ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26nov539

Within the Host Mathematical Model of Interactions Between Pathogen and Human Immune Cells' Responses

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Publication Date: 2026/11/28

Abstract: The human body is equipped with immune cells that protect its systems from any attack by pathogens. The mechanism of these interactions is vital, and dynamics of the interaction procedure is an essential phenomenon to study and analyze. This research applies the predator-prey-modelling style, to propose a classic within-the-host model using a Holling type II functional that deals with activated response by immune cells to clear invading pathogen. The mechanism of pathogens in the act of invading the host body through overpowering protective processes by immune cells to cause disease is described and analyzed. Existence of solution for the model was mathematically ascertained using Picard-Lindelof's theorem, the model's infection-free equilibrium and endemic equilibrium states were obtained. Local and global conditional stability of infection-free equilibrium of the model were established. Threshold number, analogous to basic reproduction number was calculated. Conditional endemic equilibrium was obtained as: if $\theta > \alpha\tau$, endemic equilibrium is stable, but it is unstable when $\theta < \alpha\tau$. The result showed that, when H > P, with $\theta > \alpha\tau$ and $R_{th} > 1$ then initial pathogen that invaded will be successfully cleared, hence, establishing chance of controlling the pathogens. Biological interpretations of the conditions were provided. Rates of pathogens and immune-responses were obtained in values, portraying the categories of infections that may result as consequence of the interaction, the model's simulations of the interactions were provided.

Keywords: *Immune-Response, Symptom, Pathogen, Interactions, Infection.*

How to Cite: Sani Fakai Abubakar; Farooq Abubakar Atiku; Ahmad Ibrahim Bagudo (2026) Within the Host Mathematical Model of Interactions Between Pathogen and Human Immune Cells' Responses. *International Journal of Innovative Science and Research Technology,* 11(11), 2148-2155. https://doi.org/10.38124/ijisrt/26nov539

I. INTRODUCTION

Mathematical modelling is the process of selecting and utilizing relevant mathematical ideas to scrutinize and portray real life problems in a better way to understand, apply safer decisions about it. Modelling techniques using appropriate data and information system is among the latest techniques that may contribute to the prediction and control of infectious diseases, [16]. Mathematical models can be used to describe phenomenon, drive hypothesis, heuristically support efforts of conceptualize data and predict trends. Its results especially, if collaborated with experiments, can be a proven valuable and accurately parametric tool to depend on, for any phenomena, and also to depict complex data usefulness, see [21]. Many mathematical models especially in immunology, have usefully described local molecular events at the sub-cellular level, this add to the understanding of signal processing in biology. Mathematical models help in better understanding of the long-term behavior of interacting components, and produce steady state solutions, See [27].

Immune system supposed to have capabilities of controlling, eliminating and limiting spread of infectious pathogens in human body, see [32]. It is also stated in [11], that human body immunity has been ascertained to naturally over power pathogens and virulent that may invade it, that Immune cells response can clear most infections that may be caused by pathogen, analysis of many cells from a selected

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26nov539

woman showed, HIV1 (virus) were naturally over powered by her immune system and replication was not detected, suggesting possibilities of curing HIV-1 by human immune system. Many researchers modeled mathematical models on pathogen and immune response in the literature, some of them are: [18, 31], [5, 20, 32, 33]. Immune process here refers to a network of cells, tissues, and organs that work together to provide extraordinary defense to human body against attacks by foreign invaders. Healthy immune process shall distinguish between body own cells and foreign ones. Antigens trigger immune response, and this response from immune system can thwart all forms of pathogen tactics for invading human body. The immune system mostly, stores just a few of each kind of the different cells needed to recognize millions of possible invaders, see [29].

Human body is always under attack by many and different pathogens, which mainly enter human body to propagate and disseminate, but the complexity of human body immune system, enables it to efficiently eliminate these invading pathogens or reduce their propagation to a minimum. The battle between pathogens and the immune system is a crucial area of immunological research, since the evolutionary pressure on pathogens to circumvent the immune defense mechanisms is much, this leads to the development of pathogenic strains that challenge human health, see [30]. Pathogens are generally considered as biological, chemical and even physical agents that can cause disease in a host body. Infection usually occurred when pathogens successfully avoid innate immune defense mechanisms and stably colonizes a niche in the host. It is estimated that, an average individual's immune system consist of approximately 1.8 trillion (1.8×10^{12}) cells that weigh around 12 kilograms, as stated in [34].

