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Dynamical Analysis of Peptic Ulcer Disease Model in Nigeria with the Effect of Vaccination and Treatment Program

Abdu Ibrahim; Ibrahim Danbaba ; Ibrahim Bello; Bababangida Alkali; and Kabiru Zailani

School of Secondary Education (Science), Aminu Saleh College of Education, Azare, Bauchi State, Nigeria.

Corresponding author: danbabaibrahim145@gmail.com

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Abstract

The study developed a non-linear deterministic mathematical model to investigate the transmission dynamics of peptic ulcer disease in human population by considering both direct and indirect contact transmission with vaccine and treatment as control. In developing the model, the population was compartmentalized into susceptible-vaccinated-exposed-infected-treated-helicobacter pylori concentration in the environment-recovered. The model developed is a system of differential equations. The rate of change of the system, existence and uniqueness of solution, region of absolute stability and positivity of the solution was established. Existence of disease-free equilibrium state and basic reproduction number was also established. Mathematical analysis was determined by the basic reproduction number R_0, if $R_0 < 1$, the disease-free equilibrium is locally asymptotically stable whereas if $R_0 > 1,$ then the equilibrium is unstable whereas the numerical analysis for the estimated basic reproduction number of the model is greater than unity in perspective of Nigeria, for which the Herd Immunity Threshold indicated that vaccinating 99.47% of Nigeria population can control spreading of Peptic ulcer disease in the country. The paper recommended amongst other things that, if significant changes concerning the issue of threshold target are observed to improve the rate at which peptic ulcer can be minimized from the population where Government and non-governmental organization should encourage the use of peptic ulcer prescribe drugs and vaccine among individuals.

Keywords: Positivity, Existence and Uniqueness, Absolute Stability, Disease Free Equilibrium, Basic Reproduction Number, Herd Immunity Threshold.

Introduction

Peptic Ulcer Disease (PUD) is a disruption of the mucosal integrity of the stomach and/or duodenum leading to a local defect or excavation due to active inflammation (Educate innovate research and development, 2016) Peptic ulcer Diseases (PUDs) are painful sores that can be found on the inner lining of the stomach, the esophagus or on the upper part of the small intestine, it is possible to have the 2 types of ulcers at the same time (Obafemi Awolowo University Medical Students Association, 2018). Normally, the lining of the stomach is usually protected from digestive juices by a thick layer of mucus. However, Peptic ulcer (PU) disease has unclear symptoms which is usually do not appear until

adulthood and observable symptoms may never develop but major symptoms such as abdominal pain, heartburn and nausea have been observed 3 – 4 days after ingestion of the bacteria (Public Health Agency of Canada, 2011). Peptic ulcer is more common in men than women, with lifetime prevalence for men estimated at 11–20% while for women at 8–11% and the ratio of the relative frequency of duodenal and gastric ulcers is typical in most of the developed world (Whittle, Edinburgh and Gilmore, 2008). Recombinant urease (rUrease) and parenteral vaccine containing *H. pylori* antigens such as CagA, VacA, and NAP in combination with aluminum hydroxide as an adjuvant have been found to be effective vaccines against *H. pylori.* In a research of Kabir (2007), Peptic ulcer disease has various causes but helicobacter pylori and non-steroidal anti-inflammatory drugs (NSAID) account for the majority of the disease etiology (Narayanan, Reddy and Marsicano, 2018). Peptic ulcer disease is the most common ulcer of the area of gastrointestinal tract that is usually acidic and extremely painful. It is considered as mucosal erosions equal to or greater than 0.5 cm, as many as 70% - 90% of Peptic ulcer is associated with Helicobacter pylori with a spiral-shaped bacterium that lives in the acidic environment of the stomach (Prof. (Dr.) Md. Zulfikar Ali Chief Editor, 2013). The Clinical characteristics of H. pylori infection rate indicate that half of 49% of dyspeptic patients had active H. pylori infection. Two-thirds of 71.1% of PUD patients had active H. pylori infection where the majority of 85% of H. pylori infections among PUD cases had duodenal ulcer (Assefa, Abay, Abebe, Tesfaye, Tadesse, Lakew, 2021). *Helicobacter Pylori* is commonly transmitted directly from infected to a susceptible person through oral-to-oral route or faecal to oral route which includes kissing, vomiting, oral sex, breastfeeding and the human pathogen can be transmitted by environmental factors such as using contaminated water and contaminated food (Dore and Graham, 2022). A prolonged incubation period of up to 19 days allowed successful isolation of *H pylori* from a patient who received triple therapy that failed to eradicate the bacterium (Yin, He and Zhang, 2009).

However, in 2022, the population of Nigeria was 218,541,212 with a death rate of 12.4 per 1000 people (Knoema, 2023). According to the latest World Health Organization data published in 2020, the Peptic ulcer disease deaths in Nigeria reached 5 846 which equivalents of 0.39% of the total deaths and make it to be ranks as 31st country in the world (World Health Organization, 2020). The most common causes of peptic ulcer disease are Helicobacter pylori infection, Non-steroidal Anti-Inflammatory Drugs **(**NSAIDs), Medication, Zollinger- Ellison syndrome, Malignancy, Stress, Viral infection, Vascular insufficiency, Radiation therapy, Crohn's disease and Chemotherapy (National library of Medicine and National Center for Biotechnology information, 2023). To Management the Peptic ulcer disease avoid Acid suppression, Smoking cessation, Alcohol drunk, Stress reduction and NSAIDs usage, if Peptic ulcer diseases is not diagnosed and treated promptly can lead to Peritonitis, Bowel obstruction, Bleeding, Gastric cancer, Perforation and Penetration as well as a serious complication (Educate Innovate Research and Development, 2016).

