Transmission Intensity And Impact Of Control On Skin Diseases Due To Temperature Change

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Abstract: Skin is the largest covering of human body. It is sensitive to changes in the temperature. Daytime variation in ambient temperature affects the skin as it is the contact medium between environment and human body. This notable, sometime high variation in environmental temperature is due to raise in global warming results the skin diseases. Recovery of such disease is less permissible. This model is formulated for applying Z-type control to the system of non-linear differential equations. Threshold value reveals the how much skin diseases transmit. With essential conditions, stability analysis is worked out for every equilibrium point. Periodicity depicts the chaotic oscillations on each compartment because of reinfection in skin disease. Numerical simulation shows the behavior of Z-type control and its impact on model; mainly to control chaotic oscillations and also concludes recovery rate on skin for validating data.

Index Terms: Temperature variations, Saturation function, Threshold, Periodicity, Z-type control, Chaos

1. INTRODUCTION

Sunlight is essential for human life but access of anything is dangerous; so as for sunlight that is effect of high temperature on human skin. From sunrays evaporation in terms of sweating is beneficial but; convection, conduction and radiation from sunlight are the main reasons for skin diseases as skin comes directly in contact with surroundings. Skin disease affects masses of individuals around the world. According to one study (health24), it occurs when immune system of human being reacts to a substance that causes allergic reaction. Now a days, skin diseases also affected by temperature change due to global warming. This global warming affects so much of the environment that temperature change sometimes gives extreme heat and sometime provides cool environment. But both affect human skin. Cold urticarial, chilblains, eczema etc. are skin diseases due to cold temperature while rashes, sunburns, acne etc. type disease suffers due to high temperature. In this model we are dealing with the skin diseases due to high temperature. The behavior of these diseases is chaotic in nature. In technical way, chaos is a disorder whose future scenario cannot be predictable. Usually it is assumed that chaos make troublesome for citizens to boost oneself out of paucity. Through this paper, we examine which parameters are responsible for system to convert into chaos and which parameters can control epidemic behavior of the system. This paper is expressed identical to prey-predator model. In prey-predator model, Z-type control is used to control predator population and chaotic behavior of the system. Hence, the conception of half saturation constant makes out to indicate the interface among two compartments. This model is articulated alike to prey-predator model. Therefore, the concept of half saturation constant is figure out to show the interaction between two compartments. Alzahrani et al. (2018) has studied an eco-epidemiological model by considering prey-predator model by applying Z-type control to control oscillation in disease model. Rokita et al. (2011) analyze the application of thermography for the assessment of allergen-induced skin reactions and gave paper on it through statistical approach. Capon et al. (2003) has developed healing process using thermal lasers and adjustment of growth factors. Kermack et al. (1927) has observed in paper named, a contribution to the mathematical theory of epidemics; that minor change in infectivity gives large epidemics. Jost et al. (1999) gave a new approach about deterministic extinction in ratio-dependent predator-prey models. This paper gave importance and issue regarding the dynamics near origin (0, 0). Martens et al. (1995) find a fact based on vector borne diseases that, epidemicity low or maybe inattentive in less economically developed area and construct an article on climate change and vector-borne diseases: a global modeling perspective. McMichael et al. (1997) considered patterns of air; thermal stresses etc. to observe the result of climate variation on human health and gave an article on global climate change: the potential effects on health. Samanta (2018) et al. has studied two epidemic model by Z-type control approach. In section 2, the model is formulated and by using next generation matrix method threshold value is calculated. Then stability analysis is carried out for each equilibrium points with detailed computation in section 3. Section 4 holds numerical simulation for validated data. Concluding remark has been done in section 5.

2 FORMULATION OF MODEL

This model has been formulated using three states taking as a compartment that is temperature sensitive individuals (T) come to be skin allergic (S_A) due to variation in high temperature and then recovery (R) as a medication is given to the susceptible individuals. Table 1 indicates the parameters and its parametric values which define the rate or flow at which the compartments are connected. The model parameters and state variables are depicted in Table 1.

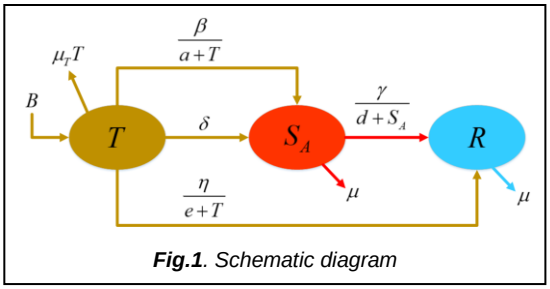

***Fig.1**. Schematic diagram*

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Using parameters defined in diagram and from this flow of diagram we obtain following system of non-linear differential equations:

$$\frac{dT}{dt} = BT - \delta TS_A - \frac{\beta TS_A}{a+T} - \frac{\eta TR}{e+T} - \mu_T T^2$$

$$\frac{dS_A}{dt} = \delta TS_A - \frac{\gamma S_A R}{d+S_A} + \frac{\beta TS_A}{a+T} - \mu S_A$$

(1)

$$\frac{dR}{dt} = \frac{c_1 \gamma S_A R}{d+S_A} + \frac{c_2 \eta TR}{e+T} - \mu R$$

with $T > 0$ *and* $S_A, R \geq 0$

TABLE 1

MODEL PARAMETERS AND STATE VARIABLES

Notation	Parameter Description	Parametric values
B	Growth rate of susceptible individuals who are sensitive to temperature change	0.05
β	Force of infection	0.02
δ	Rate at which individuals get skin allergy occurs	0.05
γ	Recovery rate of allergic individuals	0.6
η	Recovery rate of temperature sensitive individuals	0.4
μ	Escape rate of individuals	0.5
μ_T	Intra-class coefficient	0.2
a,d,e	Half-saturation constants	12,2,4
c_1, c_2	Recovery efficiency of individuals	2,3