Mechanism of the fight between pathogens and innate immune, start instantly when the pathogen invade or enter the body. Immune quickly sent its fastest response force called *neurophils*, which hunt and chase the invaders by secreting chemicals, surrounding and even swallowing the invader. After a successful fight, dead *neurophils* mixed with the destroyed pathogens and form a pus as healing begins. A healthy immune processes can easily fight and defeat invading pathogens, it can be boosted through good and healthy life style.

Human body immune cells, serve as defense force that protect the body from any form of attack by pathogen that may cause illness. The immune cells response, here entails a system or network of organs, the white blood cells and chemicals. It keeps all dangerous invaders out of the body or destroy them and can adopt new strategies and threat to any incoming harmful invader. Infection is primarily a race between pathogens and the immune cells response, when the invaders (pathogens) bypasses this immune response's barrier defenses, it starts replicating in the host body. Such immune cells response if weakened, the body becomes prone to the pathogens attacks, which leads to catching a diseases. [9] modelled immune response to infectious diseases with influence of environmental factors.

Ordinary differential equations coopting Holling type II functional, were used to formulate a hybrid predator-prey model of pathogen and immune response. The immune response, denoted by H, denotes the immune *effectors*, while P, representing pathogens, refers to the pathogenic cells invading, reflecting dynamics of their population. Depending on the situation, pathogen load can be considered as predators and immune response process as prey if disease manifest, or otherwise, but if there is no disease, the immune response process is the predator and invading pathogens are the prey, which mostly lead to successful clearing of the pathogen. Perhaps, the predator-prey-models have been applied in different fields of study, as it comes with different terminologies (names) that depends on the field of study. Ecologist use predator and prey compartments, Epidemiologist use susceptible and infective individuals, Economics use populace and predatory institution, in chemistry combustion theory of two chemical radicals that are formed in combustion of H_2O_2 and many other studies in many other different disciplines. These serve as evidences that support hypothesis of the similarity predator-prey models have with parasite-host models. Most predator-prey models are mainly on bacteria species, insects, cannibalism, coexistence of species and other multicomponent systems, see [38]. Within the host models on the coexistence of pathogen and its human host, in terms of the immune cells response's interaction with pathogen, gap of knowledge that needs more patronage by researchers exist, hence the model was proposed.

II. MODEL FORMULATION

This model describes the behaviors of pathogens, how they survive, replicate and cause different categories of diseases. H as denoted above, is the immune cells (such as *memory T cells or cytotoxic lymphocytes, antibodies, innate immune cells*), their population considered at time t and P as also denoted above, is the pathogen (load) population at time t. Without harmful pathogens, the host body is freed from pathogen related infection, but without H which is immune cells response system, the host body is under threat of harmful pathogens. On the other hand, r is the rate of increment of the pathogens (pathogens intrinsic replication rate), and φ is the rate of eliminating pathogens by immune cells response, whilst θ is the rate of stimulating, triggering, activating and proliferation of immune cells in the presence of pathogens. Sum that, α is the death rate of immune cells, and τ is time taken by immune processes to successfully handle or clear pathogens, or limit interaction at high pathogen load situations.

➢ *Basic Assumptions*

- Normal immune response destroy pathogens through encounters.
- When pathogen density become high, immune response get saturated, clearance of pathogens by immune cells response become weak.
- Pathogen can reproduce inside (human body) host at an intrinsic increment rate.

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26nov539

- Presence of pathogens, trigger or activates immune response processes.
- Immune response cells die naturally, though *neutrophils* can die as result of interaction with pathogens.
- Low pathogen load allow efficient clearing of pathogen by immune cells response.
- Normal immune processes is considered in the modelling, no treatment or vaccination.
- Homogeneous host environment is considered in the modelling.

➢ *The Model*

The model is a modified Lotka-Volterra style predator-prey model, formulated using Holling type II equation $\frac{P}{1+\tau P}$, as in [5, 31]; [17, 18]; [12, 33, 36].

