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STOCHASTIC ANALYSIS OF A TOBACCO SMOKING MODEL CONTAINING SNUFFING CLASS

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Abstract

In this study, a stochastic tobacco smoking model incorporating with snuff users and irregular smokers in view of additive noise induced structure is the main focus of this work, analysis and discussions. By considering the effect of noise on the smoking model and analysing stability with the corresponding driving factors, its dynamics are investigated in terms of stochasticity. Particularly, mathematical tools like Fourier transform and perturbation technique are used to determine the stochastic dynamics. Numerical simulations are carried out on some crucial parameters which plays a major role on population densities framed as X(t), H_1(t), H_2(t), Y(t) and Z(t) is one of the good interesting graphical solutions along with the stochastic study. Computer simulations are used to explain the outcomes of the analysis. After graphical simulations some discussions presented on the graphical results is one more interesting segment in this work. Finally conclusions drawn on impact of noise intensities and varied parametric values on our proposed stochastic structured system.

Key words: Stochastic analysis, Fourier transform, stability, snuffing class

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1. Introduction

Scientific research has a wide range of practical uses. In this area, researchers are focusing on the representation of various diseases using controls as numerical models. In 1909, Brownlee [1] stood up to the plate in support of the development of science. He focused on the potential theory and later proposed essential laws for the spread of a scourge in 1912 [2]. In 1927, the nuances of the pestilence study were dealt upon by Kermark and McKendrik [3]. Afterward, several analysts talked about various models of numerous different illnesses in their works [4–10]. On the other hand, one of the societal propensities that is expanding all through the world swiftly as an irresistible common habit is smoking, generating multiple destructive sicknesses. As a collective, we are aware that smoking harms not just the smoker's health but also the health of all the members of the entire family. Smoking causes long-term harm to the entire society. According to the World Health Organization website [18], measures analysis reveals the following crucial facts: I Tobacco kills up to 50% of its customers. (ii) Each year, 6 million people are killed by tobacco. More over 5 million of those deaths are the result of tobacco use directly, while more than 60,000 are the result of non-smokers being exposed to recycled smoke.to recycled smoke. (iii) Nearly 80% of the world's 1 billion smokers live in low and centre salary nations. As of late, a few analysts have proposed some numerical models to describe smoking conduct. To start with, Castillo-Garsow et al. [16] introduced a deterministic smoking model, at that point Sharomi and Gumel [17] further built up the deterministic model.

Simply put, smoking is the activity in which smoke is absorbed into the mouth and then expelled using channels called stogies. Smoking is the process by which people inhale tobacco smoke made up of particles and gas. Smith and Elone [26] studied, how smoking reduces fitness, health particularly respiratory system particularly who are in army, police, navy and all defence and protective forces, soldiers. Particularly in this article they presented, how military taken control strategies to reduce the usage of tobacco and how to retain their soldier's fitness. But tobacco companies trapped some teams of military by offering free tobacco and other means of gifts to spread the usage of tobacco in soldiers to increase their sales and retain their high yielded business to the top position. This type of traps or strategies applied by companies on the society are also effective and influential social causes for increasing usage of tobacco. The main addictive material present in the tobacco is nicotine which can able to simulate you brain and your activities in to aggressive levels according to their mental stability and intellectuality. The word nicotine is derived from Nicot, who was the pioneer in England who used tobacco as a cash crop and was responsible for using it as a business. When the cigarette producing machine was invented near the end of the nineteenth century, it could produce 200 cigarettes per second. Today, that capacity has increased to 9000 cigarettes per second.