Solving this above system we derive three equilibrium points:

(i) $E_0\left(\frac{B}{\mu_T}, 0, 0\right)$

(ii) $E_1\left(r_1, \frac{\mu_T r_1(\beta-\mu) - \mu_T a\mu + B\delta(r_1+a)}{\delta(r_1\delta + a\delta + \beta)}, 0\right)$

(2)

where, $r_1 = RootOf(\delta\_Z^2 + (a\delta + \beta - \mu)\_Z - a\mu)$

(iii) $E^*\left(T^*, S_A^*, R^*\right)$

where,

$$T^* = RootOf[\mu_T(c_1\gamma + c_2\eta - \mu)Z^3 + (\mu_T(c_1\gamma - \mu)(a+e) + c_2\eta)Z^2 + (c_1 a\gamma(\mu_T e - B) + ad\delta\eta(c_1 - c_2) - \mu_T ae\mu - B(c_1 e\gamma + c_2 a\eta) + \eta d(c_1(\beta-\mu) - c_2\beta) + \mu(d\delta + B)(a+e) + \beta d\mu)Z - c_1 a(Be\gamma + d\eta\mu) + e\mu(a(d\delta + B) + \beta d)]$$

Assume $T^* = r_2$

$$S_A^* = -\frac{d(r_2(c_2\eta - \mu) - e\mu)}{c_1\gamma(e+r_2) + r_2(c_2\eta - \mu) - e\mu}$$

$$R^* = (e-r_2)\{-\mu_T c_1^2 a\gamma^2 r_2[\delta e(c_1 a + c_2 e) + c_1(a\mu + \beta e)] + \mu_T c_2 a\eta\mu r_2(-2c_1^2 a\gamma + c_2 e\mu) + e\mu[c_1^2 d\delta e\gamma(\beta-\mu)] - c_2^2 a\eta\mu(B + d\delta) - c_1 d\delta\mu^2(a-e) + \mu(Bc_2 a\mu - c_1\beta d\delta e)]\} + (e+r_2)\{c_1 ae\gamma[\mu_T c_1 r_2(c_1\gamma(\delta e + \mu) + \mu(c_2\eta + 2\beta)) + c_2(\mu_T c_2\eta r_2(a\delta + \beta) - B\delta(c_1 e\gamma + c_2 a\eta)] + Bc_1 a[\beta e\gamma(c_1^2\gamma - c_2^2\eta) + a\mu(\mu^2 + c_2\eta\gamma) - \mu_T a\mu^3 r_2(c_1 a + c_2 e) + c_1 ae\mu^2(\beta - \mu)(B - \mu_T r_2) + (a-e)[c_1 a\delta e\mu^2(B - \mu_T r_2) + (\delta e - \mu)(Bc_1^3 a\gamma^2 - 2c_1^2 a\gamma\mu(B - \mu_T r_2)) + Bc_1^2 c_2 a\eta\gamma\mu]\} + c_1 c_2 ae(\eta - \gamma)\{(e+r_2)[(a\delta + \beta)(\mu_T\mu r_2 + Bc_1\gamma) - \mu_T c_1 a\delta\gamma r_2] + (a-e)\mu_T\mu^2 r_2 + (\beta d\mu/a)[e(\mu - \delta r_2) - (a\mu + \beta e)] + a\mu[(B + d\delta)(c_2\eta - \delta e - \mu)] + d\delta\mu[2e(\mu-\beta) - a\mu]\} + c_1 c_2 aer_2(\eta+\gamma)\{-\mu_T c_1[\beta\gamma(e+r_2) - c_2 a\eta\mu - \mu(\mu_T\mu r_2 + Ba\delta)]\} + c_1 c_2^2 a\eta\mu r_2[-\mu_T r_2(a\eta + e\gamma) + B(a\eta - e\gamma)] + c_2 e\mu(a+r_2)\{(\mu_T r_2 - B)[c_1\delta e^2\gamma + c_2 a\eta(c_2\eta - \delta e)] - c_2 ad\delta\eta(c_2\eta - \delta e)\} + (a-e)\{c_1 d\gamma\mu[c_2\delta e(c_1 a\eta + \delta e r_2) - c_1 a\mu^2] + e\mu(a+r_2)[c_1 d\delta^2 e(\mu - c_1\gamma) + c_2\delta e\mu(\mu_T r_2 - B) + c_2 d\mu(c_2\beta\eta - \delta^2 e r_2)]\} + c_1 c_2 de\delta\mu(e\gamma + \eta r_2)(c_2 a\eta + e(\mu - a\delta)) + Bc_1 c_2 ae\mu(e\gamma - \eta r_2)(\beta - \mu) + e\mu(\beta-\mu)\{er_2 c_2[(B - \mu_T r_2)(c_1\gamma + c_2\eta - \mu)] + d\mu[\beta e(c_1 - c_2) + c_1 e(c_1\gamma + c_2\eta) + a(c_1\mu - c_2\delta) - c_2\delta e r_2 + c_1 a\mu]\} + \mu(a\mu + \beta e)\{c_2 ae(B - \mu_T r_2)(c_2\eta - \mu) + de(c_2^2\eta - c_1^2\gamma)(2a\delta + \beta) + adc_1 c_2\eta(c_1\gamma - 2c_2\eta\mu) + \delta e\mu(2c_1 - c_2)\} - c_1 c_2\mu[de\delta r_2(2a\eta\mu + \beta e^2\gamma) + Ba\eta(2a\mu r_2 + \beta e^2)] + c_1^2 ad\delta e\gamma\mu(3e - \mu r_2) + c_1 c_2 ae\mu[B(\beta\gamma r_2 + e\mu) - c_2 d\eta(\beta\gamma + \eta\mu)] + d\mu^2[c_1\mu^2(a^2 + e^2) - c_2\delta e(a\mu r_2 - \beta e^2)] + c_2 ae\mu(\mu_T r_2 - B)[e\gamma(c_1\delta(a+e)) - c_1\beta) + \eta\mu(a(c_1 - c_2) - c_2 er_2] + de\mu^2[\beta(c_1 e(c_1 - \mu) - a(c_1^2\gamma + c_2\mu)) - c_1 a(c_1\gamma(c_2\eta - \mu) - 2\mu(c_1^2\gamma - c_2\mu))] + c_1 c_2\mu^2[2a^2\eta(\mu_T r_2^2 - d\delta e) + e(B\gamma r_2 - ad\eta\mu)] + c_2^2 ae\eta\mu d(\beta\eta + \delta\mu r_2)/(a-e)[a\delta e(c_1^3\eta\gamma^2 + \mu^3) + c_2\delta e^2\mu\gamma(c_1\gamma - \mu) + \mu^4 a] + c_2\delta e^2\gamma\mu a(c_1\gamma - \mu) - \mu^3 ae(e-\mu) + (\eta-\gamma)[c_1 c_2 a\eta\gamma(c_1(a\delta e + a\mu + \beta e - e\mu) - c_2(2a\delta e\gamma + a\eta\mu - \beta e\gamma)) + ae\mu(c_1^2\gamma(a\delta + 2\delta e - \beta\eta + 2\mu) + c_2^2\eta(a\delta\eta + \beta)) + c_1 c_2 a\eta\mu(a\delta e - 2a\mu - a\beta e + \mu) + c_1\mu^2(a^2(\delta e - \gamma + \mu) + ae(\beta - \mu) - \delta e^2) - c_2 e\mu^2(a^2\delta + 2a\delta + \beta(a-e)] - c_1^2 a^2\delta\eta e\gamma(c_2\gamma + \mu) + c_1 c_2 a^2\eta\gamma\mu(c_1 - c_2) - c_1 c_2 ae\gamma(\beta\eta(c_1\gamma + c_2\eta\mu) + 3\mu(a\delta\eta + \beta)) - c_2 a^2\eta\delta e\mu(c_2\eta\gamma + \mu) - c_1^2 a^2\mu^2\gamma(\eta + \delta e) - ae\mu(c_1\gamma\mu(c_2\eta + \mu) + \beta\eta(c_1^2\gamma + c_2\mu)) - \beta e\gamma\mu(c_2^2\eta + c_1 a\mu) - c_1 c_2 a\eta\gamma\mu^2(a+e) + e^2\mu^2(3c_1\delta^2 + c_2\beta) - c_1 a^2\gamma\mu^2(c_2\eta + \mu) + c_1 c_2 a\delta\eta\gamma^2 e^2(2c_1 - c_2) + c_2^2 ae\gamma\eta^2(c_2 a\delta + c_1\mu) + c_1^3 a\gamma^2\eta(a + e(\beta-\mu)) + c_1\delta e^3\gamma\mu(c_1\gamma - \mu) - c_1 c_2 a\delta e\gamma\mu(a\gamma + 4e) + c_2^3 a\eta^2(\beta e\gamma - a\eta\mu) + 2c_2 a\delta e^2\eta\mu(c_2\gamma + \mu) + c_1 e^2\gamma\mu^2(c_1\gamma(1-\beta) + 2(\beta - \gamma\mu)) - c_1 c_2 e\gamma\mu(a\beta - e\eta\mu) + c_2^2 a\eta\mu^2(3a - 2e\eta) - c_2 a\eta\mu^3(3a + 4e) + \mu^3\{\mu[a(a-2e) + e] - c_2 e^2\eta + ae\delta(a-2e) + e^2(\delta e - \beta) + ae\beta\}$$

