\mu}{\beta_p}$$

where: S_p^* is the positive solution of the following equation
$a_2\big(S_p^*\big)^2 + a_1S_p^* + a_0 = 0$

with
$a_2 = \beta_{pp}\gamma_{pw} > 0,$
$a_1 = -\Big(\mu\gamma_{pw} + \beta_{wp}\gamma_{pw}N_w + \beta_{pp}\gamma_{pw}N_p + \beta_{pp}(\sigma + \phi)\Big)$
$\quad < 0,$
$a_0 = \mu\big(\sigma + \phi + \gamma_{pw}N_p\big) > 0$

Theorem-4: *Let*$R_{04} = R_{03} + \frac{\beta_{pp}N_p}{\mu}$.
i. If $R_{04} < 1$, the DFEE$_7^*$ is *l.a.s.* ii. Otherwise, if $R_{04} > 1$, the DFEE$_7^*$ becomes unstable and the EEE$_8^*$ is l.a.s.

Proof
The Jacobian matrix of (2) is given by
$$J = \begin{pmatrix} -\beta_{wp}I_p^* + \beta_{pp}\big(N_p - I_p^*\big) - I_w^*\beta_{wp} - \mu & \beta_{wp}\big(N_p - I_p^*\big) \\ \gamma_{pw}(N_w - I_w^*) & -\gamma_{pw}I_p^* - \sigma - \phi \end{pmatrix} \quad (14)$$

i. The Jacobian matrix (14) evaluated at (13a) reads

$$J_1 = \begin{pmatrix} \beta_{pp}N_p - \mu & \beta_{wp}N_p \\ \gamma_{pw}N_w & -\sigma - \phi \end{pmatrix} \quad (15)$$

Characteristic equation of (8) is given by
$\Phi(\lambda) = a_0 + a_1\lambda + \lambda^2$
where
$a_1 = \mu + \sigma + \phi - \beta_{pp}N_p$
$a_0 = (\sigma + \phi)(\mu - \beta_{pp}N_p) - \beta_{wp}\gamma_{pw}N_pN_w$

Based on the Routh-Hourwitz criteria, the polinom $\Phi(\lambda)$ will have all negative real parts of its roots if$a_0, a_1 > 0$.

For $R_{04} < 1$, we obtain the following inequality
$$0 < \frac{\beta_{pp}N_p}{\mu + \sigma + \phi} < \frac{\beta_{pp}N_p}{\mu} < R_{03} + \frac{\beta_{pp}N_p}{\mu} = R_{04} < 1$$

Thus,
$a_1 = \mu + \sigma + \phi - \beta_{pp}N_p$
$\quad = (\mu + \sigma + \phi)\left(1 - \frac{\beta_{pp}N_p}{\mu + \sigma + \phi}\right) > 0$
$a_0 = (\sigma + \phi)\big(\mu - \beta_{pp}N_p\big) - \beta_{wp}\gamma_{pw}N_pN_w$
$\quad = \mu(\sigma + \phi)\left(1 - \frac{\beta_{pp}N_p}{\mu} - \frac{\beta_{wp}\gamma_{pw}N_pN_w}{\mu(\sigma + \phi)}\right)$
$\quad = \mu(\sigma + \phi)(1 - R_{04}) > 0$

Therefore, E_1^* is l.a.s. Otherwise, if $R_{03} > 1$ then $a_0 < 0$ and E_1^* become sunstable.

ii. The Jacobian matrix (14) evaluated at E_8^* (13b) is obtained
$$J = \begin{pmatrix} \beta_{pp}(N_p - 2I_p^*) - \beta_{wp}I_w^* - \mu & \beta_{wp}\big(N_p - I_p^*\big) \\ \gamma_{pw}(N_w - I_w^*) & -\gamma_{pw}I_p^* - \phi - \sigma \end{pmatrix} \quad (16)$$