Statement of the Problem

Peptic ulcer disease (PUD) is a prevalent and significant health issue affecting a considerable portion of the global population including Nigeria. Despite medical advancements and the availability of effective treatments, peptic ulcer disease continues to pose a substantial burden on individuals, healthcare systems, and society as a whole. According Daily Trues (2023), Guruma A Guruma, a fellow doctor in pharmaceutical microbiology and biotechnology at the faculty of pharmacy Gombe state university said Helicobacter pylori causes a range of gastrointestinal diseases such as chronic gastric, peptic ulcer diseases and gastric cancer. This study therefore, intends to develop the model on peptic ulcer transmission dynamic in human population with vaccine and treatment as control strategy.

Aim and Objectives of the Study

The aim of this study is to develop a mathematical model for peptic ulcer disease transmission dynamic and control. This understanding can be gained by a proper statistical analysis, involving the use of mathematical models of the peptic ulcer which possesses the under listed objectives;

i. To obtain the rate of change of the population of the model
ii. To explore the basic properties of the model such as positivity and invariant region of the model solution
iii. To check the existence and uniqueness of the model solution
iv. To obtain the disease-free equilibrium point of the model
v. To computer the basic reproduction number of the
vi. Carry out numerical analysis of the model using MATLAB 2015a Software package
vii. To make recommendation

Materials and Methods

Model Formulation and Its Basic Properties

A formulated mathematical model is compartmentalized the population into eight classes, seven compartments for human population and the remaining one is for Helicobacter pylori concentration in the environment. The human population is consist of Susceptible human, denoted as $S_H(t)$, Vaccinated represented as $V_H(t)$, Exposed expressed as $E_H(t)$, Infected represented by $I_H(t)$, Treated represented by $T(t)$, Peptic ulcer represented by $P(t)$, these are individuals who have been infected with peptic ulcer, Treated human represented by $T(t)$, Helicobacter pylori expressed by $H(t)$ and recovered denoted by $R(t)$. The forces of infection of the model are $\beta_1 S_H I_H + (1-\theta)\beta_2 H_E$ where β_1 is the ingestion rate of *H. pylori* through human to human interaction and β_2 is the *H. pylori* ingestion rate through environment to human, $(1-\theta)$ is a value that is less than unity which accounts for

infectiousness among individuals and θ is the proportion of humans use of hygiene, safe water or sanitation to avoid being contaminated with infection causing bacteria.

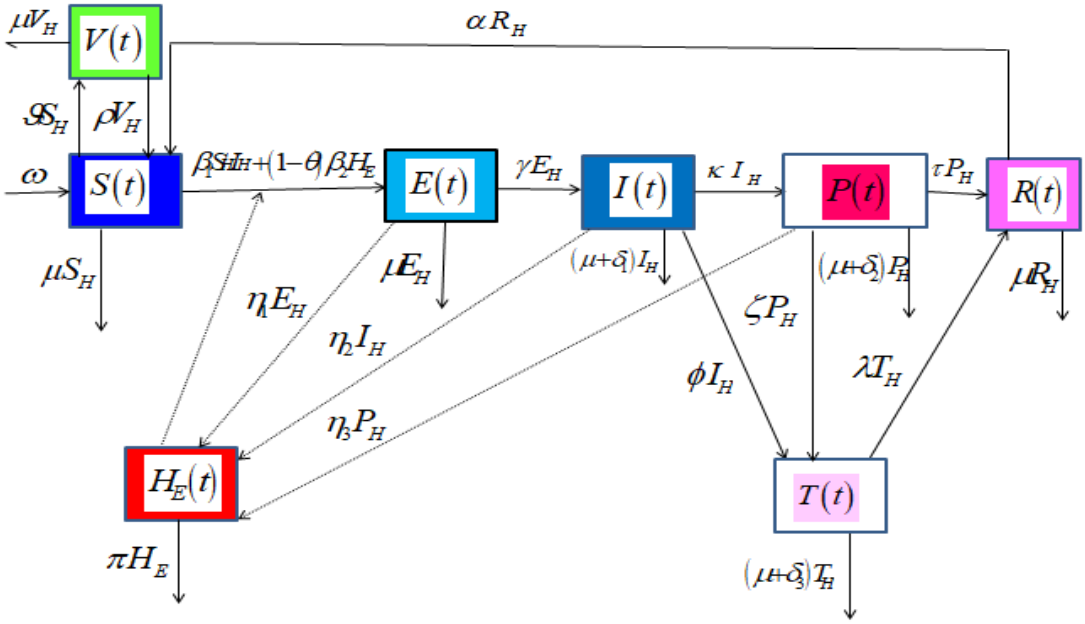

Fig. 1: Schematic Diagram of the Model

Considering these formulations and the schematic diagram, we have the following system of nonlinear ordinary equations:

$$\frac{dS}{dt} = \omega + \rho V + \alpha R - \beta_2 \theta H - (\vartheta + \beta_1 I + \mu) S \quad 1$$