It is made up of a two dimensional system of Ordinary Differential Equations (ODE), describing interactions between body immune cells response and pathogens. The autonomous ODE describes the dynamics of pathogens (P) and Immune cells response (H) (such as, antibodies or lymphocytes) that served as the model's state variables, thus:

$$\frac{dP}{dt} = rP - \frac{\phi PH}{1+\tau P} \tag{1}$$

Describes the pathogen load dynamics, while the immune cells response dynamics is described as:

$$\frac{dH}{dt} = \frac{\theta PH}{1+\tau P} - \alpha H \tag{2}$$

The model aim at predicting outcomes of interactions between pathogens and body immune cells responses. Pathogens in the body grow at a rate r, while immune responses is stimulated by the presence of pathogens at a rate θPH and the pathogens are destroyed after encounter with immune processes at φPH rate. For the model to be biologically meaningful, the rates and numbers of pathogens and immune processes are positive and remain non-negative. Rate of eliminating pathogens by immune responses can be slowed when pathogens load become very large. Mathematically, the state variables and parameters of the model, are subset of positive real numbers, hence, this biologically implies their increase can be in terms of growth, decay and/or constants, meaning negative growth can be possible.

$(r, \varphi, \theta, \tau, \alpha) > 0$, (3)

$$\mathbf{R_+^2} = \{(\mathbf{P}, \mathbf{H}) / \mathbf{P} \geq \mathbf{0}, \mathbf{H} \geq \mathbf{0}\}$$

The biologically meaningful initial values of the state variables can then be:

$P(t) = P_0 \geq 0$ (4)

And

$H(t) = H_0 \geq 0$ (5)

Now, the model as a vector value ODE, can be written as:

$$\left(\frac{d\mathbf{y}}{dt} = f(\mathbf{y})\right), \tag{6}$$

Where, $\mathbf{y} = [P, H]^T$, with all state variables non-negative.

➢ *Existence of Solution*

The model's system of equations formed with equations (1) and (2), satisfies all conditions for the Picard-Lindelof's theorem, guaranteeing a unique limited solution for any valid initial state P_0, H_0. Since the right-hand side of the function f as (in theorem 1) is locally Lipschitz continuous on the domain of biologically relevant positive state value $(P, H) \in (0, \infty) \times (0, \infty)$.

➢ *Picard-Lindelof (or ODE Existence and Uniqueness) Theorem*

Theorem 1 *Any initial value problem (IVP), $y' = f(y)$, $y(t_0) = y_0$ has a unique local solution defined on some finite time interval around t_0 if f is continuous in t and locally Lipschitz continuous in the state vector* y.

Proof

Since $y(t) = [P(t), H(t)]$, then $\frac{dy}{dt} = f(y)$,

Where

$$f(y) = \left(rP - \tfrac{\phi PH}{1+\tau P}, \tfrac{\theta PH}{1+\tau P} - \alpha H\right)$$

The right hand side function

$$f(P,H) = [f_1(P,H), \quad f_2(P,H)], \tag{7}$$

$$= \left[rP - \tfrac{\phi PH}{1+\tau P}, \quad \tfrac{\theta PH}{1+\tau P} - \alpha H\right] \tag{8}$$

A function f is Lipschitz continuous on a domain D, if there exist a finite constant $L > 0$ such that, for any two points $|\boldsymbol{y_1}, \boldsymbol{y_2}| \in D$, $f \leq L|\boldsymbol{y_1} - \boldsymbol{y_2}|$.

For differentiable functions, the Lipschitz constant equals the maximum supremum norm of the Jacobian for Lipschitz continuity of smooth functions. It is comprehensible, to see that f has no explicit time dependence, therefore the model is automatically continuous in t. For the Lipschitz continuous, we also see that any continuously differentiable C^1 on an open domain is locally Lipschitz, since its partial derivatives that form the Jacobian matrix, are bounded on any closed bounded subset of the domain. The partial derivatives of f with respect to P and H are continuous and finite for any $P > 0$ and $H > 0$, also $1 + \tau P$ as common denominator, is always greater than one, for

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26nov539

all valid *P*, hence, the biologically relevant state space has no singularity.

