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Studying the Epidemiological Model for the Infection by Spiral Gastric Bacteria

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Abstract

Helicobacter pylori (H. pylori), a bacterium that has captured the attention of researchers for over a century, is well-known for its association with peptic ulcer and gastric cancer. Despite a decline in its prevalence to improved hygienic conditions and effective curative and preventive measures, *H. pylori* still persists in various communities and continues to spread, maintaining its global presence in both developing and developed nations. Persistent efforts in scientific research have led to the discovery of diverse management options to combat this pathogen. In this article, we direct our attention to the global prevalence of *H. pylori* infection, delve into the factors contributing to its transmission, and explore prevention methods. Through the study's findings, we gain valuable insights into how these factors influence *H. pylori* infection rates. In addition, we study the local and global stability and the numerical solution for the models.

Key words *H. pylori* infection, SEIR model, local stability, global stability.
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1 Introduction

Helicobacter pylori, a bacterium discovered nearly four decades ago, remains a significant global health concern. It poses substantial risks to human well-being, leading to considerable morbidity and mortality attributed to peptic ulcer and gastric cancer. Regrettably, *Helicobacter pylori* infections often persist for a person's lifetime unless addressed with antibiotics or, in some fortunate cases, eradicated naturally through the body's immune response [1,2]. Managing this infection continues to be a crucial challenge in safeguarding public health and preventing severe gastrointestinal complications. Numerous epidemiological studies provided compelling evidence that first line *Helicobacter pylori* transmission directly through person-to-person contact. The most plausible mode of transmission involves the ingestion of the bacterium through the gastro-oral or oral-oral routes. Additionally, fecal-oral transmission is a recognized pathway, and there is also a possibility of transmission through water and food contaminated with the pathogenic organism [3,4]. Despite approximately half of the world's population being estimated to be infected with *Helicobacter pylori* the prevalence of the infection shows widespread variation both among and within countries. Even within a single city, variations in prevalence can be observed among different subgroups within the population. To mitigate the risk of infection, practicing good hygiene is crucial. Simple measures like washing hands thoroughly with soap and water after using the bathroom and before eating can significantly reduce the chances of transmission. It is also important to consume food that has been well-washed and properly cooked, while ensuring that drinking water is sourced from clean and safe supplies. By following these hygiene practices, individuals can take proactive steps to help prevent *H. pylori* infection and maintain better overall gastrointestinal health [5,6]. Given the significant impact of *H. pylori* on the development of diseases and health issues in Iraq, as well as in other regions globally, there is a pressing need for further studies in this area. Particular emphasis should be placed on research that explores its pathogenicity and transmission patterns. Understanding these aspects of the bacterium's behavior is crucial in addressing and managing its effects on human health effectively. Such research efforts can lead to better prevention strategies and improved healthcare approaches to combat *H. pylori*-related conditions [7].

2 The model and the existence of equilibrium

Consider the functions of time t, $S(t), E(t), I(t)$ and $R(t)$, where $S(t)$ represents a susceptible person, $E(t)$ an exposed person, $I(t)$ an infected person and $R(t)$ a recovered person [8].

$$\begin{aligned} S'(t) &= \pi - \alpha\rho(1-\delta)S(t)I(t) - \varepsilon S(t) \\ E'(t) &= \alpha\rho(1-\delta)S(t)I(t) - (\tau+\varepsilon)E(t) \\ I'(t) &= \tau E(t) - (\theta+\gamma+\varepsilon)I(t) \\ R'(t) &= \theta I(t) - \varepsilon R(t) \end{aligned} \tag{2.1}$$

where π is the constant recruitment of the host, α is the contact rate with pathogen either direct or indirect way, $\delta \in (0,1)$ is the commitment of susceptible person with healthy rule, ρ is the direct contact between the susceptible host with the infected host, τ represents the transition rate from E class to I class, θ is the recovered person due to treatment, γ is the disease death rate, ε is the natural death rate of the host.
As the initial three equations in (2.1) are not reliant on the variable R, we can simplify the analysis by focusing on the following reduced model:

$$\begin{aligned} S' &= \pi - \alpha\rho(1-\delta)SI - \varepsilon S \\ E' &= \alpha\rho(1-\delta)SI - (\tau+\varepsilon)E \\ I' &= \tau E - (\theta+\gamma+\varepsilon)I \end{aligned} \tag{2.2}$$

It follows from system (2.2) that [9]:
$(S' + E' + I') = \pi - \varepsilon(S+E+I) - (\theta+\gamma)I \leq \pi - \varepsilon(S+E+I)$.