$$\frac{dV}{dt} = \vartheta S - (\rho + \mu) V \quad 2$$

$$\frac{dE}{dt} = \beta_1 IS + (1-\theta)\beta_2 H - (\eta_1 + \gamma + \mu) E \quad 3$$

$$\frac{dI}{dt} = \gamma E - (\delta_1 + \phi + \kappa + \eta_2 + \mu) I \quad 4$$

$$\frac{dP}{dt} = \kappa I - (\tau + \zeta + \delta_2 + \mu) P \quad 5$$

$$\frac{dT}{dt} = \phi I + \zeta P - (\lambda + \delta_3 + \mu) T \quad 6$$

$$\frac{dR}{dt} = \tau P + \lambda T - (\alpha + \mu) R \quad 7$$

$$\frac{dH}{dt} = \eta_1 E + \eta_2 I + \eta_3 P_H - \pi H \quad 8$$

Model Analysis

Rate of Change of the System

From the above model equations, we derive the rate of change of the total population as.

$$\frac{dN(\mathrm{t})}{dt} = \omega - \mu S - \mu V + (1-2\theta)\beta_2 H - \mu E - (\delta_1 + \mu) I - (\delta_2 + \mu) P - (\delta_3 + \mu) T - \pi H_E - \mu R \quad 9$$

Existence and Uniqueness of the Solution

First, we formulate the theorem on existence and uniqueness of the solution of system of the model developed as;

$$\left.\begin{aligned} x_1' &= f_1(t, x_1, x_2, ..., x_n) \\ x_2' &= f_2(t, x_1, x_2, ..., x_n) \\ &. \\ &. \\ &. \\ x_n' &= f_n(t, x_1, x_2, ..., x_n) \end{aligned}\right\} \quad 10$$

And establish the proof.

The above may be expressed in compact form as

$$x_i' = f_i(\mathrm{t}, \mathrm{x}), \mathrm{x}(\mathrm{t}_0) = \mathrm{x}_0, i = 1, ..., n \quad 11$$

Theorem 1

Let D denote the region

$|\mathrm{t} - \mathrm{t}_0| \le \mathrm{a}, \ \|x - x_0\| \le \mathrm{b}, \ x = (\mathrm{x}_1, \mathrm{x}_2, ..., \mathrm{x}_n), \ x_0 = (\mathrm{x}_{10}, \mathrm{x}_{0,2}, \mathrm{x}_{20}, ..., \mathrm{x}_n)$ and suppose that $f(\mathrm{t}, \mathrm{u})$ satisfies Lipchitz condition

$\|\mathrm{f}(\mathrm{t}, \mathrm{x}_1) - \mathrm{f}(\mathrm{t}, \mathrm{x}_2)\| \le k \|\mathrm{x}_1 - \mathrm{x}_2)\|$

Whenever the pairs $(\mathrm{t}, \mathrm{x}_1)$ and $(\mathrm{t}, \mathrm{x}_2)$ belongs to D where k is a positive constant. Then there is a constant $\delta > 0$ such that there exist a unique continuous vector solution $x(t)$ of the model in the interval $|t - t_0| \le \delta.$ It is essential to note that the condition is satisfied by the requirement $\frac{\partial f_i}{\partial x_j} i, j = 1, 2, \ldots, n$ are continuous and bounded in D.

Lemma 1

Let D denote the region $0 \le N \le K,$ then system has a unique solution.

Proof:

Let $x_1 = S_H, \mathrm{x}_2 = V_H, x_3 = E_H, x_4 = I_H, x_5 = P_H, x_6 = T_H, x_7 = R_H, x_8 = H_E$

Also let, $F_1,\ F_2,\ F_3,\ F_4,\ F_5,\ F_6, F_7\ and\ F_8$ be the model equations respectively. Our goal is to show that $\frac{\partial F_i}{\partial X_j}.i, j = 1,2,...,7$ are continuous and bounded in D.

Now,

$$\left.\begin{aligned}
F_1 &= \omega + \rho x_2 + \alpha x_6 - \beta_2\theta x_7 - \left(\vartheta + \beta_1 x_4 + \mu\right)x_1 \\
F_2 &= \vartheta x_1 - \left(\rho + \mu\right)\mathrm{x}_2 \\
F_3 &= \beta_1 x_1 x_4 + \left(1-\theta\right)\beta_2 x_7 - \left(\eta_1 + \gamma + \mu\right)\mathrm{x}_3 \\
F_4 &= \gamma \mathrm{x}_3 - \left(\delta_1 + \phi + \kappa + \eta_2 + \mu\right)\mathrm{x}_4 \\
\mathrm{F} &= \kappa \mathrm{x}_4 - \left(\tau + \zeta + \delta_2 + \mu\right)\mathrm{x}_5 \\
F_5 &= \phi x_4 + \zeta \mathrm{x}_5 - \left(\lambda + \delta_2 + \mu\right)\mathrm{x}_6 \\
F_6 &= \tau \mathrm{x}_5 + \lambda x_6 - \left(\alpha + \mu\right)\mathrm{x}_7 \\
F_7 &= \eta_1 x_3 + \eta_2 x_4 - \pi x_8
\end{aligned}\right\} \quad 12$$

Consider the partial derivatives for $f_1, f_2, \ldots\ldots\ldots\ldots\ldots\ldots\ldots, f_7$ below.