For stability of model, we have calculated basic reproduction number R_0 using next generation matrix method. [3]

Let $X = (T, S_A, R)'$ and $X' = \frac{dX}{dt} = F(X) - V(X)$

$F(X) =$ The rate of appearance of new individual in component

$V(X) =$ The rate of disease spread

$$F(X) = \begin{bmatrix} \delta TS_A + \frac{\beta TS_A}{a+T} \\ \frac{c_1\gamma S_A R}{d+S_A} + \frac{c_2\eta TR}{e+T} \\ 0 \end{bmatrix} \text{ and}$$

$$V(X) = \begin{bmatrix} \frac{\gamma S_A R}{d+S_A} + \mu S_A \\ \mu R \\ -BT + \delta TS_A + \frac{\beta TS_A}{a+T} + \frac{\eta TR}{e+T} + \mu_T T^2 \end{bmatrix}$$

Now, $DF(X_0) = \begin{bmatrix} f & 0 \\ 0 & 0 \end{bmatrix}$ and $DV(X_0) = \begin{bmatrix} v & 0 \\ J_1 & J_2 \end{bmatrix}$.

where, f and v are 3×3 matrices defined as $f = \left[\frac{\partial F_i(X_0)}{\partial X_j}\right]$

and $v=\left[\frac{\partial V_i(\mathrm{X}_0)}{\partial \mathrm{X}_j}\right]$. Finding f and v we get,

$$f=\begin{bmatrix} \delta T+\frac{\beta T}{a+T} & 0 & \delta S_A+\frac{\beta a S_A}{(a+T)^2} \\ \frac{c_1\gamma dR}{(d+S_A)^2} & \frac{c_1\gamma S_A}{d+S_A}+\frac{c_2\eta T}{e+T} & \frac{c_2\eta eR}{(e+T)^2} \\ 0 & 0 & 0 \end{bmatrix} \text{ and}$$

$$v=\begin{bmatrix} \frac{\gamma dR}{(d+S_A)^2}+\mu & \frac{\gamma dR}{d+S_A} & 0 \\ 0 & \mu & 0 \\ \delta T+\frac{\beta T}{a+T} & \frac{\eta T}{e+T} & 2\mu_T T-B+\delta S_A+\frac{\beta a S_A}{(a+T)^2}+\frac{\eta eR}{(e+T)^2} \end{bmatrix}$$

Here, v is non-singular matrix.