For any closed, bounded sub-domain of the space, in this context, $0 < P_{min} \leq P \leq Pmax$, $0 < Hmin \leq H \leq Hmax$, which cover all the conceivable pathogens and immune counts for a specific experiment, so that the partial derivatives are bounded. This implies f satisfies the local Lipschitz condition with a finite Lipchitz constant L derived from the maximum absolute value of the Jacobian entries over the sub-domain.

Therefore, any initial infection state (P_0, H_0) will produce exactly one unique trajectory of pathogens and immune cells response and population over time, with no bifurcations or multiple incompatible outcomes from the same starting conditions. The local Lipschitz continuity, also guarantees the solution depends continuously on initial conditions. Meaning small change to the initial pathogen load and immune cells' response can only produce small variation on the resulting trajectory, this is critical property in modelling any real-life biological systems where initial state measurements always have small errors. When $P = 0$, $\frac{dP}{dt} = 0$, also when $H = 0$, $\frac{dH}{dt} = 0$. This implies that the trajectories cannot cross the coordinate axes, hence, the solution of the model remain and will remain positive for all $t > 0$.

➢ *Equilibrium Points of the Model*

Equating derivatives of the model to zero, thus:

$$0 = rP - \frac{\phi PH}{1+\tau P} \Rightarrow 0 = P\left(r - \frac{\phi H}{1+\tau P}\right) \quad (9)$$

$P = 0$ or $r = \frac{\phi H}{1+\tau P}$, on the other hand,

$$0 = \frac{\theta PH}{1+\tau P} - \alpha H \Rightarrow 0 = H\left(\frac{\theta P}{1+\tau P} - \alpha\right) \quad (10)$$

$H = 0$ or $\alpha = \frac{\theta P}{1+\tau P} P^* = \frac{\alpha}{\theta - \alpha\tau}$ and $H^* = \frac{\theta r}{\phi(\theta - \alpha\tau)}$

The equilibrium points are:

- $P = 0$ meaning no pathogens exist to stimulate immune cells response, while $H = 0$ implies, activating immune response is zero. The equilibrium state is denoted by $E_1 = (0,0)$ and is an infection free state, no pathogens persist in the host and it is biologically relevant.

- The endemic equilibrium state denoted by

$E_2 = P^* > 0, H^* > 0$ where $P^* = \frac{\alpha}{\theta - \alpha\tau}$ and $H^* = \frac{\theta r}{\phi(\theta - \alpha\tau)}$

- Theorem 2 *Necessary and sufficient condition for equilibrium point of a model to be locally asymptotically stable, is the Jacobian matrix of the model must have negative real part eigenvalues. [26]*

- Theorem3 *A 2x2 Jacobian matrix, evaluated at an equilibrium point, with constant entries, if its determinant (DJ) is not equal to zero (DJ* 6= *0), then the equilibrium point is locally asymptotically stable if and only if, its trace (TJ* < *0) is less than zero and its determinant (DJ* > *0) greater than zero. It is unstable if the trace is greater than zero (TJ* > *0) or its determinant is less than zero (DJ* < *0). [26]*

The general Jacobian matrix of model is:

$$J = \begin{bmatrix} r - \frac{\phi H}{(1+\tau P)^2} & -\frac{\phi P}{1+\tau P} \\ \frac{\theta H}{(1+\tau P)^2} & \frac{\theta P}{1+\tau P} - \alpha \end{bmatrix}. \quad (11)$$

Let $E_1 = (0,0) = (P, H)$, and $E_2 = \frac{\alpha}{\theta - \alpha\tau}, \frac{\theta\tau}{\phi(\theta - \alpha\tau)} = (P, H)$

Starting with E_1 equilibrium, where $P = 0$ and $H = 0$, meaning, no pathogen present and no activated immune response, we called it, the model's Infection-Free Equilibrium (IFE) state. This E_1 is parallel to parasite-free or disease-free equilibrium, in this model, it is used in analogous to within-host models, as in [2, 14, 19].

