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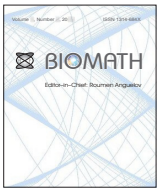

Impact of Perfect Drug Adherence on Immunopathogenic Mechanism for Dynamical System of Psoriasis

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***Abstract*—Psoriasis is a frequent autoimmune chronic skin disease differentiated by T-Cells agreeable hyperproliferation of epidermal Keratinocytes. The feature of T-Cells held up Psoriatic scratches is the epidermal penetration of basically oligoclonal $CD8^+$ T-Cells and also of $CD4^+$ T-Cells in the dermis. Psoriatic lesions are sharply distinguished, red and enlarged scratches together with whitish silver scales. In this research article, we propose a mathematical depiction for Psoriasis, involving a set of differential equations, regarding T-Cells, Dendritic Cells, $CD8^+$ T-Cells and epidermal Keratinocytes. Here, we specially introduce the interaction between Dendritic Cells and $CD8^+$ T-Cells to monitor the impact of this interaction upon the system dynamics. We also analyze the mathematical model both in presence and absence of effectiveness of two drugs. We study the system analytically and numerically to comprehend the significance of effectiveness of the drugs, integrated in the model system. Here, we reduce the Keratinocyte population to restrict Psoriasis by applying the combination of two drugs and able to enlighten the perspective of the disease dynamics for Psoriasis.**

***Keywords*-T-Cells; Dendritic Cells; $CD8^+$ T-Cells; Keratinocytes; MHC; pMHC; T-Cells Receptor; Dermis; Epidermis; Lymphocytes; Monocytes; Neutrophils; Cytokines; Drug Efficacy**

I. Introduction

In spite of precise fundamental and experimental studies for more than a few decades, many queries continue relating to Psoriasis. Inflammatory tissues respond along with enormous influxes of T-Cells and Dendritic Cells (Nickoloff, 2000). A "Perfect Cytokine Storm" is produced through this multicellular scheme that synchronizes the cellular attack and links mutually with connection of both soluble intermediaries and cellular ingredients (Uyemura et. al., 1993, Nickoloff and Nestle, 2004) [1]. Psoriasis has been measured as a dermatological chaos, in which T-Cells and epidermal Keratinocytes perform a relevant pathogenic function. DCs play an essential role in pathogenesis of Psoriasis by attending antigens throughout principal major histo compatibility (MHC) complex II molecules [2]. Psoriasis is observed as a widespread inflammatory skin chaos with an inherited contact. It is illustrious through epidermal hyperplasia by means of cellular diffusion of Lymphocytes, Monocytes and Neutrophils [3]. Local production of T-Cells is observed as a significant immunological constituent of Psoriatic lesions. The enormous numbers of Dendritic Cells below the hyperplastic epidermis, are surrounded by T-Cells within the Psoriatic plaques [4]. Roy and Bhadra [5] have clarified that, suppression made on Dendritic Cells will reduce the expansion of Keratinocytes and will give better effect than suppression made on T-Cells. For suppression made on T-Cells, the pathogenesis continues due to auxiliary basis in presence of DCs, as the suppression on DCs presents a superior result. In our very recent work, we have formed a set

of differential equations to exhibit a course of stable connection to the growth of epidermal Keratinocytes through negative feedback control, that is comparable to the favorable drug management. We also integrate a time delay in our model to furnish the time from creation of T-Cells and DCs to the enhancement of epidermal Keratinocytes [6]. In our present research article, we introduce $CD8^+$ T-Cells population which interacts with DCs in the dynamical system. This interaction leads to generate Keratinocytes, which in turn supports to expand the Keratinocytes growth. Cyclosporin and FK506 are applied as drugs, that perform to restrict Psoriasis [7]. To confine this growth, we apply drug at the interaction between $CD8^+$ T-Cells and DCs. Another method to create Keratinocytes is the interaction between T-Cells and Keratinocytes itself. Here, we also set the drug in that interaction to control the growth of Keratinocytes, whose surplus production directs to create Psoriasis. In this article, we study the effectiveness of two drugs on the cell biological scheme to build a comparative analysis for the drugs to restrain the disease.