Thus, basic reproduction number R_0 is given by:

$$R_0=\frac{B[\mu_T{}^2((a+\mu_T B)(c_2\eta+\delta e)+\beta e)+\mu_T B(a\delta+\beta)+B^2\delta]}{\mu_T\mu(\mu_T a+B)(\mu_T e+B)} \qquad (3)$$

3 STABILITY

By solving system (1), we have three equilibrium points given in equation (2). In this section, local stability and global stability has been deliberated for each point.

3.1 Local Stability: Local stability analysis is done by using Jacobian matrix which is given as below:

$$J=\begin{bmatrix} J_{11} & -\delta T-\frac{\beta T}{a+T} & -\frac{\eta T}{e+T} \\ \delta S_A+\frac{\beta a S_A}{(a+T)^2} & -\delta T-\frac{\gamma dR}{(d+S_A)^2}+\frac{\beta T}{a+T}-\mu & -\frac{\gamma S_A}{d+S_A} \\ \frac{c_2\eta eR}{(e+T)^2} & \frac{c_1\gamma dR}{(d+S_A)^2} & \frac{c_1\gamma S_A}{d+S_A}+\frac{c_2\eta T}{e+T}-\mu \end{bmatrix}$$

where, $J_{11}=B-\delta S_A-\frac{\beta a S_A}{(a+T)^2}-\frac{\eta eR}{(e+T)^2}-2\mu_T T$

Theorem 3.1.1: The disease free equilibrium point E_0 of model is locally asymptotically stable with conditions:
$\mu_T B(a\delta+\beta)+B^2\delta<\mu_T\mu(\mu_T a+B)$ and $Bc_2\eta<\mu(\mu_T e+B)$.

Proof. The Jacobian matrix at equilibrium point, E_0 is given by,

$$J_0=\begin{bmatrix} -B & -\frac{\delta B}{\mu_T}-\frac{\beta B}{\mu_T\left(a+\frac{B}{\mu_T}\right)} & -\frac{\eta B}{\mu_T\left(e+\frac{B}{\mu_T}\right)} \\ 0 & \frac{\delta B}{\mu_T}+\frac{\beta B}{\mu_T\left(a+\frac{B}{\mu_T}\right)}-\mu & 0 \\ 0 & 0 & \frac{c_2\eta B}{\mu_T\left(e+\frac{B}{\mu_T}\right)}-\mu \end{bmatrix}$$

This Jacobian J_0 gives eigenvalues, $\omega_1=-B$,

$\omega_2=\frac{\mu_T B(a\delta+\beta)+B^2\delta-\mu_T\mu(\mu_T a+B)}{\mu_T(\mu_T a+B)}$ and

$\omega_3=\frac{Bc_2\eta-\mu_T(\mu_T e+B)}{\mu_T e+B}$.

The disease free equilibrium point E_0 is stable if these above all eigenvalues are negative. ω_2 and ω_3 are non-negative gives conditions:

(i) $\mu_T B(a\delta+\beta)+B^2\delta<\mu_T\mu(\mu_T a+B)$ and

(ii) $Bc_2\eta<\mu(\mu_T e+B)$.

Theorem 3.1.2: The recovery free equilibrium point E_1 of model is locally asymptotically stable with six conditions:

Proof. The Jacobian matrix at E_1,

$$J_1=\begin{bmatrix} B-\delta b_1-\frac{\beta a b_1}{(a+r_1)^2}-2\mu_T r_1 & -\delta r_1-\frac{\beta r_1}{a+r_1} & -\frac{\eta r_1}{e+r_1} \\ \delta b_1+\frac{\beta a b_1}{(a+r_1)^2} & \delta r_1+\frac{\beta r_1}{a+r_1}-\mu & -\frac{\gamma b_1}{d+b_1} \\ 0 & 0 & \frac{c_1\gamma b_1}{d+b_1}+\frac{c_2\eta r_1}{e+r_1}-\mu \end{bmatrix}$$

where, $b_1=\frac{\mu_T r_1(\beta-\mu)-\mu_T a\mu+B\delta(r_1+a)}{\delta(r_1\delta+a\delta+\beta)}$

Jacobian has three eigenvalues and after simplification gives the following conditions for equilibrium E_1 point to be stable:

(i) $c_1 b_1\gamma<d\mu$ (ii) $c_1\gamma<\mu$ (iii) $c_2\eta<\mu$

(iv) $c_2\eta r_1<e\mu$ (v) $\mu<\delta r_1$ (vi) $2\mu_T r_1<B$.

Theorem 3.1.3: The endemic equilibrium point E^* of model is locally asymptotically stable with conditions:

Proof. The Jacobian matrix at E^* is,

$$J^*=\begin{bmatrix} -J_{11} & -T^*(\delta+f_1) & -T^* f_3 \\ S_A{}^* a f_2+\delta S_A{}^* & -J_{22} & -S_A{}^* f_5 \\ c_2 R^* \mathrm{e}\, f_4 & c_1 R^* d f_6 & -J_{33} \end{bmatrix},$$

where, $J_{11}=B-\delta S_A{}^*-S_A{}^* a f_2-R^*\mathrm{e}\, f_4-2\mu_T T^*$,

$J_{22}=\delta T^*-dR^* f_6+T^* f_1-\mu$ and $J_{33}=c_1 S^* f_5+c_2 T^* f_3-\mu$

in which

$$f_1=\frac{\beta}{a+T^*},\ f_2=\frac{\beta}{(a+T^*)^2},\ f_3=\frac{\eta}{e+T^*},\ f_4=\frac{\eta}{(e+T^*)^2},$$

$$f_5=\frac{\gamma}{d+S_A{}^*},\ f_6=\frac{\gamma}{(d+S_A{}^*)^2}$$

Characteristic polynomial for this Jacobian matrix is, (Routh, E. J. (1877))