Substituting $P = 0$ and $H = 0$ into (11) gives

$$J(E_1) = \begin{bmatrix} r & 0 \\ 0 & -\alpha \end{bmatrix}$$

To find characteristic equation of the Jacobian at E_1

$$J(E_1 - \lambda I) = \begin{bmatrix} r - \lambda & 0 \\ 0 & -\alpha - \lambda \end{bmatrix}. \quad (12)$$

Where I is identity matrix, the characteristic equation of the Jacobian matrix at E_1 equilibrium point is:

$$\lambda^2 + (\alpha - r)\lambda - \alpha r = 0 \quad (13)$$

The trace of equation (13) is $T = \alpha - r$ and its determinant is $D = -\alpha r$. Therefore from theorem (3), the equilibrium E_1 is unstable. It has to always be unstable, because any introduction of pathogens, r can grow highly for the infection to begin, and onset activation of immune response commence, this can lead to decay (a negative growth), if α is high, therefore infection can begin. Hence, the equilibrium point (0,0) cannot always be stable, meaning a healthy uninfected individual can immediately be infected, if harmful pathogen is allowed grow in the individual's body. However, since $r > 0$, this actually shows that, the pathogens-free equilibrium has to be unstable, because a small pathogen population unchallenged grows up. Mathematically, initial pathogen load behave as follows: $P(t) \approx P_0 e^{rt}$, meaning the pathogens can invade whenever $r > 0$, and otherwise when $r < 0$, that means decay of r.

➢ *Global Stability of IFE (0, 0)*

Using Lyapunov's direct method tests, we see that, if an equilibrium is asymptotically stable (that is, all trajectories of a system are near the equilibrium and converges to it over

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26nov539

time), that is $V(P, H) = 0$ is true only at E_1 and $V(P, H) > 0$ $\forall (P, H)$, then $\dot{V}(P, H) < 0$.

Therefore, at $E_1 = (0,0)$,

$$\dot{V} = \frac{dP}{dt} + \frac{dH}{dt} \tag{14}$$

$$= \left(rP - \frac{\phi PH}{1+\tau P}\right) + \left(\frac{\theta PH}{1+\tau P} - \alpha H\right) \tag{15}$$

Simplifying (15) gives

$$\dot{V} \approx rP - \alpha H \tag{16}$$

Therefore, for $\dot{V} < 0$ to hold in a biologically meaningful context, we must get either $rP < \alpha H$ or r will be negative. Hence, negative r and $rP < \alpha H$ implies $\dot{V} < 0$, which are the required conditions for the global stability of this model. But biologically, negative r is obtainable when decay growth of the pathogen exists, this implies, r can be negative growth of the pathogen. The equilibrium point $E_1 = (0,0)$ is therefore globally unstable, since $\dot{V} \approx rP - \alpha H$ hold, whenever there is a decay in r.

➢ *Basic Reproduction Number (R_0) of the Model*

Basic reproduction number (R_0) of this model, supposed to be specifically, the expected number of new pathogens generated by a single initial pathogen in host body that has survived interaction with activated existing immune cells response to replicate. Using method of next-generation matrix to compute R_0 of the model by obtaining (F) matrix of new infection terms and (V) matrix of decay, loss and transition of compartments terms of the model. Jacobian of each matrix to be obtained and evaluated at equilibrium point E_1 as in [4, 15, 39]. This failed to give us required inverse matrix for V, hence, the model was only decomposed into F and V, only through the P compartment that has new infected terms, as in [2, 7]. Therefore the model no basic reproduction number R_0.

From the pathogens equation (1), it can be mathematically proved that if $1 + \tau P \to 1$, then $\frac{dP}{dt} = rP - \phi PH$, where $F(P,H) = rP$, meaning the rate of new pathogens appearance r, and $V(P,H) = -\varphi PH$, which is the rate of pathogens removal by the immune cells response φ.

However, the immune cells response equation (2) does not contribute to F or V, because it does not contain required terms for that and H is not an infected compartment in the framework of the method, it is only a response compartment as in [2].