The characteristic equation is given by
$\lambda^2 + a_1\lambda + a_0 = 0$

with
$a_1 = \big(\gamma_{pw} + 2\beta_p\big)I_p^* + \beta_{wp}I_w^* + \sigma + \phi + \mu - \beta_{pp}N_p$
$a_0 = -\gamma_{pw}\beta_{wp}N_wN_p + \gamma_{pw}\beta_{wp}N_pI_w^* + \gamma_{pw}\beta_{wp}N_wI_p^*$
$\quad +2\gamma_{pw}\beta_{pp}I_p^{*2} + \gamma_{pw}\mu I_p^* + 2\beta_{pp}I_p^*(\phi + \sigma)$
$\quad +(\gamma_{pw}I_p^* + \sigma + \phi)\big(\mu - \beta_{pp}N_p\big)$

we have
$a_1 = \big(\gamma_{pw} + 2\beta_p\big)I_p^* + \beta_{wp}I_w^* + \sigma + \phi + \mu - \beta_{pp}N_p$
$\quad = \big(\gamma_{pw} + \beta_p\big)I_p^* + \beta_{wp}I_w^* + \sigma + \phi + \mu - \beta_{pp}S_p^* > 0$
$a_0 = \gamma_{pw}\beta_{wp}N_pI_w^* - \gamma_{pw}\beta_{wp}N_wS_p^* + 2\gamma_{pw}\beta_{pp}I_p^{*2} + \gamma_{pw}\mu I_p^*$
$\quad +2\beta_{pp}I_p^*(\phi + \sigma) + (\gamma_{pw}I_p^* + \sigma + \phi)\big(\mu - \beta_{pp}N_p\big) > 0$

Therefore, based on the Routh-Hourwitz criteria, the polinom $\Phi(\lambda)$ will have all egative real parts of its roots. This completes the theorem.

Numerical Simulation

In this section, we discuss the numerical simulation of the reduced model (2), by using Runge-Kutta order four scheme. The parameters and their values are presented in the following table.

Table-1: Parameter values used in the numerical simulation of the model, total population and initial values.

Notation	Parameter description	Value	Ref
β_{pp}	Transmission $I_p \to S_p$	0.0 - 0.1	*)
β_{wp}	Transmission $I_w \to S_p$	0.0 - 0.1	*)
γ_{pw}	Transmission $I_p \to I_w$	0.001	*)
μ	Removal rate of S_p&I_p	0.02	*)
σ	Removal rate of S_w&I_w	0.05	*)
ϕ	Control rate of I_w	0 - 0.1	*)
N_p	Total number of patients	300	*)
N_w	Total number of workers	50	*)

*) assumed.

Figure 1 represents the population dynamics of Model1 for case 1.1. In the simulation, there are initially ten infected patients but no infected workers. The number of susceptible patients and workers is set to be $N_p = 300$ and $N_w = 50$, respectively. Model parameters are fixed for natural death of susceptible and infective at $\mu = 0.02; \sigma = 0.05$, respectively, no self infection ($\beta_{pp} = 0$), infection rate of susceptible worker from infected patients $\gamma_{pw} = 0.001$ and let the infection rate from infected workers to susceptible patients be varied $\beta_{wp} = 4e-5; 7e-5; 8e-5$.

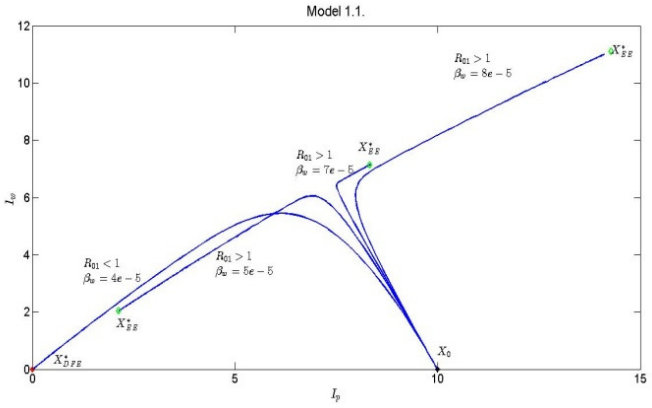

Figure-2: Simulation for several rates of cross infection; $\beta_{wp} = 4e-5$ ($R_{01} < 1$) and $\beta_{wp} = 5e-5; 7e-5; 8e-5$ ($R_{01} > 1$).

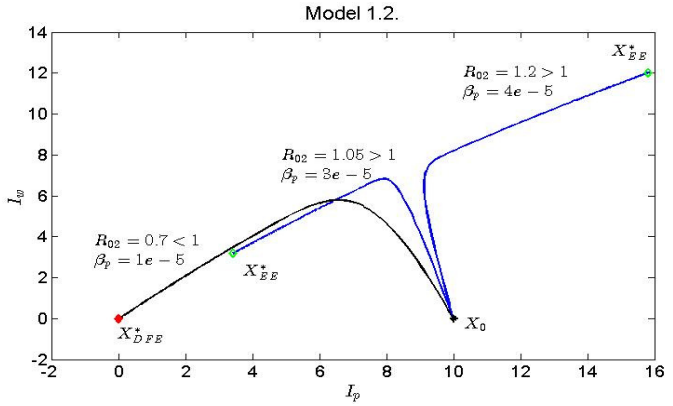

Figure-3: Simulation for several rates of cross infection at fixed $\beta_{wp} = 4e-5$ and varying β_{pp}.