II. The Basic Assumptions and Formulation of the Mathematical Model

We consider the mathematical model of Psoriasis to describe the dynamical cell biological system. Let us assume $l(t)$, $m(t)$, $c(t)$ and $k(t)$ to represent the densities of T-Cells, Dendritic Cells, $CD8^+$ T-Cells and epidermal Keratinocytes correspondingly at a specific time t to attain a set of differential equations.

In the region proximity, the accumulation of T-Cells is considered at a constant rate a and the accumulation of Dendritic Cells is taken at a constant rate b at the appropriate regime. It is assumed that, the rate of activation of T-Cells by DCs is δ and β is the rate of activation of DCs by T-Cells. Expansion of Keratinocytes density is taken to be proportional to the production of T-Cells and DCs densities with a rate η. The rate of activation of Keratinocytes by T-Cells due to T-Cells mediated Cytokines is referred as γ_1 and γ_2 is the rate at which growth of Keratinocytes takes place. The per capita removal rate of T-Cells is denoted by μ and μ' is the per capita removal rate of Dendritic Cells throughout normal procedure. The premature Dendritic Cells turn into mature in the course of some cell biological procedures and move into the lymph node. In that lymph node, the mature DCs interrelate with $CD8^+$ T-Cells at a rate qn, where q is the average peptide specific T-Cells Receptor (TCR) and n is the average number of the related pMHC complexes per DCs and this contact gives a negative effect to DCs as well as positive effect to $CD8^+$ T-Cells. The $CD8^+$ T-Cell proliferation is stimulated by similar antigen presenting DCs at a rate r. We assume here also that, α is the rate of interaction between DCs and $CD8^+$ T-Cells. It gives negative impact to $CD8^+$ T-Cell population. In addition, Keratinocytes are produced through interaction between DCs and $CD8^+$ T-Cells at a rate α_1. Again, we assume ξ and λ as the per capita removal rate of $CD8^+$ T-Cells and epidermal Keratinocytes respectively. All the parameters, described above, are always positive. Here, we assimilate the combination of two drug efficacy parameters u_1 and u_2, placed between the interaction of T-Cells and epidermal Keratinocytes and Dendritic Cells and $CD8^+$ T-Cells respectively to restrain the growth of epidermal Keratinocytes, whose excess production is one of the main reasons to form Psoriasis.

Accumulating collectively the above assumptions, we can formulate the mathematical model given below:

$$\begin{aligned}
\frac{dl}{dt} &= a - \delta lm - \gamma_1 lk(1-u_1) - \mu l,\\
\frac{dm}{dt} &= b - \beta lm - qnmc - \mu' m,\\
\frac{dc}{dt} &= rqnmc - \alpha mc(1-u_2) - \xi c, \qquad (1)\\
\frac{dk}{dt} &= \eta lm + \gamma_2 lk(1-u_1) + \alpha_1 mc(1-u_2) - \lambda k,
\end{aligned}$$

where $l(0) > 0$, $m(0) > 0$, $c(0) > 0$ and $k(0) > 0$ at a specific time period t.

The communication is organized as follows: We comprise the general outlook and discuss about the effectiveness of drugs on the cell biological system of Psoriasis in section I. In section II, we represent the mathematical model of Psoriasis including basic assumptions. Section III describes theoretical analysis of the model system (1). This section is also integrated with two equilibrium points of the system dynamics. Theoretical explanation of the model parameters, centering on its stability and associated features are discussed in the same section. In section IV, we include results from numerical simulation of the system and finally section V ends with the conclusion of the model dynamics.

III. Local Stability Analysis for the System

The RHS of the equation (1) is a smooth function of $l(t)$, $m(t)$, $c(t)$ and $k(t)$ and also the parameters, as long as these quantities are non-negative. For that reason, local existence and uniqueness properties hold in the positive octant.