For f_1, we have:

$$F_1 = \omega + \rho x_2 + \alpha x_6 - \beta_2\theta x_7 - \left(\vartheta + \beta_1 x_4 + \mu\right)x_1$$

$$\left|\frac{\partial F_1}{\partial x_1}\right| = \left|-\left(\vartheta + \beta_1 x_4 + \mu\right)\right| < \infty, \left|\frac{\partial F_1}{\partial x_2}\right| = \left|\rho\right| < \infty, \left|\frac{\partial F_1}{\partial x_7}\right| = \left|\alpha\right| < \infty, \left|\frac{\partial F_1}{\partial x_9}\right| = \left|-\beta_2\theta\right| < \infty, \left|\frac{\partial F_1}{\partial x_3}\right| = \left|\frac{\partial F_1}{\partial x_4}\right| = \left|\frac{\partial F_1}{\partial x_5}\right| = \left|\frac{\partial F_1}{\partial x_6}\right| = 0 < \infty$$

Clearly $\left|\frac{\partial F_1}{\partial x_1}\right|, \left|\frac{\partial F_1}{\partial x_2}\right|, \left|\frac{\partial F_1}{\partial x_7}\right|, \left|\frac{\partial F_1}{\partial x_9}\right|, \left|\frac{\partial F_1}{\partial x_3}\right|, \left|\frac{\partial F_1}{\partial x_4}\right|, \left|\frac{\partial F_1}{\partial x_5}\right|\ and\ \left|\frac{\partial F_1}{\partial x_6}\right|$ its continous and bounded

If proceed with the same processes, we proved for $f_2, f_3, \ldots\ldots\ldots\ldots, f_8$.

Hence, it clearly showed there exist a unique solution of equations (12) in the region D.

Positivity of the Solution

The positivity of the solution of the model equations (1) to (8) will be explored by the following theorem:

Theorem 2

Giving an initial solution set of the model equation giving by

$$\left\{S_H(0) > 0, \mathrm{V}_H(0) > 0, E_H(0) > 0, \mathrm{I}_H(0) > 0, \mathrm{P}_H(0) > 0, \mathrm{T}_H(0) > 0, , R_H(0) > 0, \mathrm{H}_E(0) > 0\right\} \in \mathbb{R}^7_+$$

then the solution set

$$\left\{S_H(\mathrm{t}), \mathrm{V}_H(\mathrm{t}), E_H(\mathrm{t}), \mathrm{I}_H(\mathrm{t}), \mathrm{P}_H\mathrm{t}), . \mathrm{T}_H(\mathrm{t}), R_H(\mathrm{t}), \mathrm{H}_E(\mathrm{t})\right\} \text{is } positive\ for\ all\ time\ t\ >\ 0$$

Proof: From the first differential equation in equation (), we have

$$\frac{dS}{dt} = \omega + \rho V + \alpha R - \vartheta S - \beta_1 IS - \beta_2\theta H - \mu S \qquad (13)$$

By comparison theorem we deduce

$$\frac{dS_H}{dt} \geq -\left(\vartheta + \beta_1 I + \mu\right) S_H$$

$$\Rightarrow \frac{dS_H}{S_H} \geq -\left(\vartheta + \beta_1 I + \mu\right) dt \qquad (14)$$

Integrating (14), we have

$$\int \frac{dS_H}{S_H} \geq -\int \left(\vartheta + \beta_1 I + \mu\right) dt \qquad (15)$$

Solving (15) using separation of variable, we get

$$S_H(t) \geq S_H(0) e^{-(\vartheta+\beta_1 I+\mu)t} > 0 \qquad (16)$$

From equation (2), we have

$$\frac{dV}{dt} = \vartheta S - \rho V - \mu V \qquad (17)$$

By comparison theorem we deduce

$$\frac{dV_H}{dt} \geq -\left(\rho + \mu\right) V_H$$

$$\Rightarrow \frac{dS_H}{V_H} \geq -\left(\rho + \mu\right) dt \qquad (18)$$

Integrating equations (18), we have;

$$\int \frac{dV_H}{V_H} \geq -\int (\rho + \mu) dt \qquad (19)$$

Solving equation (19) using separation of variable, we get

$$V_H(t) \geq V_H(0) e^{-(\rho+\mu)t} > 0 \qquad (20)$$

Similarly, it follows that from the differential equations of the model in equations, if we proceed with the same processes, we proved that;

$$E_H(t) \geq E_H(0) e^{-(\eta_1+\gamma+\mu)t} > 0 \qquad (21)$$

$$I_H(t) \geq I_H(0) e^{-(\delta_1+\phi+\kappa+\eta_2+\mu)t} > 0 \qquad (22)$$

$$\mathrm{P}_H(t) \geq \mathrm{P}_H(0) e^{-(\delta_1+\phi+\kappa+\eta_2+\mu)t} > 0 \qquad (23)$$

$$T_H(t) \geq T_H(0) e^{--(\lambda-\delta_3-\mu)t} > 0 \qquad (24)$$

$$R_H(t) \geq R_H(0) e^{-(\alpha+\mu)t} > 0 \qquad (25)$$

$$H_E(t) \geq H_E(0) e^{-\pi t} > 0 \qquad (26)$$

Hence, all the solution of system of model equations with non-negative initial conditions remain non-negative for all t>0.

Region of Absolute Stability

The paper obtained the region of absolute stability by considering the total population under study $N_H(t)$ and $N_{H_E}(t)$ to be closed. Therefore, the model equations will be analyzed in a biologically feasible region.