Hence, the model has no susceptible-host compartment, also it has no pathogen removal term independent of H, therefore, we can only derive a threshold or reproduction quantity for the immune cells' response, as follows:

From (10), the per-capita growth rate of the immune cells is $\frac{\theta P}{1+\tau P} - \alpha$. We denote the natural threshold quantity as $R_H(P)$, so that

$$R_H(P) = \frac{\theta P}{\alpha(1+\tau P)}. \tag{17}$$

Since, $\frac{dH}{dt} > 0 \iff R_H(P) > 1$, so that

- $R_H(P) > 1 \Rightarrow$ immune cells increase.
- $R_H(P) < 1 \Rightarrow$ immune cells decrease.
- $R_H(P) = 1 \Rightarrow$ immune cells threshold

Immune cells threshold $R_H(P) = 1 \Longrightarrow \frac{\theta P}{\alpha(1+\tau P)} = 1$, this gives $P(\theta - \alpha\tau) = \alpha$.

So that the threshold pathogens level is $P_\tau = \frac{\alpha}{\theta - \alpha\tau}$ which is only true if $\theta > \alpha\tau$. For the non-zero equilibrium, we consider E_2, it is clear the threshold condition of this model at the positive pathogens-immune cells response equilibrium, r and φ affect the equilibrium size, but $\theta > \alpha\tau$ does not depend on them.

From $\theta > \alpha\tau$, we obtained $\frac{\theta}{\alpha\tau} > 1$, which implies $R_{th} = \frac{\theta}{\alpha\tau}$, meaning $R_{th} > 1 \Longleftrightarrow \theta > \alpha\tau$. Therefore, we obtain threshold quantity R_{th}, so that $R_{th} < 1$, means $\theta < \alpha\tau$, which implies the positive pathogen-immune cells response equilibrium does not exist. Observe also that, r and φ do not appear in the threshold, they determine the equilibrium level of the immune cells' response, while θ, α, τ determine the possibilities of overcoming loss or saturation in immune cells response threshold.

On the other hand, we see that: $P^* = \frac{\alpha}{\theta - \alpha\tau}$ from the endemic equilibrium point E_2, meaning for $P^* > 0$ to be true, then $\theta - \alpha\tau > 0$, which gives $\theta > \alpha\tau$, dividing by $\alpha\tau$, we obtain $\frac{\theta}{\alpha\tau} > 1$. This implies that $R_{th} = \frac{\theta}{\alpha\tau}$. Hence, the threshold quantity for the model's positive pathogens-immune cells response interaction equilibrium is $\frac{\theta}{\alpha\tau}$.

Similarly, from $\frac{dH}{dt} = H\left(\frac{\theta P}{1+\tau P} - \alpha\right)$,

The immune cells per heads growth rate becomes $\frac{1}{H}\frac{dH}{dt} = \frac{\theta P}{1+\tau P} - \alpha$ as the pathogens concentration P tends to infinity,

$$\lim_{P\to\infty} \frac{\theta P}{1+\tau P} = \frac{\theta}{\tau}$$

The maximum immune cells production rate is $\frac{\theta}{\tau}$, while the removal rate of the immune cells is α.

➢ *Endemic Equilibrium State*

We substitute E_2 into equation (11), so that

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26nov539

$$J(E_2) = \begin{bmatrix} r - \frac{\alpha\tau r}{\theta} & -\frac{\alpha\phi}{\theta} \\ \frac{r(\theta-\alpha\tau)}{\phi} & 0 \end{bmatrix}. \quad (18)$$

Characteristic equation of $J(E_2)$ is

$$\lambda^2 - \frac{\alpha\tau r\lambda}{\theta} + \frac{\alpha r(\theta - \alpha\tau)}{\theta} = 0. \quad (19)$$

The trace of equation (19), (Jacobian matrix of the model at $J(E_2)$) is $\frac{\alpha\tau r}{\theta}$, while its determinant is $\frac{\alpha r(\theta-\alpha\tau)}{\theta}$.

The local stability of endemic equilibrium state of the model is unstable, because trace of $J(E_2) > 0$ and the determinant is $J(E_2) > 0$. For the local stability of endemic equilibrium state of the model to asymptotically stable, we must get trace of $J(E_2) < 0$. Biologically this can be achieved if α which is the death rate of immune cells is negative (meaning the cells are not reducing). This leads to getting $\theta < \alpha\tau$.

Now, $\theta < \alpha\tau$ means pathogens load can exist or persist in the body. If rate of activating, stimulating and triggering immune cells response is less than product of "time taken by immune cells to handle pathogen" and "rate of death of immune cells". Therefore, the following conditions surface:

- $R_{th} < 1$ infection cannot occur, since pathogens load cannot exist or persist in the body.
- $R_{th} > 1$ infection can occur; pathogens load can exist and persist in the body.
- $R_{th} = 1$ presence of pathogen may or may not occur.