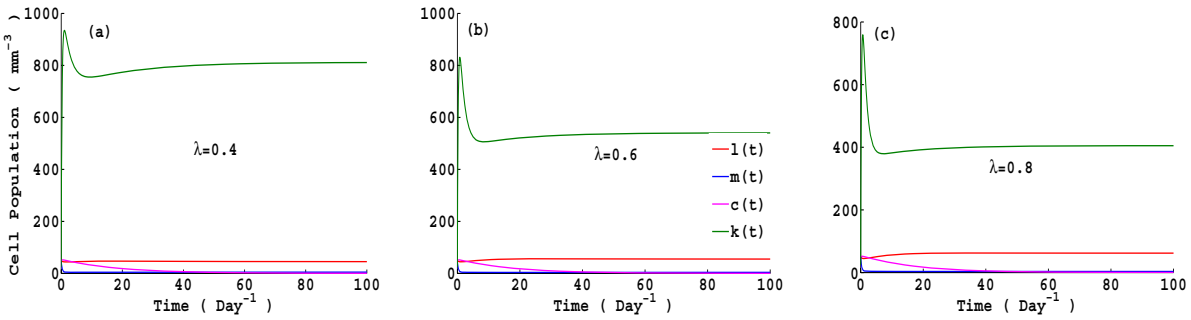

Fig. 1. Behaviors of different cell biological masses of the system (1) with u_1=0.5 and u_2=0.7 for $\lambda = 0.4$ (Panel a), $\lambda = 0.6$ (Panel b) and $\lambda = 0.8$ (Panel c), keeping other parameters at their standard values as in Table 1.

A. Equilibria of the Model System

The model equation (1) has two equilibrium points, i.e., $\tilde{E}(\tilde{l}, \tilde{m}, 0, \tilde{k})$ and $E^*(l^*, m^*, c^*, k^*)$. Now, $\tilde{m}$=$\frac{b}{\beta\tilde{l}+\mu'}$, $\tilde{k}$=$\frac{a-\delta\tilde{l}\tilde{m}-\mu\tilde{l}}{\gamma_1\tilde{l}(1-u_1)}$ and $\tilde{l}$ is the positive root of the equation

$$A\tilde{l}^3 - B\tilde{l}^2 + C\tilde{l} + D = 0, \qquad (2)$$

where

$A = \beta\delta\gamma_1\gamma_2\tilde{m}(1-u_1) + \beta\gamma_1\gamma_2\mu(1-u_1) > 0,$
$B = a\beta\gamma_1\gamma_2(1-u_1) + b\eta\gamma_1^2(1-u_1) + \delta\gamma_1\tilde{m}(\beta\lambda - \gamma_2\mu') + \gamma_1\mu(\beta\lambda - \gamma_2\mu') + \gamma_1\gamma_2\mu' u_1(\delta\tilde{m} + \mu) > 0,$
$C = a\gamma_1(\beta\lambda - \gamma_2\mu') + \gamma_1\mu'(a\gamma_2 u_1 - \delta\lambda\tilde{m} - \mu\lambda) > 0,$
$D = a\gamma_1\mu'\lambda > 0.$

This cubic equation (2) has positive real root if the coefficients of $\tilde{l}^3$, $-\tilde{l}^2$ and $\tilde{l}$ are positive. Now, considering Descartes' rule of sign, we may conclude that the equation $A\tilde{l}^3 - B\tilde{l}^2 + C\tilde{l} + D = 0$ has two positive real roots (multiplicities of roots are adequate) [8] if and only if the following conditions are hold:

(i) $\beta\lambda > \gamma_2\mu'$ and (ii) $a\gamma_2 u_1 > \lambda(\delta\tilde{m} + \mu)$.

From the second equation of system (1), we include $\tilde{m}$ is always positive by our necessary assumptions. From the first equation of system (1), we state that $\tilde{k}$ is realistic if $a > \tilde{l}(\delta\tilde{m} + \mu)$. As a result, if (i) and (ii) are persuaded, then we may bring to an end that, the equation (2) has two positive real roots and henceforth positive equilibrium point $\tilde{E}(\tilde{l}, \tilde{m}, 0, \tilde{k})$ of the system (1) exists. Finally, for the interior equilibrium point $E^*(l^*, m^*, c^*, k^*)$, l^*, m^*, c^* and k^* are the non-trivial solutions of the model equation (1).

Remark 1. The system (1) exists if the two conditions are hold, (a) the product of the rate of activation of DCs by T-Cells and the per capita removal rate of Keratinocytes should be greater than the product of the rate of growth of Keratinocytes due to T-Cells mediated Cytokines and the per capita removal rate of DCs and (b) the rate of accumulation of T-Cells itself and the product of the rate of accumulation of T-Cells, the rate of growth of Keratinocytes due to T-Cells mediated Cytokines and the first drug efficacy parameter must be greater than a pre-assigned positive quantity.