$\lambda^3+a_1\lambda^2+a_2\lambda+a_3=0$

where, a_i's are defined as;

$$a_1=J_{11}+J_{22}+J_{33}$$

$$a_2=c_1 S_A{}^* f_5 dR^* f_6+c_2 T^* f_3 R^*\mathrm{e}\, f_4+J_{11}J_{33}+J_{11}J_{22}+J_{22}J_{33}$$
$$+S_A{}^* a f_2 T^*(\delta T^*+f_1)+\delta S_A{}^* T^*(\delta+f_1)$$

$$a_3=c_2 R^* T^* e f_4(J_{22} f_3-S_A{}^*\delta f_5)+R^* S_A{}^* f_5(c_1 d J_{11} f_6-c_2 T^* e f_4 f_1)$$
$$+c_1 R^* S_A{}^* T^* d f_3 f_6(a f_2+\delta)+J_{33} S_A{}^* T^*(a f_1 f_2+\delta^2+\delta f_1)+J_{33}J_{22}J_{11}$$

Using Routh - Hurwitz criteria for endemic equilibrium point gives following conditions to be stable:

(i) $J_{11} > 0 \Rightarrow \delta S_A^* + S_A^* a f_2 + R^* \mathrm{e}\, f_4 + 2\mu_T T^* > B$

(ii) $J_{22} > 0 \Rightarrow dR^* f_6 + \mu > T^*(\delta + f_1)$

(iii) $J_{33} > 0 \Rightarrow \mu > c_1 S_A^* f_5 + c_2 T^* f_3$

(iv) $J_{22} f_3 > S_A^* \delta f_5$

(v) $c_1 d J_{11} f_6 > c_2 T^* e f_4 f_1$

All above conditions derived in theorem 3.1.1 to 3.1.3, we conclude following conditions for each of three equilibrium point to be stable:

(i) $\mu_T \mu(\mu_T a + B) > \mu_T B(a\delta + \beta) + B^2\delta$ (ii) $\min\{\mu, e\delta\} > c_2\eta$

(iii) $\min\{d, b_1\} > c_1 b_1 \gamma$ (iv) $B > 2\mu_T r_1$

(v) $(Rdf_6 + \mu) f_3 > max\{Rdf_6 + c_1 S_A f_5 + c_2 T f_3, T(\delta + f_1)\}$

(vi) $c_1 d f_6 (Ref_4 + S_A a f_2 + 2\mu_T T + \delta S_A - B) > c_2 T e f_1 f_4$

3.2 Global Stability: For each equilibrium point, global stability is established through Lyapunov function.

Theorem 3.2.1: The disease free equilibrium point E_0 of model is globally asymptotically stable without conditions.

Proof. Let's consider Lyapunov function,

$$L(t) = \frac{T^2 + S_A^{\,2} + R^2}{2}$$

$$\therefore L'(t) = TT' + S_A S_A' + RR'$$

$$\therefore L'(t) = T\left(BT - \delta TS_A - \frac{\beta TS_A}{a+T} - \frac{\eta TR}{e+T} - \mu_T T^2\right) + S_A\left(\delta TS_A - \frac{\gamma S_A R}{d+S_A} + \frac{\beta TS_A}{a+T} - \mu S_A\right) + R\left(\frac{c_1\gamma S_A R}{d+S_A} + \frac{c_2\eta TR}{e+T} - \mu R\right)$$

$$\therefore L'(t) = \frac{B^3}{\mu_T^{\,2}} - \frac{\delta B^2 S_A}{\mu_T^{\,2}} - \frac{\beta B^2 S_A}{\mu_T^{\,2}\left(a + \frac{B}{\mu_T}\right)} - \frac{\eta B^2 R}{\mu_T^{\,2}\left(e + \frac{B}{\mu_T}\right)} + \frac{\delta B S_A^{\,2}}{\mu_T} - \frac{\gamma S_A^{\,2} R}{d+S_A} + \frac{\beta B S_A^{\,2}}{\mu_T\left(a + \frac{B}{\mu_T}\right)} - \mu S_A^{\,2} + \frac{c_1\gamma S_A^{\,2} R}{d+S_A} + \frac{c_2\eta B^2 R}{\mu_T^{\,2}\left(e + \frac{B}{\mu_T}\right)} - \mu R^2$$

$$\therefore L'(t) = \frac{B^3}{\mu_T^{\,2}} + \left(S_A - \frac{B}{\mu_T}\right)\left(\frac{\delta B S_A}{\mu_T} + \frac{\beta B S_A}{\mu_T\left(a + \frac{B}{\mu_T}\right)}\right) + \left(c_2 R - \frac{B}{\mu_T}\right)\frac{\eta BR}{\mu_T\left(e + \frac{B}{\mu_T}\right)} + \left(c_1 R - S_A\right)\frac{\gamma S_A R}{d+S_A} - \mu\left(S_A^{\,2} + R^2\right)$$

After $E_0\left(\frac{B}{\mu_T}, 0, 0\right)$ putting into above equation we attained condition $\frac{B}{\mu_T} > max\{S_A, c_2 R\}$ gives $\frac{B}{\mu_T} > 0$ which is obvious; it indicates that E_0 is globally asymptotically stable.

Theorem 3.2.2: The recovery free equilibrium point E_1 of model is globally asymptotically stable with conditions.