Values provided in (1) table 1, are based on calculations using baseline values from literature. No specific values for the parameters, but we range from literature to arrive at an average accepted value for the parameters.

III. CONCLUSION

The model was formulated with a system of equations that are perfectly symmetric, so that increase in pathogen load and replication, consequently increase risk of infection, but increase in immune cells response and its killing capacity, reduce or clear the invaders (pathogens). The model therefore fascinate a concept that describe a critical transition of how the infection may behave. A natural threshold quantity $R_H(P)$ which is similarly denoted as R_{th}, was obtained as analogous to basic reproduction number (R_0). The quantity was used to describe the expected new pathogens that can be generated by single initial pathogen in the presence of immune cells response, after their interaction. Predictions of possible infection were successful established. Generally, the model showed that, when $H > P$, with $\theta > \alpha\tau$ and $R_{th} > 1$, then a successful clearance of initial pathogens that invaded will surfaced, hence, the model established chance of controlling the pathogens.

We established existence of unique endemic equilibrium state when $\theta > \alpha\tau$ at E_2, which is locally unstable, but asymptotically stable when α is negative, signifying decay of immune cells, thereby triggering infection. Finally, this is generalized model of pathogens and immune cells response, specified diseases can be model using their exact pathogen load and specific immune response to those pathogens.

Table 1 Approximate Baseline Values of Parameters used in the Model.

Parameters	Biological definition	Typical baseline values	Common Units	Source
r	Rate of increment or growth of pathogen	0.02–2.5	Per hour	[35]
φ	Rate of pathogen elimination by immune cells	$0.3–0.5 \times 10^6$	Per hour	Calculated from [37]
θ	Rate of immune activation and proliferation	0.05–0.1	Per hour	Calculated from [34]
α	Natural death/decay of immune cells	0.0002–0.185	Per hour	Calculated from [23]
τ	Clearance time/delay time for immune action	0.00595–0.167	Per hour	Calculated from [13]

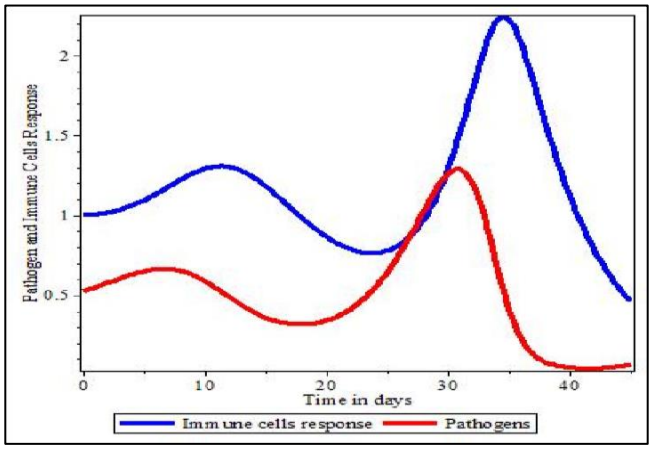


Fig 1 Full Body Immune Cells Response Keeps Pathogen Always Low, Even at 0.5 Rate of Pathogen, with $H > P$, $\theta > \alpha\tau$ and $R_{th} > 1$ if $\alpha = 0.2$, $\theta = 0.5$, $\tau = 0.595$, $\varphi = 0.6$, $r = 0.5$, $P_0 = 0.526$ and $H_0 = 1$. It implies infection can be prevented by immune cells response.