Theorem 3

The solutions to the system of model equations with initial condition in theorem 1 satisfy $s(0)>0, \mathrm{e}(0)>0, i(0)>0, q(0)>0, r(0)>0, p(0)>0$.

Let $\Omega = \Omega_H \cup \Omega_{H_E}$ imply $\Omega = \left\{N_H(t) \cup N_{H_E}(t)\right\} \in R^8$. is positively invariant

Proof:

Adding the first seven equation of ( ) gives

$$N_H(t) = S_H + V_H + E_H + I_H + \mathrm{P}_H + \mathrm{T}_H + R_H$$

By different ion is written as;

$$\frac{dN_H}{dt} = \frac{dS_H}{dt} + \frac{dV_H}{dt} + \frac{dE_H}{dt} + \frac{dI_H}{dt} + \frac{dP_H}{dt} + \frac{d\,\mathrm{T}_H}{dt} + \frac{dR_H}{dt} \qquad (27)$$

Substituting the first seven equation of the model and differentiate with respect to t, we obtain

$$\frac{dN_H}{dt} = \omega - \mu N_H - (\zeta + \delta_2 + \tau)\ \mathrm{P}_H \qquad (28)$$

When there is no infectious human with peptic ulcer disease, it indicates that $(\zeta = \delta_2 = \tau = 0)$ then (28) as upon simplify becomes

$$\frac{dN_H}{dt} + \mu N_H = \omega \qquad (29)$$

This is linear differential equation, compared it with general form and use integrating factor method that is, $\left[\frac{dy}{dx} + P(\mathrm{x})\,\mathrm{y} = \mathrm{Q}(x)\, I.F = e^{\int p(\mathrm{x})\mathrm{dx}}\right]$ on equation (28.), Hence;

$$P(t) = \mu,\ \mathrm{Q}(t) = \omega \text{ and } I.F = e^{\int \mu dt} = e^{\mu t}$$

Then general solution is given by

$$N_H(t) \le \frac{1}{(I.F)} \int Q(t)(I.F)\, dt \qquad (30)$$

Making substitution and simplify on equation (30), we get

$$N_H(t) \le \frac{\omega}{\mu} + \left(N_H(0) - \frac{\omega}{\mu} \right) e^{-\mu t}$$

At $t \to \infty$ we have

$$\lim_{x \to t} N_H(t) = \frac{\omega}{\mu} \tag{31}$$

$$\Rightarrow 0 \le N_H(t) \le \frac{\omega}{\mu} \tag{32}$$

As $t \to \infty$, the population size $N_H(t) \to \frac{\omega}{\mu}$ which implies that $0 \le N_H(t) \le \frac{\omega}{\mu}$. Thus,

$$\Omega_H = \left\{ (S_H, V_H, E_H, I_H, \mathrm{T}_H, R_H) \in \mathrm{R}_+^6 : 0 \le N_H(t) \le \frac{\omega}{\mu} \right\} \tag{33}$$

By consider the region of the absolute stability solution for the bacteria concentration at time t

From equation (8), we have

$$\frac{dN_{H_E}(t)}{dt} = \eta_1 E + \eta_2 I + \eta_3 P - \pi H$$

$$\Rightarrow \frac{dN_{H_E}(t)}{dt} + \pi H_E = \eta_1 E + \eta_2 I + \eta_3 P \tag{34}$$

Let $K = \eta_1 E + \eta_2 I + \eta_3 P$ which is the recruitment rate of the bacteria from infectious classes, then we obtain a differential inequality

$$\frac{dN_{H_E}(t)}{dt} + \pi H_E \le K \tag{35}$$

Using integrating factor method, we get

$$N_{H_e}(t) \le \frac{\mathrm{K}}{\pi} + \hat{C} e^{-\pi t}, \text{ where } \hat{C} \text{ is a constant}$$

At t approaches infinity, we have

$$\lim_{t \to \infty} N_{H_E}(t) = \frac{\mathrm{K}}{\pi} \tag{36}$$

$$\Rightarrow 0 \le N_{H_E}(t) \le \frac{\mathrm{K}}{\pi} \tag{37}$$

As $t \to \infty$, the population size $N_{H_E}(t) \to \frac{\mathrm{K}}{\pi}$ which implies that $0 \le N_{H_E}(t) \le \frac{\mathrm{K}}{\pi}$. Thus,

$$\Omega_{H_E} = \left\{ (\mathrm{H}_E) \in \mathrm{R}_+^1 : 0 \le N_{H_E}(t) \le \frac{\mathrm{K}}{\pi} \right\} \tag{38}$$

Hence, the feasible solution set of the system equation of the model enter and remain in the region that makes a biological sense and it is well posed.