Then $\lim sup_{t\to\infty}(S+E+I) \leq \frac{\pi}{\varepsilon}$. Hence, the feasible region for system (2.2) is: $\psi = \{\omega = (S,E,I) : S+E+I \leq \frac{\pi}{\varepsilon}, S \geq 0, E \geq 0, I \geq 0\}$

Verifying the positive invariance of region ψ with respect to system (2.2). Let $R_0 = \frac{\pi\rho\alpha(1-\delta)\tau}{\varepsilon(\varepsilon+\tau)(\theta+\gamma+\varepsilon)}$. Having established a disease-free equilibrium $\omega_0 = (\frac{\tau}{\varepsilon}, 0, 0)$ within the system (2.2), our attention turns to determine the conditions that give rise to an endemic equilibrium. This endemic equilibrium must necessarily satisfy:

$$\begin{aligned} &\pi - \alpha\rho(1-\delta)SI - \varepsilon S = 0 \\ &\alpha\rho(1-\delta)SI - (\tau+\varepsilon)E = 0 \\ &\tau E - (\theta+\gamma+\varepsilon)I = 0 \end{aligned}$$

By some simple calculation, we have $S^\star = \frac{(\varepsilon+\tau)(\theta+\gamma+\varepsilon)}{\rho\alpha(1-\delta)\tau}$,$E^\star = \frac{(\theta+\gamma+\varepsilon)\varepsilon}{\tau\rho\alpha(1-\delta)}(R_0-1)$ and $I^\star = \frac{\varepsilon}{\rho\alpha(1-\delta)}(R_0-1)$. The system (2.2) exhibits a solitary disease-free

equilibrium (ω_0). However, when $R_0 > 1$, the system (2.2) further presents a distinctive endemic equilibrium denoted as $\omega^\star = (S^\star, E^\star, I^\star)$.

3 The local stability analysis of equilibrium

We will assess the stability of the equilibrium by analyzing the eigenvalues of the Jacobian matrices associated with system (2.2).

Theorem 3.1. *If $R_0 < 1$, then the (ω_0) is locally asymptotically stable.*

Proof.

The Jacobian of system (2.2) at ω_0 is:

$$J(\omega_0) = \begin{bmatrix} -\varepsilon & 0 & -\frac{\alpha\rho(1-\delta)\pi}{\varepsilon} \\ 0 & -(\tau+\varepsilon) & \frac{\alpha\rho(1-\delta)\pi}{\varepsilon} \\ 0 & \tau & -(\theta+\gamma+\varepsilon) \end{bmatrix}$$

The characteristic equation of system (2.2) at ω_0 takes the following form: $(\varepsilon+\lambda)[(\tau+\varepsilon+\lambda)(\theta+\gamma+\varepsilon+\lambda) - \alpha\rho(1-\delta)\gamma(\frac{\pi}{\varepsilon})] = 0$, If $R_0 < 1$, then the characteristic equation has negative roots, leading to the (ω_0) being locally asymptotically stable. Conversely, when $R_0 > 1$, the characteristic equation has at least one positive root, indicating that the disease-free equilibrium ω_0 becomes unstable.

Theorem 3.2. *If $R_0 > 1$, then the ($\omega^\star$) is locally asymptotically stable .*

Proof.
The Jacobian of system (2.2) at $\omega^\star$ is

$$J(\omega^\star) = \begin{bmatrix} -(\alpha\rho(1-\delta)I^\star+\varepsilon) & 0 & -\alpha\rho(1-\delta)S^\star \\ \alpha\rho(1-\delta)I^\star & -(\tau+\varepsilon) & \alpha\rho(1-\delta)S^\star \\ 0 & \tau & -(\theta+\gamma+\varepsilon) \end{bmatrix}$$

The characteristic equation of system (2.2) at ω and simplification of the following form:
$(\alpha\rho(1-\delta)I^\star+\varepsilon+\lambda)(-\alpha\rho\tau(1-\delta)S^\star + (\theta+\gamma+\varepsilon+\lambda)(\tau+\varepsilon+\lambda)) + \alpha^2\rho^2\tau(1-\delta)^2S^\star I^\star = 0$, yield the following polynomial characteristic equation: $(\alpha\rho(1-\delta)I^\star+\varepsilon+\lambda)(\theta+\gamma+\varepsilon+\lambda)(\tau+\varepsilon+\lambda) = \alpha\rho(1-\delta)\tau S^\star(\varepsilon+\lambda)$

Let the equation have a solution with a non-negative real part:
$\frac{|\alpha\rho(1-\delta)I^\star+\varepsilon+\lambda||\theta+\gamma+\varepsilon+\lambda||\tau+\varepsilon+\lambda|}{|\varepsilon+\lambda|} = \alpha\rho(1-\delta)\tau S^\star$, recall the value of $S^\star$.