The characteristic equation of the matrix related to the equilibrium point $\tilde{E}(\tilde{l}, \tilde{m}, 0, \tilde{k})$ in presence of effectiveness of both drugs ($u_1 = u_2 = 1$) is illustrated by,

$$(-\lambda - \phi)(rqn\tilde{m} - \xi - \phi)[\phi^2 - (trace\ V)\phi + det\ V] = 0,$$

where $trace\ V = -(\beta\tilde{l} + \delta\tilde{m} + \mu + \mu') < 0$ and $det\ V = \beta\mu\tilde{l} + \delta\mu'\tilde{m} + \mu\mu' > 0$.

Now, ϕ_1 (=$-\lambda$) is always negative, ϕ_2=$rqn\tilde{m} - \xi$ and the roots of the equation $\phi^2 - (trace\ V)\phi + det\ V = 0$ are negative since $trace\ V < 0$ and $det\ V > 0$. Hence the equilibrium point $\tilde{E}(\tilde{l}, \tilde{m}, 0, \tilde{k})$ in presence of effectiveness of both drugs is stable only if $\tilde{m} < \frac{\xi}{rqn}$.

Remark 2. The CD8$^+$ T-Cells free equilibrium point in presence of effectiveness of both drugs is stable if DC population is less than some pre-determined positive value.

The characteristic equation of the matrix related to the equilibrium point $\tilde{E}(\tilde{l}, \tilde{m}, 0, \tilde{k})$ in absence of effectiveness of both drugs ($u_1 = u_2 = 0$) is furnished by,

$$(rqn\tilde{m} - \alpha\tilde{m} - \xi - \psi)(\psi^3 + A_1\psi^2 + A_2\psi + A_3) = 0.$$

Here, ψ_1=$rqn\tilde{m} - \alpha\tilde{m} - \xi$ and from Routh-Hurwitz criterion, $A_1 > 0$ if $\beta > \gamma_2$, $A_3 > 0$ if $\eta\gamma_1 > \delta\gamma_2$, $\beta\lambda > \gamma_2\mu'$ and $\frac{\tilde{k}}{\tilde{l}} > \frac{\gamma_2\mu}{\gamma_1\lambda}$ and $A_1A_2 - A_3 > 0$ if $\beta > \gamma_2$. Thus the equilibrium point $\tilde{E}(\tilde{l}, \tilde{m}, 0, \tilde{k})$ in absence of effectiveness of both drugs is stable if $\tilde{m} < \frac{\xi}{rqn-\alpha}$, $\beta >$max$[\gamma_2, \frac{\gamma_2\mu'}{\lambda}]$ and $\frac{\gamma_2}{\gamma_1} <min[\frac{\eta}{\delta}, \frac{\lambda\tilde{k}}{\mu\tilde{l}}]$, provided $rqn > \alpha$.

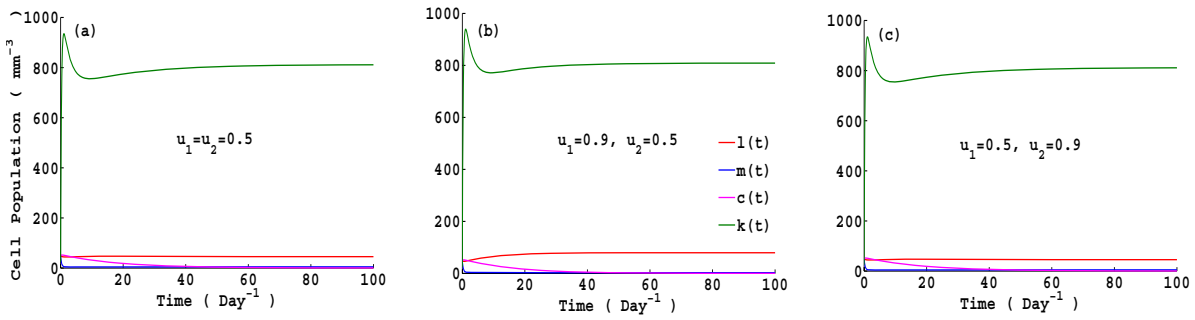

Fig. 2. Behaviors of different cell biological masses of the system (1) for different values of two drug efficacy parameters u_1 and u_2, keeping other parameters at their standard values as in Table 1.