Existence of Disease-Free Equilibrium State

It is a state in which the disease does not exist. So, from this, the class where the disease exists will be equals zero (o). By setting $E=0,\ I=0,\ P=0,\ T=0, H_E=0$ in the model equations and solve it, we get;

$$E_0=\left(S^*,\mathrm{V}^*,\mathrm{E}^*,\mathrm{I}^*,\mathrm{H}_E{}^*,\mathrm{T}^*,\mathrm{R}^*\right)=\left(\frac{\omega(\rho+\mu)}{\left(\left[(\rho+\mu)(\vartheta+\mu)\right]-\rho\vartheta\right)},\frac{\omega\vartheta(\rho+\mu)(\rho+\mu)}{\left(\left[(\rho+\mu)(\vartheta+\mu)\right]-\rho\vartheta\right)},0,0,0,0\right) \quad (39)$$

The equilibrium state of the model exists when $(\rho+\mu)(\vartheta+\mu)>\rho\vartheta$

Basic Reproduction Number (R_0)

The basic reproduction number denoted by R_0 is the average number of secondary infections generated by single infected individual in a totally susceptible population; it is an important parameter that is used to study the behavior of epidemiological models. In order to obtain the basic reproduction number R_0 we used next generation matrix technique. By considering a non-negative matrix F and non-singular matrix V, represents new infections terms and transfer of infections terms from the model equations respectively, we have

$$F=\begin{bmatrix}\beta_1 I_H S_H+(1-\theta)\beta_2 H_E\\0\\0\\0\\\eta_1 E+\eta_2 I+\eta_3 P\end{bmatrix} \quad (40)$$

$$\mathrm{V}=\begin{bmatrix}(\eta_1+\gamma+\mu)E_H\\(\delta_1+\phi+\kappa+\eta_2+\mu)I_H-\gamma E_H\\(\tau+\delta_2+\xi+\eta_3+\mu)\mathrm{P}-\kappa I\\(\lambda+\delta_2-\mu)T_H-\phi I_H\\\pi H_E\end{bmatrix} \quad (41)$$

By simplify equation (40 and 41) at DFE and evaluating the Jacobean matrix, we get;

$$F=\begin{bmatrix}0&G_1&0&0&G_2\\0&0&0&0&0\\0&0&0&0&0\\0&0&0&0&0\\\eta_1&\eta_2&\eta_3&0&0\end{bmatrix} \quad (42)$$

$$V=\begin{bmatrix} W_1 & 0 & 0 & 0 & 0 \\ -\gamma & W_2 & 0 & 0 & 0 \\ 0 & -\kappa & W_3 & 0 & 0 \\ 0 & -\phi & -\zeta & W_4 & 0 \\ 0 & 0 & 0 & 0 & W_5 \end{bmatrix} \qquad (43)$$

Where;

$G_1=\frac{\beta_1\omega(\rho+\mu)}{\left(\left[(\rho+\mu)(\vartheta+\mu)\right]-\rho\vartheta\right)}$, $G_2=(1-\theta)\beta_2$, $W_1=(\eta_1+\gamma+\mu)$, $W_2=(\delta_1+\phi+\kappa+\eta_2+\mu)$, $W_3=(\tau+\delta_2+\xi+\eta_3+\mu)$ $W_4=(\lambda+\delta_2+\mu)$ and $W_5=\pi$,

Inverse of V is evaluated and given by;

$$V^{-1}=\begin{bmatrix} \frac{1}{W_1} & 0 & 0 & 0 & 0 \\ \frac{\gamma}{W_1 W_2} & \frac{1}{W_2} & 0 & 0 & 0 \\ \frac{\kappa\gamma}{W_2 W_1 W_3} & \frac{\kappa}{W_2 W_3} & \frac{1}{W_3} & 0 & 0 \\ \frac{\gamma(\zeta\kappa+\phi W_3)}{W_3 W_2 W_1 W_4} & \frac{\zeta\kappa+\phi W_3}{W_3 W_2 W_4} & \frac{\zeta}{W_3 W_4} & \frac{1}{W_4} & 0 \\ 0 & 0 & 0 & 0 & \frac{1}{W_5} \end{bmatrix} \qquad (44)$$

$$FV^{-1}=\begin{bmatrix} \frac{G_1\gamma}{W_1 W_2} & \frac{G_1}{W_2} & 0 & 0 & \frac{G_2}{W_5} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ \frac{\gamma(\zeta\kappa+\phi W_3)}{W_4^2 W_3 W_2 W_1} & \frac{\zeta\kappa+\phi W_3}{W_4^2 W_3 W_2} & \frac{\zeta}{W_4^2 W_3} & \frac{1}{W_4^2} & 0 \\ \frac{\eta_1}{W_1}+\frac{\eta_2\gamma}{W_1 W_2}+\frac{\eta_3\kappa\gamma}{W_2 W_1 W_3} & \frac{\eta_2}{W_2}+\frac{\eta_3\kappa}{W_2 W_3} & \frac{\eta_3}{W_3} & 0 & 0 \end{bmatrix} \qquad (45)$$

The basic reproduction number which is the largest eigenvalue obtained by solving the characteristic polynomial of equation (45) given as;

Then

$$R_0^*=\frac{\beta_1\omega\gamma(\rho+\mu)(\eta_1+\gamma+\mu)(\delta_1+\phi+\kappa+\eta_2+\mu)}{\left(\left[(\rho+\mu)(\vartheta+\mu)\right]-\rho\vartheta\right)}+X \qquad (46)$$

Where;

$$X=\sqrt{\left(\frac{\beta_1\omega\gamma(\rho+\mu)(\eta_1+\gamma+\mu)(\delta_1+\phi+\kappa+\eta_2+\mu)}{\left(\left[(\rho+\mu)(\vartheta+\mu)\right]-\rho\vartheta\right)}\right)^2+4\left(\frac{(1-\theta)\beta_2}{(\lambda+\delta_2+\mu)}\cdot\frac{\eta_1(\delta_1+\phi+\kappa+\eta_2+\mu)(\eta_1+\gamma+\mu)+\eta_2\gamma(\eta_1+\gamma+\mu)+\eta_3\kappa\gamma}{(\eta_1+\gamma+\mu)^2(\delta_1+\phi+\kappa+\eta_2+\mu)}\right)}$$