Smoking can cause a wide range of disorders, including cancers of the mouth, throat, and lungs as well as many other conditions that are dangerous to human health [24–33]. Castillo-Garsow et al. [16] established a scientific model for smoking in 1997, which had never been done before. They divided the entire population into three distinct classes for this model (expected smokers, chain smokers and for all time quit smokers). Sharomi and Gumel modified their model in 2008 [17]. They offered a different course (briefly quit smokers). In 2007, Ham [18] conducted an overview in several professional specialised schools in Korea and identified the distinct stages and practises of smoking among understudies. By adding a new class (intermittent smokers), Zaman [19] expanded the model. He also added a dynamical collaboration in a whole number request. The square root components of a model for giving up smoking under any pretence were deduced by Zeb et al. [23] because the framework only allows for a finite amount of time expansion. Others [18–22] introduced the smoking models in full and partial requests. Smoking and snuffing are additional ways that tobacco is used.

In this section we will study the stochastic perturbation effect on the model to understand the randomness and its effects on proposed

system due to physical, chemical, or biological factors. This idea of replacing the whole deterministic system with stochastic differential equation system with the addition of Gaussian white noise and its intensity ranges is quite interesting and recently adopted by several new researchers for understanding the complex dynamics for any compartmental ecological system like the present one. There are many numbers of interesting dynamics in which discrete delay, white noise, and diffusion are gaining more attraction to present the system dynamics innovatively. The univariate nonlinear systems that appear in the work have been successful in describing various modes of disease variability. We are describing the most common and simple problem , which has to be addressed well in this pandemic period.in which random behaviour changes are identified and analysed, which are occurred due to nonlinearity and direct impact for adding white noise for a stochastic differential equation system.

As an external driving forces, where noise may arise either from random fluctuations of one or more model parameters around some known mean values or from stochastic fluctuations of the population densities around some constant values. We also find the population intensities of fluctuations/variances around the positive equilibrium due to white noise (Nisbet and Gunny (27), Tapaswi and Mukhopadhyay [28], Carletti [29-30], Codeco et.al 2008 [31], Sun et.al [32], Wang et.al [33]).

The other sections of the study paper's material are organised as follows in this approach. Section 2 presents the model formation of stochastic system with parameter tabulation and dynamics of stochastic system is in section 3. Section-4 consists of numerical simulations along with observations. Concluding remarks are provided in section-5.

2. Model formation of stochastic system

Smoking is one of the common and sensitive habit which can harm themselves as well as people around them also. It can spoil the next generation people's habits and health by closely moving with these people. This becomes an addiction who becomes susceptible, irregular smokers to regular smokers. This addiction is might habituated by moving closely with smokers, to face any sort of pressure or to work under any pressure conditions, closely working with smokers, working for long hours also main causes for this smoking habit and addiction. To study and analyse this problem in the presence of physical or psychological or social factors as noise intensities is our main objective in this segment

Noise (may be due psychological, physical, social exploiter's) is one of the most considerable factor for any sort of addiction, out of which this smoking is sensitive and simple, but the consequences and effects are in large content. Now we would like to model the current problem as stochastic model with stochastic parameters as Gaussian white noise intensities. The noise induced stochastic model with corresponding noise intensities is framed and structured as follows.

$$X'(t) = A - \beta_1 X H_1 - \mu X + \alpha Y + \alpha_1 \psi_1(t) \quad (1)$$

$$H_1'(t) = \beta_1 X H_1 - \beta_2 H_1 H_2 - (\rho + \mu) H_1 + \alpha_2 \psi_2(t) \quad (2)$$

$$H_2'(t) = \beta_2 H_1 H_2 - (d + \omega_1 + \mu) H_2 + \alpha_3 \psi_3(t) \quad (3)$$

$$Y'(t) = \omega_1 H_2 - (\alpha + \mu) Y - \gamma Y + \alpha_4 \psi_4(t) \quad (4)$$

$$Z'(t) = \gamma Y - \mu Z + \alpha_5 \psi_5(t) \quad (5)$$

Table1: Physical description of the parameters for the structured frame (1-5)