Hence, $\alpha\rho(1-\delta)\tau S^\star = (\theta+\gamma+\varepsilon+\lambda)(\tau+\varepsilon+\lambda)$. We have $\frac{|\alpha\rho(1-\delta)I^\star+\varepsilon+\lambda|}{|\varepsilon+\lambda|} > 1$, for all values of λ, as long as $Re(\lambda) \geq 0$. Then $\mid \theta+\gamma+\varepsilon+\lambda \mid\mid \tau+\varepsilon+\lambda \geq \mid \theta+\gamma+\varepsilon+x \mid\mid \tau+\varepsilon+x \mid \geq (\theta+\gamma+\varepsilon)(\tau+\varepsilon) = \alpha\rho(1-\delta\tau S^\star)$

This implies that for any λ with $Re(\lambda) \geq 0$, the left hand side of the equation is consistently greater than the right-hand side, rendering it impossible for the characteristic equation to possess such a solution.

4 The global stability analysis of equilibrium.

In this section, we delve into the global stability analysis of both the ω_0 and $\omega^\star$ steady states. We begin by investigating the global stability of the ω_0.

Theorem 4.1. *Assuming $R_0 < 1$, (ω_0) is globally asymptotically stable.*

Proof.
Consider the following positive definite function:
$x_1 = \beta_1(S - S_0 - S_0 \ln \frac{S}{S_0}) + \beta_2 E + \beta_3 I$, where $\beta_1 > 0, \beta_2 = \frac{1}{\tau+\varepsilon}, \beta_3 = \frac{1}{\tau}$.
Evidently, $x_1 : R^3 =\rightarrow R$ is a continuously differentiable function satisfying $x_1(\omega_0) = 0$, and $x_1(\omega) > 0$, for all $\omega \neq \omega_0$. Furthermore, we have:
$x_1' = \frac{S-S_0}{S} S' + E' + I'$.
Simplifying this equation we get
$x_1' = -\pi(\frac{\pi}{\varepsilon S} + \frac{\varepsilon S}{\pi} - 2) + \frac{\theta+\gamma+\varepsilon}{\tau}(R_0 - 1) = -\varepsilon S(\frac{\pi}{\varepsilon S} - 1)^2 + \frac{(\theta+\gamma+\varepsilon)}{\tau}(R_0 - 1)$,
Since $R_0 < 1$, the last term is non-positive, Hence we have, $x_1' < 0$, for all $\omega \neq \omega_0$. Therefore, by Lyapunov's theorem [10], we can conclude that the disease-free equilibrium is globally asymptotically stable.

Theorem 4.2. *Assuming $R_0 > 1$, $\omega^\star$ is globally asymptotically stable.*

Proof.
Consider the following positive definite function:
$x_1 = (S - S^\star \ln \frac{S}{S^\star} - S^\star) + (E - E^\star \ln \frac{E}{E^\star} - E^\star) + \frac{\varepsilon+\tau}{\tau}(I - I^\star \ln \frac{I}{I^\star} - I^\star)$.

Clearly , $x_1 : R_+^3 \rightarrow R$ is a continuously differentiable function such that $x_1(\omega^\star) = 0$, and $x_1(\omega) = 0$ for all $\omega \neq \omega^\star$. Furthermore,
$x_1' = (\frac{S-S^\star}{S})S' + (\frac{E-E^\star}{E})E' + \frac{\varepsilon+\tau}{\tau}(\frac{I-I^\star}{I})I'$
$x_1' = -\varepsilon\frac{(S-S^\star)^2}{S} + \alpha\rho(1-\delta)S^\star I^\star[3 - \frac{S^\star}{S} - \frac{SIE^\star}{S^\star I^\star E} - \frac{EI^\star}{IE^\star}]$, such that $\frac{S^\star}{S} + \frac{SIE^\star}{S^\star I^\star E} + \frac{EI^\star}{IE^\star} > 3$

Since $R_0 > 1$, the last term is non-positive, Hence $x_1' < 0$ for all $\omega \neq \omega^\star$. Therefore, according to Lyapunov's theorem [10], the endemic equilibrium is globally asymptotically stable.