And the control reproduction number for reduced system (1) to (7) is given as;

$$R_0^* = \frac{\beta_1 \omega\gamma(\rho+\mu)(\eta_1+\gamma+\mu)(\delta_1+\phi+\kappa+\eta_2+\mu)}{\left(\left[(\rho+\mu)(\vartheta+\mu)\right]-\rho\vartheta\right)} \quad (47)$$

It is known that the expected duration of infectious is the inverse of the removal rate (Hethcote, 2000 and British international school, 2023). According to the definition by Hethcote, (2000), the removal rate (τ), Exposed rate (γ) and transmission rate (β) are given as follows:

$$\tau = \frac{1}{Mean \text{ infectious period}} \quad (48)$$

$$\gamma = \frac{1}{Mean \text{ latency period}} \quad (49)$$

$$\beta = \frac{Effective}{Total\ contact} \quad (50)$$

Herd Immunity Threshold

In order control the transmission of the disease, the population has to be immunized. The percentage of the population that needs to be immunized for controlling the transmission is the Herd Immunity (HIT), It protects directly the immunized individuals from infection and also provides protection of being susceptible individuals in (Islamr, Biswas and Jamali, 2017). To evaluate HIT, we use the equation given as follows;

$$H_T = 1 - \frac{1}{R_0} \quad (51)$$

This means that vaccinating $1-\frac{1}{R_0}$ of the population of Nigeria by 100% can control the spreading of the disease.

Numerical Analysis

In order to evaluate the Herd Immunity Threshold as well as numerical analysis of the models, we have considered the information about peptic ulcer disease (Educate innovate research and development, 2016). and H. pylori virus as defined in (Dore and Graham, 2022). and real numerical data in perspective of Nigeria (Knoema, 2023). The latency period of peptic ulcer disease is 3 to 4 days (Public Health Agency of Canada, 2011). and infectious period is some days prior to onset of symptoms to 19 days after symptom onset (Yin, He, Zhang, 2009)/ So, the mean latency period of peptic ulcer disease is 3 days and mean infectious period is 9.5 days. Therefore, by using equations (49) and (48), we have $\tau = 0.10526,\ \gamma = 0.2857.$ According to (Yin, He, Zhang, 2009). from February 2014 to September 2019, 434 individuals had been tested and among them 31 individuals were found peptic ulcer positive. Again, for month January 3, 2014 to January 21, 2021, 2, 735

individuals have been tested and among them 298 individuals were found i peptic ulcer positive (Yahya, 2023). Therefore, we have;

$$\beta_1 = \frac{31}{434},\ \beta_2 = \frac{298}{2735} \text{ and } \beta = \frac{\beta_1 + \beta_2}{2} = 0.0902$$

Table 1: Parameters used for computational results

Parameters	Values	References
ω	10	(Mutua, Ngari, Muthuri and Kitavi, 2022)
β	0.00902	Estimated
ϑ	0.000031	Assumed
ρ	0.0031	Assumed
τ	0.2857	Estimated
γ	0.10526	Estimated
κ	0.003	(Rupnow, et al. 2000).
δ_1	0.00001	Assumed
ϕ	0.0073	(Teng, et al. 2017).
η_1	0.0009	(Mutua, Ngari, Muthuri and Kitavi, 2022)
η_2	0.0008	(Mutua, Ngari, Muthuri and Kitavi, 2022)

(Authors computation)

We have the death rate of population of Nigeria as $\mu = 12.4$ and the total population of Nigeria as $N = 218,541,212$ (Knoema, 2023) Using the value from table 4.1 in (47) we get R_0 =188.2530, So Disease Free Equilibrium (s, v, e, i, p, t, h, r) = (0.80645, 0.00031, 0, 0, 0, 0, 0) is unstable as $\left(R_0 = 188.2530 > 1\right)$. By substituting this value in equation (51), the Herd Immunity Threshold is calculated as follows:

$$H_T = 1 - \frac{1}{R_0} = 0.9947$$

Now, R_0 >1 revealed that the states of disease-free equilibrium is unstable. In the case of unstable state of disease-free equilibrium, the diseases will spread out. So, to control the spread of the disease, we need to calculate the Herd Immunity Threshold. In perspective Nigeria, we have calculated the Herd Immunity Threshold as 0.9947. This means that vaccinating 99.47% population of Nigeria can control spreading of Peptic ulcer disease.

Conclusion

The study presents model of the transmission dynamics of Peptic ulcer disease that account for vaccination of susceptible individuals and treatment of infected individuals as control, the region of absolute stability result shown that as time tend to infinite the population size will remain in a biologically feasible region, results also indicated that, the dynamics of the spread of Peptic ulcer is determined by the basic reproduction number R_0, the estimated basic reproduction number value of the developed model is greater than 1 in perspective of Nigeria. This significantly indicated that the disease will spread out in the society among individuals. In the study one of the controlling strategies is vaccinating, for which the Herd Immunity Threshold in perspective of Nigeria for peptic ulcer disease is found to be 0.9947 by using the model developed, this means that vaccinating 99.47% of Nigeria population can control spreading of Peptic ulcer disease.