Remark 3. The CD8$^+$ T-Cells free equilibrium point in absence of effectiveness of both drugs is stable if (1) DC population is less than some pre-assigned positive quantity, (2) the rate of activation of DCs by T-Cells should be always greater than the maximum of $[\gamma_2, \frac{\gamma_2\mu'}{\lambda}]$ and (3) the ratio of γ_2 and γ_1 should be always less than the minimum of $[\frac{\eta}{\delta}, \frac{\lambda\tilde{k}}{\mu\tilde{l}}]$.

Also we study another two cases, i.e., first drug (u_1) is present and second drug (u_2) is absent and vice-versa in the system dynamics. The characteristic equation of the matrix related to the equilibrium point $\tilde{E}(\tilde{l},\ \tilde{m},\ 0,\ \tilde{k})$ in presence of effectiveness of first drug ($u_1 = 1$) and absence of effectiveness of second drug ($u_2 = 0$) is illustrated by,

$$(-\lambda-\varphi)(rqn\tilde{m}-\alpha\tilde{m}-\xi-\varphi)[\varphi^2-(trace\ W)\varphi + det\ W] = 0,$$

where $trace\ W = -(\beta\tilde{l}+\delta\tilde{m}+\mu+\mu') < 0$ and $det\ W = \beta\mu\tilde{l}+\delta\mu'\tilde{m}+\mu\mu' > 0$.

Now, φ_1 (=$-\lambda$) is always negative, φ_2=$rqn\tilde{m}-\alpha\tilde{m}-\xi$ and the roots of the equation $\varphi^2-(trace\ W)\varphi+det\ W = 0$ are negative since $trace\ W < 0$ and $det\ W > 0$. Hence the equilibrium point $\tilde{E}(\tilde{l},\ \tilde{m},\ 0,\ \tilde{k})$ in presence of effectiveness of first drug ($u_1 = 1$) and absence of effectiveness of second drug ($u_2 = 0$) is stable only if $\tilde{m} < \frac{\xi}{rqn-\alpha}$, provided $rqn > \alpha$.

Remark 4. The CD8$^+$ T-Cells free equilibrium point in presence of effectiveness of first drug and absence of effectiveness of second drug is stable if DC population is less than some pre-determined positive value, provided the product of the rate at which CD8$^+$ T-Cell proliferation is stimulated by antigen presenting DCs, average peptide specific T-Cells Receptor (TCR) and average number of the related pMHC complexes per DCs is greater than the rate of interaction between DCs and CD8$^+$ T-Cells.

The characteristic equation of the matrix related to the equilibrium point $\tilde{E}(\tilde{l},\ \tilde{m},\ 0,\ \tilde{k})$ in absence of effectiveness of first drug ($u_1 = 0$) and presence of effectiveness of second drug ($u_2 = 1$) is demonstrated by,

$$(rqn\tilde{m}-\xi-\chi)(\chi^3+B_1\chi^2+B_2\chi+B_3) = 0.$$

Here, χ_1=$rqn\tilde{m}-\xi$ and from Routh-Hurwitz criterion, we obtain $\beta > \gamma_2$, $\eta\gamma_1 > \delta\gamma_2$, $\beta\lambda > \gamma_2\mu'$ and $\frac{\tilde{k}}{\tilde{l}} > \frac{\gamma_2\mu}{\gamma_1\lambda}$. Thus the equilibrium point $\tilde{E}(\tilde{l},\ \tilde{m},\ 0,\ \tilde{k})$ in absence of effectiveness of first drug ($u_1 = 0$) and presence of effectiveness of second drug ($u_2 = 1$) is stable if $\tilde{m} < \frac{\xi}{rqn}$, $\beta >$max$[\gamma_2, \frac{\gamma_2\mu'}{\lambda}]$ and $\frac{\gamma_2}{\gamma_1} <min[\frac{\eta}{\delta}, \frac{\lambda\tilde{k}}{\mu\tilde{l}}]$.