As seen in Figure 2, when the parameters result in R_{01} below one ($R_{01} < 1$ for $\beta_{wp} = 4e-5$), the number of infectives both patients and workers tend to disease free equilibrium. Meanwhile, if R_{01} is above one ($R_{01} > 1$ for $\beta_w = 5e-5; 7e-5; 8e-5$), the dynamics of model 1 case 1.1 approach endemic equilibrium.

When we consider the self infection rate $\beta_{wp} > 0$ the dynamics of model 1 case 1.2 becomes depending on the parameter β_{wp} as well.

As shown in Figure-3, the dynamics of the infectives is determined by the basic reproductive number R_{02}. If $R_{02} < 1$ the number of both infectives (patients and health workers) is died out. Otherwise, the number approaches the endemic state as R_{02} is above one.

Figure-4 represents the population dynamics of case 2.1. The dynamics of model 2 case 2.1 is mostly determined by the reproductive number R_{03}.

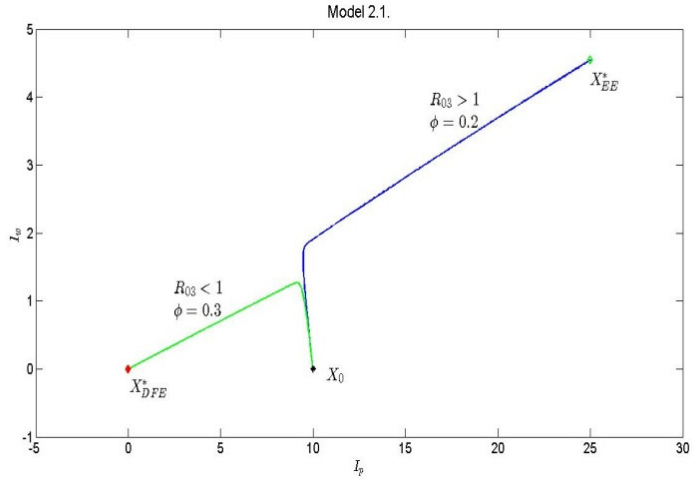

Figure-4: Dynamic behavior of model 2.1 as the reproduction number R_{03} below and above one when no self infection but the control rate is varied from $\phi = 0.2$ to $\phi = 0.3$.

In the numerical experiment, one can see that R_{04} becomes a threshold for the model 2 case 2.2. R_{04} is the basic reproductive number. As depicted in Figure 5, if this number is below one the dynamics approaches disease free equilibrium (DFE) and if it is above one the dynamics goes to the positive endemic state (EE).

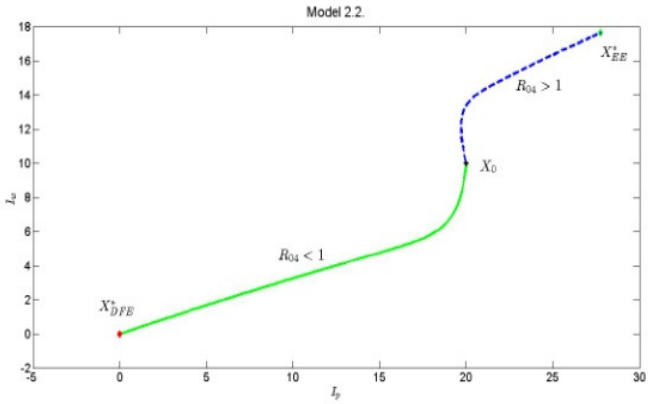

Figure-5: Simulation for two different control rates; $\phi = 0.001$ ($R_{04} > 1$) dashed line and $\phi = 0.1$ ($R_{04} < 1$) bold line.

Conclusion

We have developed several models describing the dynamics of HCAI by considering self and cross infection both with and without control. We also derive the thresholds that determine the behaviors of the dynamics for each model. If the threshold is below one, the disease dies out and as the threshold is above one it approaches to endemic phase. In the numerical simulation, the dynamics of the number of infectives becomes significant as several model parameters are increased to be above threshold. In future, it is interesting to consider units in a healthcare facility so that we can evaluate the source of infection based on units. One can also consider the length of stay in health facility for those infected patient.

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