Parameters	Description of the Parameters	Values
A	Recruitment rate of population	0.481
β_1	The Rate of transmission from susceptible class to snuffing class	0.003
β_2	The Rate of transmission from snuffing class to irregular smoker's class	0.002
ω_1	The Rate of transmission from irregular smoker's class to regular smoker's class	0.004
γ	Rate of quitting from smoking	0.05
μ	Natural death rate	0.002
ρ	Death rate of population class snuffing due to the usage of tobacco	0.003
α	Relapse rate	0.004
d	Death rate due to tobacco related diseases	0.003
ξ_i	Intensities of each individual population	Variable
Π_i	Gaussian white Noise of each individual population	Variable

3. Dynamics of stochastic system

Noise may be due psychological, physical, social(exploiter's like tobacco companies) causes is one of the most considerable factor along with the addictive nature, working closely with smoker's also prone to irregular smoking initially and later on move to regular smoking class. Even quitting smoker's class can also be trapped to smoker's class again by some social evil agents to promote their high yielding business. Now we would like to model the current problem as stochastic model with stochastic parameters as Gaussian white noise intensities. The noise induced stochastic model with corresponding noise intensities is framed in the above structured system (1-5) can be analysed through stochastic process using Fourier transformation technique as follows

Let us consider, $X(t)=u_1(t)+X^*$; $H_1(t)=u_2(t)+H_1^*$; $H_3(t)=u_3(t)+H_3^*$; $Y(t)=u_4(t)+Y^*$; $Z(t)=u_5(t)+Z^*$ then the respective linear system of (1)-(5) will changes as

$$u_1'(t)=-\beta u_2(t)X^*+\alpha_1\xi_1(t) \quad (6)$$

$$u_2'(t)=\beta_1 u_1(t)H_1^*-\beta_2 u_3 H_1^*+\alpha_2\xi_2(t) \quad (7)$$

$$u_3'(t)=\beta_1 u_2(t)H_2^*+\alpha_3\xi_3(t) \quad (8)$$

$$u_4'(t)=\alpha_4\xi_4(t) \quad (9)$$

$$u_1'(t)=\alpha_5\xi_5(t) \quad (10)$$

Taking the Fourier transform on (6)-(10) we get,

$$M(\omega)\tilde{u}(\omega)=\tilde{\xi}(\omega) \quad (11)$$

where

$$M(\omega)=\begin{pmatrix} i\omega & \beta_1 X^* & 0 & 0 & 0 \\ -\beta_1 H_1{}^* & i\omega & \beta_2 H_1{}^* & 0 & 0 \\ 0 & -\beta_1 H_2{}^* & i\omega & 0 & 0 \\ 0 & 0 & 0 & i\omega & 0 \\ 0 & 0 & 0 & 0 & i\omega \end{pmatrix};\ \tilde{u}(\omega)=\begin{bmatrix} \tilde{u}_1(\omega) \\ \tilde{u}_2(\omega) \\ \tilde{u}_3(\omega) \\ \tilde{u}_4(\omega) \\ \tilde{u}_5(\omega) \end{bmatrix};\quad \tilde{\xi}(\omega)=\begin{bmatrix} \alpha_1\tilde{\xi}_1(\omega) \\ \alpha_2\tilde{\xi}_2(\omega) \\ \alpha_3\tilde{\xi}_3(\omega) \\ \alpha_4\tilde{\xi}_4(\omega) \\ \alpha_5\tilde{\xi}_5(\omega) \end{bmatrix};$$

From (11), we have $\tilde{u}(\omega)=K(\omega)\tilde{\xi}(\omega)$ (12)

where $K(\omega)=\left[M(\omega)\right]^{-1}=\dfrac{Adj\,M(\omega)}{\left|M(\omega)\right|}$

Now we will give some preliminary information for a random population function. If the function $Y(t)$ has a zero mean value then the fluctuation intensities/variances of its components in the closed frequency interval $[\omega,\omega+d\omega]$ will be $S_Y(\omega)d\omega$, where $S_Y(\omega)$ is the spectral density of Y and is defined as $S_Y(\omega)=\lim_{\tilde{T}\to\infty}\dfrac{\left|\tilde{Y}(\omega)\right|^2}{\tilde{T}}$ (13)