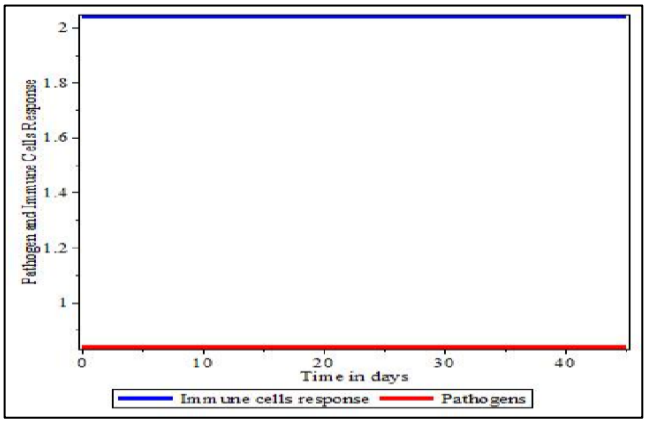


Fig 2 Doubling Immune Cells Response Can Keep it High Above even 0.8 Rate of Pathogens, with $H > P$, $\theta > \alpha\tau$ and $R_{th} > 1$ if $\alpha = 0.07$, $\theta = 0.5$, $\tau = 5.95$, $\varphi = 0.0022$, $r = 0.00075$, $P_0 = 0.838$ and $H_0 = 2.04$. This will Perfectly Make Immune Cells Response Control any Pathogen and Causing Infection.

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26nov539

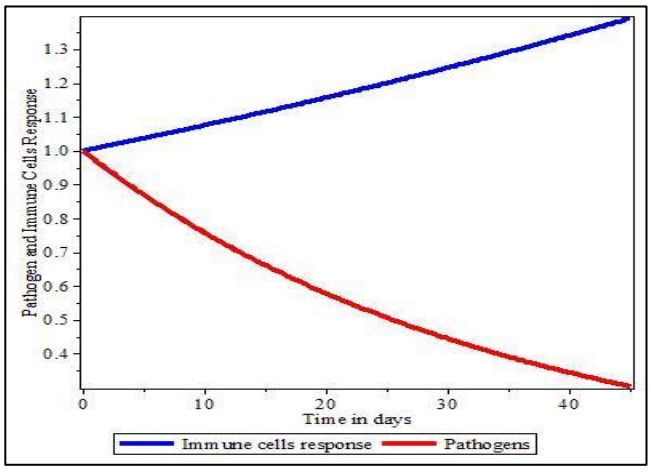


Fig 3 Equal Rates of Immune Cells Response and Pathogen Can Make the Immune Cells Response Over Power the Pathogen, with $H = P$, $\theta < \alpha\tau$ and $R_{th} < 1$ if $\alpha = 0.1$, $\theta = 0.5$, $\tau = 5.95$, $\varphi = 0.0022$, $r = 0.0075$, $P_0 = 1$ and $H_0 = 1$. This is by Chance, it Does Not Obviously Happen in Many Cases.

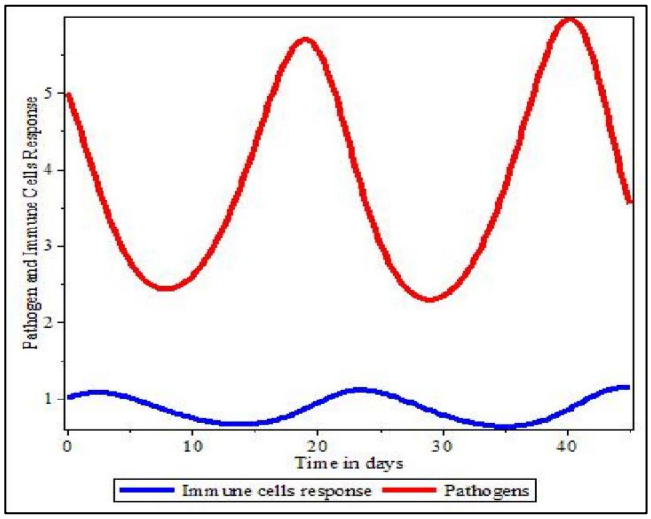


Fig 4 Higher Pathogen Rate will Over Power Immune Cells Response, with $H < P$, $\theta > \alpha\tau$ and $R_{th} > 1$ if $\alpha = 0.185$, $\theta = 0.05$, $\tau = 0.00595$, $\varphi = 0.6$, $r = 0.5$, $P_0 = 5$ and $H_0 = 1$.

ACKNOWLEDGMENT

This paper is part of the research project funded by *TETFund*, AFUSTA's, Institution Based Research (IBR), The authors would like to thank Abdullahi Fodio University of Science and Technology, Aliero.

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