5 Discussion

θ: transmission of the infection depending on θ when a decrease leads to an increase in spreading of disease and vice versa, as shown in figure (1-A) when θ was high 0.15 the recovery rate was high, while when θ rate was low 0.05, we noticed a reduction in the recovery rate and when recorded the lowest θ rate 0.01 with lowest recover rate. δ: from figure(1-B) we can see that when δ rate was high (0.9) disease rate transmission was low but when δ rate was low (0.6) disease rate transmission increase. ρ: as figure(1-C) showed that when ρ rate increase disease rate increase. When $\rho = 0.25$ disease transmission rate was high and when $\rho = 0.1$ was disease rate was decrease. α: when α increase infection rate increase and α decrease the infection rate decrease too. As shown in figure (1-D) with lowest rate of $\alpha = 0.05$ recorded low disease rate and when α was high (0.2) disease rate was high. All these considered parameters are important parameters as infection with *H. pylori* depends on them. Therefore, commitment to treatment will help in reduction of disease as well as affecting on θ value (increase). In addition, commitment with health rules and hygiene is the most important thing that affecting δ value (increase). Direct contact between healthy and infected persons result in an increase of ρ value as well as an increased disease rate; that is, each person responsible for spreading of disease just like the infected mother transmission infection to her family and doctors to their community. Direct or indirect exposure to bacterial pathogen through contaminated food and water and contaminated surface with bacteria these lead to an increased α value as well as an increased disease rate.

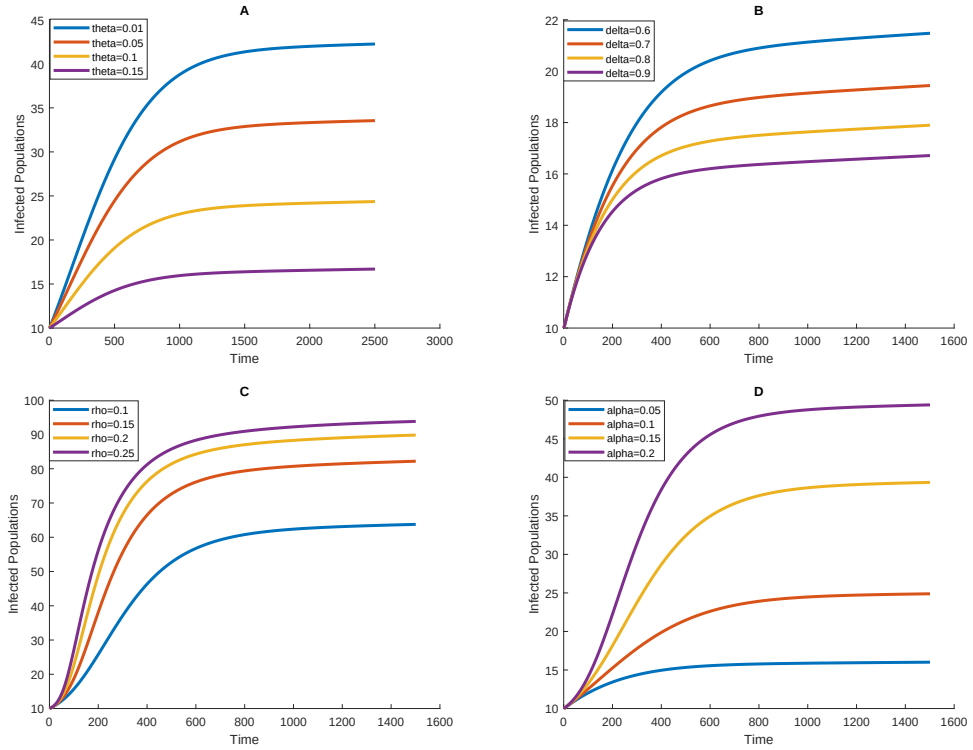

Figure 1: (A) represents the recovered person due to treatment, (B)represents the commitment of susceptible person with healthy rule, (C) represents the direct contact between the susceptible host with the infected host, (D) represents the contact rate with pathogen either direct or indirect way.

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