If Y has a zero mean value, the inverse transform of $S_Y(\omega)$ is the auto covariance function as

$$C_Y(\tau)=\frac{1}{2\pi}\int_{-\infty}^{\infty}S_Y(\omega)e^{i\omega\tau}d\omega \qquad (14)$$

The corresponding variance of fluctuations in $Y(t)$ is given by

$$\sigma_Y{}^2=C_Y(0)=\frac{1}{2\pi}\int_{-\infty}^{\infty}S_Y(\omega)d\omega \qquad (15)$$

and the auto correlation function as the normalized auto covariance is $P_Y(\tau)=\dfrac{C_Y(\tau)}{C_Y(0)}$

For a Gaussian white noise process, it will be

$$S_{\xi_i\xi_j}(\omega)=\lim_{\hat{T}\to+\infty}\frac{E\left[\tilde{\xi}_i(\omega)\tilde{\xi}_j(\omega)\right]}{\hat{T}}=\lim_{\hat{T}\to+\infty}\frac{1}{\hat{T}}\int_{-\frac{\hat{T}}{2}}^{\frac{\hat{T}}{2}}\int_{-\frac{\hat{T}}{2}}^{\frac{\hat{T}}{2}}E\left[\tilde{\xi}_i(t)\tilde{\xi}_j(t')\right]e^{-i\omega(t-t')}dt\,dt'=\delta_{ij} \qquad (16)$$

From (12), we have $\tilde{u}_i(\omega)=\sum_{j=1}^{3}K_{ij}(\omega)\tilde{\xi}_j(\omega);i=1,2,3,4,5$ (17)

From (13), we have $S_{u_i}(\omega)=\sum_{j=1}^{3}\eta_j\left|K_{ij}(\omega)\right|^2$; $i=1,2,3,4,5$ (18)

Hence by (15) and (18), the intensities of fluctuations in the variable u_i ; $i=1,2,3,4,5$ are given by

$$\sigma_{u_i}{}^2=\frac{1}{2\pi}\sum_{j=1}^{3}\int_{-\infty}^{\infty}\eta_j\left|K_{ij}(\omega)\right|^2d\omega;\ i=1,2,3,4,5 \qquad (19)$$

and by (12), we obtain

$$\sigma_{u_1}{}^2 = \frac{1}{2\pi}\left\{\begin{array}{l}\int_{-\infty}^{\infty}\alpha_1\left|\frac{Adj(1)}{|M(\omega)|}\right|^2 d\omega + \int_{-\infty}^{\infty}\alpha_2\left|\frac{Adj(2)}{|M(\omega)|}\right|^2 d\omega + \int_{-\infty}^{\infty}\alpha_3\left|\frac{Adj(3)}{|M(\omega)|}\right|^2 d\omega \\ + \int_{-\infty}^{\infty}\alpha_4\left|\frac{Adj(4)}{|M(\omega)|}\right|^2 d\omega + \int_{-\infty}^{\infty}\alpha_5\left|\frac{Adj(5)}{|M(\omega)|}\right|^2 d\omega\end{array}\right\}$$

$$\sigma_{u_2}{}^2 = \frac{1}{2\pi}\left\{\begin{array}{l}\int_{-\infty}^{\infty}\alpha_1\left|\frac{Adj(6)}{|M(\omega)|}\right|^2 d\omega + \int_{-\infty}^{\infty}\alpha_2\left|\frac{Adj(7)}{|M(\omega)|}\right|^2 d\omega + \int_{-\infty}^{\infty}\alpha_3\left|\frac{Adj(8)}{|M(\omega)|}\right|^2 d\omega \\ + \int_{-\infty}^{\infty}\alpha_4\left|\frac{Adj(9)}{|M(\omega)|}\right|^2 d\omega + \int_{-\infty}^{\infty}\alpha_5\left|\frac{Adj(10)}{|M(\omega)|}\right|^2 d\omega\end{array}\right\}$$