Proof. Let the Lyapunov function be,

$$L(t) = w_1 T + w_2 S_A + w_3 R$$

$$\therefore L'(t) = w_1\left(BT - \delta TS_A - \frac{\beta TS_A}{a+T} - \frac{\eta TR}{e+T} - \mu_T T^2\right) + w_2\left(TS_A - \frac{\gamma S_A R}{d+S_A} + \frac{\beta TS_A}{a+T} - \mu S_A\right) + w_3\left(\frac{c_1\gamma S_A R}{d+S_A} + \frac{c_2\eta TR}{e+T} - \mu R\right)$$

$$\therefore L'(t) = w_1 BT + (w_2 - w_1)\left(\delta TS_A + \frac{\beta TS_A}{a+T}\right) + \frac{\eta TR}{e+T}(c_2 w_3 - w_1) - \mu_T T^2 w_1 + \frac{\gamma S_A R}{d+S_A}(c_1 w_3 - w_2) - \mu(S_A w_2 + R w_3)$$

By taking $w_1 = \gamma, w_2 = \delta$ and $w_3 = \beta$; gives conditions as $\gamma > max\{\delta, \beta c_2\}$ and $\delta > c_1\beta$.

Theorem 3.2.3: The endemic equilibrium point E^* of model is globally asymptotically stable with three conditions.

Proof. Let the Lyapunov function be,

$$L(t) = \frac{1}{2}\left(T - T^*\right)^2 + k_1\frac{1}{2}\left(S_A - S_A^*\right)^2 + k_2\frac{1}{2}\left(R - R^*\right)^2$$

$$\therefore L'(t) = \left(T - T^*\right)\left(BT - \delta TS_A - \frac{\beta TS_A}{a+T} - \frac{\eta TR}{e+T} - \mu_T T^2\right) + k_1\left(S_A - S_A^*\right)\left(\delta TS_A - \frac{\gamma S_A R}{d+S_A} + \frac{\beta TS_A}{a+T} - \mu S_A\right) + k_2\left(R - R^*\right)\left(\frac{c_1\gamma S_A R}{d+S_A} + \frac{c_2\eta TR}{e+T} - \mu R\right)$$

$$\therefore L'(t) = \left(T - T^*\right)^2\left(B - \delta S_A - \frac{\beta S_A}{a+T} - \frac{\eta R}{e+T} - \mu_T\left(T + T^*\right)\right) + k_1\left(S_A - S_A^*\right)^2\left(\delta T - \frac{\gamma R}{d+S_A} + \frac{\beta T}{a+T} - \mu\right) + k_2\left(R - R^*\right)^2\left(\frac{c_1\gamma S_A}{d+S_A} + \frac{c_2\eta T}{e+T} - \mu\right) + T^*\left(T - T^*\right)(B - \mu_T T^*) - \frac{\beta}{a+T}\begin{pmatrix} T^* S_A\left(T - T^*\right) \\ -TS_A^*\left(S_A - S_A^*\right)\end{pmatrix} - \frac{\eta}{e+T}\left(T^* R\left(T - T^*\right) - k_2 c_2 TR^*\left(R - R^*\right)\right) - \delta\left(T^* S_A\left(T - T^*\right) - k_1 TS_A^*\left(S_A - S_A^*\right)\right) - \frac{\gamma}{d+S_A}(k_1 RS_A^*(S_A - S_A^*) - k_2 c_1 R^* S_A(R - R^*)) - \mu\left(k_1 S_A^*\left(S_A - S_A^*\right) + k_2 R^*\left(R - R^*\right)\right)$$

E^* is globally asymptotically stable if $L'(T) < 0$; hence we have following conditions:

(i) $\delta T + \frac{\beta T}{a+T} < \frac{\gamma R}{d+S_A} + \mu$ (ii) $\frac{c_1\gamma S_A}{d+S_A} + \frac{c_2\eta T}{e+T} < \mu$ (iii) $B < \mu_T T^*$.

4 Z-TYPE CONTROL

Z-type control is a powerful mathematical tool which influence the disease spread control. Due to temperature variation; skin diseases cannot be protected and hence it is assumed that 10% skin infection is permissible. The purpose of this paper is to control disease spread and these chaotic oscillations. To control these periodic oscillations, Z-type control is being used and to force the actual output $y(t)$ (Let us say) to attain desired output $y_d(t)$ (Let us say)[1]. This control helps to eradicate the skin infections which in model are error function. So, basically we are minimizing the error function means that error tends to zero. Hence this control is beneficial when the error between actual output and desired output is zero

as $t \to \infty$ that is $e(t) = y(t) - y_d(t) \to 0$ as $t \to \infty$. Error function tends to zero if it satisfies following differential equation,

$$\dot{e}(t) = -\lambda e(t) \tag{4}$$

Here, λ is design parameter. Z-type control depends upon two tools that to define error function and to use design formula for input $y(t)$.

Now we apply Z-type control to the above system (1) of differential equations. Applying control as a treatment on recovery as a medication can give cured skin. Therefore, we have following system,

$$\begin{aligned}
\frac{dT}{dt} &= BT - \delta T S_A - \frac{\beta T S_A}{a+T} - \frac{\eta T R}{e+T} - \mu_T T^2 \\
\frac{dS_A}{dt} &= \delta T S_A - \frac{\gamma S_A R}{d+S_A} + \frac{\beta T S_A}{a+T} - \mu S_A \\
\frac{dR}{dt} &= \frac{c_1 \gamma S_A R}{d+S_A} + \frac{c_2 \eta T R}{e+T} - \mu R - u(t) R
\end{aligned} \tag{5}$$

Here, $u(t)$ is control variable. Our ambition is to reduce the skin allergic individuals $S_A(t)$ to desired state $S_{Ad}(t)$ i.e. $S_A(t) \to S_{Ad}(t)$.

Let's consider $S_{Ad} = S_{Ad}(t)$ the desired state.