Recommendations

The cases of Peptic Ulcer disease can be minimized in the population, if the following recommendations are considered.

- Early implementation of infection control precautions to minimize the spread of Helicobacter pylori concentration in the environment and prompt treatment to prevent severe illness and death should be put in place at all time.
- Amoxicillin, clarithromycin and metronidazole together with a proton pump inhibitor and bismuth compound drug should be provided for treatment of confirmed complicated cases of Peptic ulcer disease.
- Government and non-governmental organization should encourage the use of Peptic ulcer vaccine among individuals because it plays a vital role in reducing the spread of the bacteria.

References

Assefa, B., Abay, Z., Abebe, A., Tesfaye, T., Tadesse. M., Lakew, A.M. (2021) Prevalence of peptic ulcer disease and associated factors among dyspeptic patients at endoscopy unit, university of gondar hospital, Research Article, northwest Ethiopia. DOI: https://doi.org/10.21203/rs.3.rs-1152130/v1

British international school, Phuket (2023) modeling of infectious diseases; I.B Maths Resource retrieve on 30/11/2023 fro www.ibmathsresources.com/2014/05/17/modelling-infectious-diseases/

Daily Trust (2023). Peptic Ulcer Disease in Nigeria retrieve from www.dailytrust.com on 16 March, 2023.

Dore, M.P., Graham, D.Y. (2022) Modern approach to the diagnosis of Helicobacter pylori infection. Aliment Pharmacol. Ther. 55. S14-S21. https://doi.org/10.1111/apt.16566

Educate innovate research and development (2016) Epidemiology peptic ulcer disease, retrieved on 06/07/2023 http://bestpractice.bmj.com/best-practice/monograph/80/basics/epidemiology.html

Hethcote H.W, (2000) The mathematics of Infectious Diseases, SIAM Review, 42(4):599 -653

Islamr. R., Biswas, M. H. A. and Jamali, A. J. U. (2017). Mthematical analysis of Epidemiological dynamics of influenza A (H1N1) virus Transmission Dynamics in Bangladesh. Math,Sci, 37, 39 50

Kabir, S. (2007). The current status of Helicobacter pylori vaccines: a review. *Helicobacter, 12*(2), 89-102. DOI:10.1111/j.1523-5378.2007.00478.x

Knoema (2023) Nigerian demographic, retrieved on 31/12/2023 from https://knoema.com/atlas/Nigeria/popilation

Mutua, K.G, Ngari, C.G., Muthuri, G. G., Kitavi, D.M. (2022) Mathematical modeling and simulating of *Helicobacter Pylori* treatment and transmission implications on stomach cancer dynamics.

Narayanan M, Reddy KM, Marsicano E. (2018) Peptic Ulcer Disease and *Helicobacter pylori* infection. Mo Med. 115(3), 219-224.

National library of Medicine and National Center for Biotechnology information, (2023), peptic ulcer disease, retrieved on 06/07/2023 from https://www.ncbi.lnm.nih.gov/books//NBK534792/

Obafemi Awolowo University Medical Students Association (2018) peptic ulcer disease in Nigeria, Ifemed Journal of Medicine vol 33, issue 1 pp 1-4.

Prof. (Dr.) Md. Zulfikar Ali Chief Editor (2013). Editorial, Peptic ulcer disease, KYAMC Journal; Vol. 3, No.-2, pp 270-272

Public Health Agency of Canada, PHAC (2011) Pathogen Safety Data Sheets: Infectious Substances Heliobacter pylori, retrieved on 27/08/2023 from https://www,canada.ca/en/public-health/services/laboratory-biosecurity/pathogen-safety-data-sheete-risk=assessment/helicobacter-pyloru.html

Ray-Offor, E. & Opusunjuk. A. (2020). Current status of peptic ulcer disease in PortHarcourt metropolis, Nigeria, African Health Science, 20(), 1446 – 1451.

Rupnow, M.F., Shachter, R.D., Owens, D.K., et al. (2000). A dynamic transmission model for predicting trends in helicobacter pylori and associated diseases in the United States, Emerg. Infect. Dis. 6, 228–237.https://doi.org/10.3201/eid0603.000302.

Teng, A.M., Kvizhinadze, G., Nair, N., et al. (2017). A screening program to test and treat for Helicobacter pylori infection: Cost-utility analysis by age, sex and ethnicity, BMC Infect. Dis. 17, 156. https://doi.org/10.1186/s12879-017-2259-2.

Whittle, R., Edinburgh, M.T., Gilmore, I. T. (2008) Ministry of Defence. Synopsis of Causation, Royal Liverpool University Hospital, Liverpool.

World Health Organization (2020) Peptic Ulcer Disease in Nigeria; world Life Expectancy retrieve on 20/12/2023 from https://www.worldlifeexpectancy.com/Nigeria-peptic-ulcer-disease

Yahya, H. (2023). Diagnostic Upper Gastrointestinal Endoscopy in Kaduna, Northwest Nigeria: Indications and Findings, Nigerian Journal of Basic and Clinical Sciences, 20(1), 82-88**.**

Yin Y, He, L. H., Zhang, J, Z. (2009). Successful isolation of *Helicobacter pylori* after prolonged incubation from a patient with failed eradication therapy. *World J Gastroenterol*; 15(12): 1528-1529 Available from: URL: http://www.wjgnet.com/10079327/15/1528.asp DOI: http://dx.doi.org/10.3748/wjg.15.1528