Remark 5. If (1) DC population is less than some pre-assigned positive quantity, (2) the rate of activation of DCs by T-Cells should be always greater than the maximum of $[\gamma_2, \frac{\gamma_2\mu'}{\lambda}]$ and (3) the ratio of γ_2 and γ_1 should be always less than the minimum of $[\frac{\eta}{\delta}, \frac{\lambda\tilde{k}}{\mu\tilde{l}}]$, then the CD8$^+$ T-Cells free equilibrium point in absence of effectiveness of first drug and presence of effectiveness of second drug is stable.

Now, we analyze the roots of the characteristic equation of the matrix related to the interior equilibrium point in presence of effectiveness of both drugs and study it's stability analysis. We discard the cases where (a) effectiveness of both drugs are absent and (b) one is present and other is absent and vice-versa, as they are not realistic for the interior equilibrium in true sense. The characteristic equation is furnished by,

$$(-\lambda-\tau)(\tau^3+C_1\tau^2+C_2\tau+C_3) = 0.$$

Here, τ_1 (=$-\lambda$) is always negative and from Routh-Hurwitz criterion, we have $\delta > rqn$ and $m^* < \frac{\xi}{rqn}$. Hence the interior equilibrium point $E^*(l^*,\ m^*,\ c^*,\ k^*)$ in presence of effectiveness of both drugs ($u_1 = u_2 = 1$) is stable if $rqn <$min$[\delta, \frac{\xi}{m^*}]$.

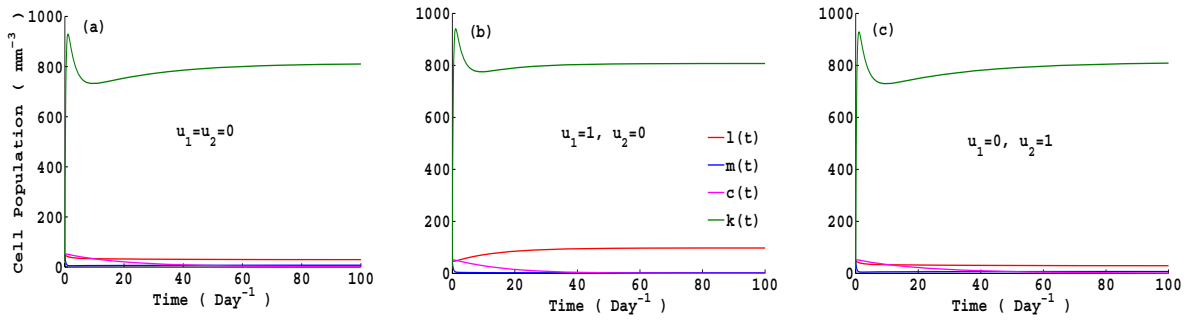

Fig. 3. Behaviors of different cell biological masses of the system (1) for perfect absence and presence of two drug efficacy parameters u_1 and u_2, keeping other parameters at their standard values as in Table 1.

Remark 6. The interior equilibrium point in presence of effectiveness of both drugs is stable if the product of the rate at which $CD8^+$ T-Cell proliferation is stimulated by antigen presenting DCs, average peptide specific T-Cells Receptor (TCR) and average number of the related pMHC complexes per DCs should be less than the minimum of $[\delta, \frac{\xi}{m^*}]$.

IV. Numerical Simulation of the System

In the previous section, we have initiated analytical techniques for qualitative study of the system with effectiveness of two drugs. In this part, we perform numerical simulation of the model system (1). We approximate the parameters in the course of our analytical outcomes and conditions. Numerical values of the model parameters, used in our numerical simulation, have been specified in Table 1.

Table 1. Parameters used in the model equation (1)

Parameter	Default Values Assigned
a	9 mm^{-3} Day^{-1} [6]
b	14 mm^{-3} Day^{-1} [6]
δ	0.01 mm^3 Day^{-1} [6]
β	0.065 mm^3 Day^{-1} [6]
η	1.5 mm^3 Day^{-1} [6]
γ_1	0.0002 mm^3 Day^{-1} [6]
γ_2	0.0001 mm^3 Day^{-1} (estimated)
r	0.9 Day^{-1} (estimated)
q	0.001 Day^{-1} (estimated)
n	7 (estimated)
α	0.0007 mm^3 Day^{-1} (estimated)
α_1	0.0005 mm^3 Day^{-1} (estimated)
μ	0.07 Day^{-1} (estimated)
μ'	0.002 Day^{-1} [6]
ξ	0.08 Day^{-1} (estimated)
λ	0.4 Day^{-1} [6]