Define the error function as the difference of actual output and desired output i.e.

$$e_1 = S_A(t) - S_{Ad}(t)$$

Using the design formula (equation (4)) of the Z-type dynamic method, we have

$$\dot{S}_A(t) - \dot{S}_{Ad}(t) = -\lambda(S_A(t) - S_{Ad}(t))$$

Then second error function,

$$e_2 = \dot{e}_1 + \lambda e_1 = \dot{S}_A(t) - \dot{S}_{Ad}(t) + \lambda(S_A(t) - S_{Ad}(t))$$

But, $\dot{e}_2 = -\lambda e_2$

$$\Rightarrow \ddot{S}_A(t) - \ddot{S}_{Ad}(t) + \lambda(\dot{S}_A(t) - \dot{S}_{Ad}(t)) = -\lambda[\dot{S}_A(t) - \dot{S}_{Ad}(t) + \lambda(S_A(t) - S_{Ad}(t))]$$

$$\Rightarrow \delta \dot{T} S_A + \delta \dot{S}_A T - \frac{\gamma \dot{R} S_A}{d+S_A} - \frac{\gamma \dot{S}_A R}{d+S_A} + \frac{\gamma R \dot{S}_A S_A}{(d+S_A)^2} + \frac{\beta \dot{S}_A T}{a+T} + \frac{\beta \dot{T} S_A}{a+T}$$

$$- \frac{\beta S_A \dot{T} T}{(a+T)^2} - \mu \dot{S}_A - \ddot{S}_{Ad}(t) + \lambda(\dot{S}_A(t) - \dot{S}_{Ad}(t)) = -\lambda[\dot{S}_A(t) - \dot{S}_{Ad}(t)$$

$$+ \lambda(S_A(t) - S_{Ad}(t))]$$

$$\Rightarrow \dot{R}(t) = \frac{d+S_A}{\gamma S}[\delta \dot{T} S_A + \delta \dot{S}_A T - \frac{\gamma \dot{S}_A R}{d+S_A} + \frac{\gamma R \dot{S}_A S_A}{(d+S_A)^2} + \frac{\beta \dot{S}_A T}{a+T} + \frac{\beta \dot{T} S_A}{a+T}$$

$$- \frac{\beta S_A \dot{T} T}{(a+T)^2} - \mu \dot{S}_A - \ddot{S}_{Ad}(t) + \lambda(\dot{S}_A(t) - \dot{S}_{Ad}(t)) + \lambda[\dot{S}_A(t) - \dot{S}_{Ad}(t)$$

$$+ \lambda(S_A(t) - S_{Ad}(t))]]$$

$$\Rightarrow u(t) = \frac{c_1 \gamma S_A}{d+S_A} + \frac{c_2 \eta T}{e+T} - \mu - \frac{d+S_A}{\gamma + S_A}[\delta \dot{T} S_A + \delta \dot{S}_A T - \frac{\gamma \dot{S}_A R}{d+S_A}$$

$$+ \frac{\gamma R \dot{S}_A S_A}{(d+S_A)^2} + \frac{\beta \dot{S}_A T}{a+T} + \frac{\beta \dot{T} S_A}{a+T} - \frac{\beta S_A \dot{T} T}{(a+T)^2} - \mu \dot{S}_A - \ddot{S}_{Ad}(t)$$

$$+ \lambda(\dot{S}_A(t) - \dot{S}_{Ad}(t)) + \lambda[\dot{S}_A(t) - \dot{S}_{Ad}(t) + \lambda(S_A(t) - S_{Ad}(t))]]$$

Theorem 4.1: The tracking error function e_1 with Z-type control exponentially converges to zero for a continuously differentiable and bounded desired state $S_{Ad}(t)$ starting from initial state $[T(0), S_A(0), R(0)]^T$.

Proof. We have equation of second error function $e_2 = \dot{e}_1 + \lambda e_1$ putting into equation $\dot{e}_2 = -\lambda e_2$

After simplifying both the equations we develop,

$$\ddot{e}_1 + 2\lambda \dot{e}_1 + \lambda^2 e_1 = 0.$$

Further solving the second order ordinary differential equation we have,

$$e_1 = (C_1 + C_2 t) exp(-\lambda t), \forall t \geq 0$$

where, C_1 and C_2 are arbitrary constants.

By putting initial value of each compartments,

$C_1 = S_A(0) - S_{Ad}(0)$ and $C_2 = \dot{S}_A(0) - \dot{S}_{Ad}(0) + \lambda(S_A(0) - S_{Ad}(0))$.

According to the Lemma 1 in [16], $\exists \bar{C} > 0$ and $\bar{\lambda} > 0$ such that $e_1 \leq \bar{C}.exp(-\lambda t)$.

Thus, the tracking error exponentially converges to zero which implies that the population of temperature sensitive individuals $S_A(t)$ converges exponentially to the desired state $S_{Ad}(t)$.

5 NUMERICAL SIMULATION

Numerical simulation has been analyzed in this section to depict different outcomes of model for distinct observations.

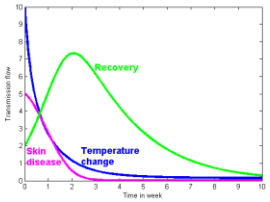

Fig. 2. Transmission rate in compartment

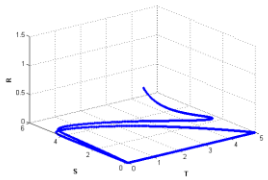

Fig. 3. Scatter diagram

Figure 2 describes the transmission flow of each compartment. One can observe that approximately after six days, temperature sensitive individuals develops skin allergy. Therefore, recovery as a medication is obligatory. It started with less value of rate then raises when it needed; after approximately third week it reduces and because of reinfection it will occur periodically. Scatter diagram using each compartment has been observed in the figure 3. This depicts the periodic nature of the compartments. It advocates that as temperature sensitive individual increases skin diseases arises and if individual concerned with medication for recovery, then reinfection befalls.