We are trying to monitor the cell behavioral patterns of different cells, involved in the system dynamics for variation in the values of the parameters. Firstly, we set $u_1 = 0.5$ and $u_2 = 0.7$ and assume the value of the decay rate of Keratinocytes (λ) as 0.4 Day^{-1}, then we notice that, Keratinocytes initially increase around 900 cells/mm^3, decline slightly just below 800 cells/mm^3 and finally become stable, displayed in Fig. 1(a). When the value of λ is 0.6 Day^{-1}, Keratinocytes raise up to 800 cells/mm^3, after that decrease below 600 cells/mm^3, at last turn into stable, shown in Fig. 1(b). Finally, considering the value of λ as 0.8 Day^{-1}, Keratinocytes increase below 800 cells/mm^3, next decrease at about 400 cells/mm^3 and lastly develop into stable in nature, portrayed in Fig. 1(c). Thus we conclude that, due to increase in the value of decay rate of Keratinocytes, its population must be decreased. Next, we monitor the performances of four different cells in the system for alteration in the values of two drug efficacy parameters (u_1 and u_2). At Fig. 2(a), we consider u_1 and u_2 are both as 0.5. When the value of u_1 is increased by 0.4 at Fig. 2(b) (i.e., $u_1 = 0.9$), then we study that, T-Cells are increased than the earlier case. Again, when u_1 is decreased to 0.5, the behavior of T-Cells at Fig. 2(c) is rolled back like Fig. 2(a). Besides, we notice that, Keratinocytes arrive quicker in the direction of the stable region than for the lower value of effectiveness of first drug. No specific change is occurred for the presence and increase of effectiveness of second drug in the system. At Fig. 3(a), we assume perfect absence of effectiveness for both the drugs, effectiveness of first drug is perfectly present and effectiveness of second drug is absent perfectly, illustrated at Fig. 3(b) and vice-versa at Fig. 3(c). The increasing and decreasing natures of T-Cells are repeated as like Fig. 2 for the perfect absence and presence of effectiveness of two drugs. There is not at all any significant change in the behavioral pattern, observed in DC and $CD8^+$ T-Cell

population for variation in the values of the system parameters and also for the change (increase or decrease) in the values of two drug efficacy parameters.

V. Conclusion

The effectiveness of first drug has an imperative effect rather than the second drug upon our proposed model. In presence of first drug, the system develops into stable condition very smoothly. There is not at all any significant difference between absence and presence of second drug. Thus the second drug has not any significant impact on the interaction between DCs and $CD8^+$ T-Cells, which are not influenced for change in the value of the model parameters. They maintain the same pattern, which is independent of any model parameter of the system. It is numerically revealed that, increase in the value of decay rate of Keratinocytes leads to the decrease in Keratinocytes and hence increase in T-Cells is occurred simultaneously. Enhancing in the value of decay rate of Keratinocytes has no impact on DCs and $CD8^+$ T-Cells. With the increasing value of decay rate of Keratinocytes, population of Keratinocyte is decreased and thus T-Cell population is gradually increased. Increasing in the value of u_1 forwards to the increase in T-Cells, because increase in the value of u_1 reduces Keratinocytes and thus T-Cells are increased. Keratinocytes reach stable situation more rapidly with the increase in the value of first drug efficacy parameter. But unfortunately, the second drug has not any significant effect on the system, specially on the interaction between DCs and $CD8^+$ T-Cells and thus the second drug does not take part to control the Keratinocyte population in broad sense. We may also predict another important feature that, decrease in Keratinocytes provides increase in T-Cells but increase in T-Cells does not essentially lead to decrease in Keratinocytes or the decrease is not too prominent to observe. We have used the combination of two drugs to get enhanced result but from analytical and numerical points of view, the first drug has the significant effect compared to the second drug on our proposed mathematical model for Psoriasis. Hence, to put the drug in the interaction between DCs and $CD8^+$ T-Cells may not be successful enough to restrict the expansion of Keratinocytes growth. Thus applying the drug at the place of interaction between T-Cells and DCs on Psoriatic patients in a systematic way would be able to obtain the improved outcome for better cure of the disease Psoriasis.

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