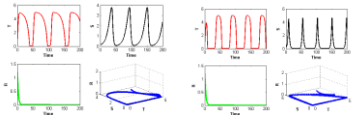

Fig. 4(a). *Oscillations for* $\beta = 0.5$

Fig. 4(b). *Oscillations for* $\beta = 0.9$

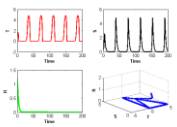

Figure 4(c). *Oscillations for* $\beta = 1.5$

In this fragment, we first found simulation for different values of β that is force of infection. As we increase the value of β the whole system cross the threshold value and converts into chaotic oscillations. Then we perceive the outcome of error function and control variable as a treatment on the model. In proceeding, we see the consequences of Z-type control on oscillations. In Figures 4 (a) - (c), the oscillations for different value of β are shown. Periodic oscillations around the endemic equilibrium point increases with value of β. One can depict that if the force of infection is less than 0.5 then model is stable but increase in it results in chaotic oscillation. In the fourth part of each figure, depicts periodic phenomenon with respect to all compartments. It indicates that once there is skin allergy, reinfection will be there and cannot be cured.

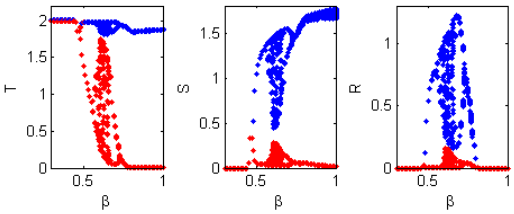

***Fig. 5**. Periodicity in each compartment*

The periodicity has been scrutinized in all three compartments with figure 5. Here, maximum and minimum values are plotted in blue and red colour respectively. Periodicity is proportional to the value of force of infection. If force of infection increases from 0.4, disease can reappear; that is two periodic then after it turns out to be in chaos.

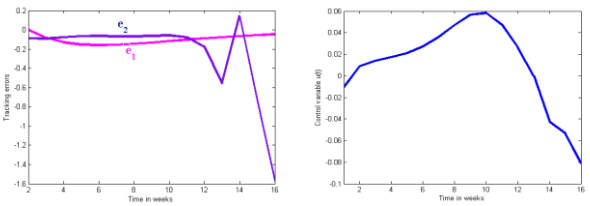

***Fig. 6**. Error function **Fig. 7.** Behavior of control variable*

Figure 6 determines the tracking errors which have been used in design formula. Figure 7 signifies the behavior of control variable as treatment after applying Z-type control on the model. It suggests that susceptible individuals require treatment after one week. Then the treatment gradually increases to 6% in around ten weeks and after that it diminish to zero advocating that no further treatment is needed.

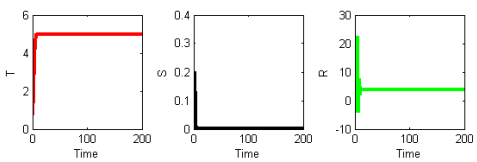

***Fig. 8**. Sensitivity analysis*

Figure 8 describes that, after applying Z-type control on each compartment, one can free from chaotic oscillations and stability has been taken place. After approximately two months at temperature changes, after one month at skin disease and after three and half months at recovery stage; stability has been reached indicates that how Z-type control is effective.

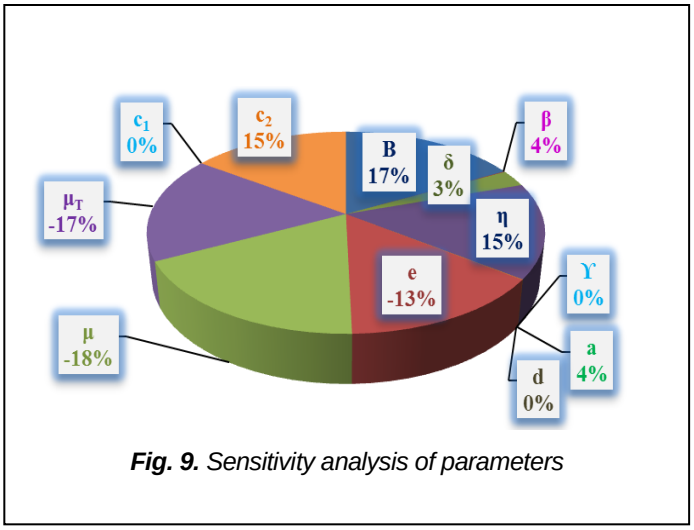

***Fig. 9.** Sensitivity analysis of parameters*

In figure 9, the effect of growth rate (B) of susceptible individuals who are sensitive to temperature change is 17%. With the 4% force of infection (β), 3% individuals get skin allergy (δ). The half saturation constant (a) has positive impact on model so it moderates the rate of force of infection; while (e) gives negative impact. Recovery rate of individuals (γ) connected with saturation constant does not affect model which indicates that it controls intensity of the skin disease. Recovery efficiency constant of individuals (c_2) and recovery rate (η) of skin disease are 15% which is beneficial for reducing skin disease. Escape rate (μ) of individuals in terms of keeping oneself away from temperature change is advocated.

6 CONCLUSION

In view of dynamics of skin allergenic disease, the model is formulated using system of non-linear differential equations with applied Z-type control. Local and global stability of three non-dependent equilibrium points explored with definite conditions. Implementation of numerical simulation helps us to find scenario when treatment is necessary for sensitive individuals. Reinfection is perceived through scatter diagram. Simulation also suggests the reliance of periodicity to the rate of force of infection and chaotic behavior. For a stability of model we put Z-type control as a cure to regulate periodic oscillations. Periodic oscillations can be controlled by decreasing the value of force of infection. Significant change in value of force of infection contributes notable remark on skin disease. Result of application of Z-type control concludes that minimum treatment is required to achieve the disease-free system. Implication of Z-type control is suitable approach for this epidemic disease model. Sensitivity analysis is carried out to mark the impact of each parameter on the model. By taking all parametric values, threshold value is calculated that is 16.69% shows that the model is epidemic. Thus, this model is a good approach to predict biological dynamics through

mathematical model.

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