$$\sigma_{u_3}{}^2 = \frac{1}{2\pi}\left\{\begin{array}{l}\int_{-\infty}^{\infty}\alpha_1\left|\frac{Adj(11)}{|M(\omega)|}\right|^2 d\omega + \int_{-\infty}^{\infty}\alpha_2\left|\frac{Adj(12)}{|M(\omega)|}\right|^2 d\omega + \int_{-\infty}^{\infty}\alpha_3\left|\frac{Adj(13)}{|M(\omega)|}\right|^2 d\omega \\ + \int_{-\infty}^{\infty}\alpha_4\left|\frac{Adj(14)}{|M(\omega)|}\right|^2 d\omega + \int_{-\infty}^{\infty}\alpha_5\left|\frac{Adj(15)}{|M(\omega)|}\right|^2 d\omega\end{array}\right\}$$

$$\sigma_{u_4}{}^2 = \frac{1}{2\pi}\left\{\begin{array}{l}\int_{-\infty}^{\infty}\alpha_1\left|\frac{Adj(16)}{|M(\omega)|}\right|^2 d\omega + \int_{-\infty}^{\infty}\alpha_2\left|\frac{Adj(17)}{|M(\omega)|}\right|^2 d\omega + \int_{-\infty}^{\infty}\alpha_3\left|\frac{Adj(18)}{|M(\omega)|}\right|^2 d\omega \\ + \int_{-\infty}^{\infty}\alpha_4\left|\frac{Adj(19)}{|M(\omega)|}\right|^2 d\omega + \int_{-\infty}^{\infty}\alpha_5\left|\frac{Adj(20)}{|M(\omega)|}\right|^2 d\omega\end{array}\right\}$$

$$\sigma_{u_5}{}^2 = \frac{1}{2\pi}\left\{\begin{array}{l}\int_{-\infty}^{\infty}\alpha_1\left|\frac{Adj(21)}{|M(\omega)|}\right|^2 d\omega + \int_{-\infty}^{\infty}\alpha_2\left|\frac{Adj(22)}{|M(\omega)|}\right|^2 d\omega + \int_{-\infty}^{\infty}\alpha_3\left|\frac{Adj(23)}{|M(\omega)|}\right|^2 d\omega \\ + \int_{-\infty}^{\infty}\alpha_4\left|\frac{Adj(24)}{|M(\omega)|}\right|^2 d\omega + \int_{-\infty}^{\infty}\alpha_5\left|\frac{Adj(25)}{|M(\omega)|}\right|^2 d\omega\end{array}\right\}$$

where $|M(\omega)| = |R(\omega)| + i|I(\omega)|$

Now, real part of $|M(\omega)| = R(\omega) = 0$

and imaginary part of $|M(\omega)| = I(\omega) = -\omega^5 + \omega^3\left(\beta_1^2 H_1^* X^* + \beta_1\beta_2 H_1^* H_2^*\right)$

So, $|Adj(i)|^2 = X_i^2 + Y_i^2, i = 1, 2, \ldots 25$

where

$$X_1 = \omega^4 - \omega^2\beta_1\beta_2 H_1^* H_2^*; Y_1 = 0; X_2 = 0; Y_2 = \omega^3\beta_1 X^*; X_3 = -\omega^2\beta_1\beta_2 H_1^* X^*; Y_3 = 0;$$

$$X_4 = 0; Y_4 = 0; X_5 = 0; Y_5 = 0; X_6 = 0; Y_6 = -\omega^3\beta_1 H_1^*; X_7 = \omega^4; Y_7 = 0; X_8 = 0; Y_8 = \omega^3\beta_2 H_1^*;$$

$$X_9 = 0; Y_9 = 0; X_{11} = -\omega^2\beta_1\beta_2 H_1^* H_2^*; Y_{11} = 0; X_{12} = 0; Y_{12} = -\omega^3\beta_1 H_2^*; X_{13} = \omega^4 - \omega^2\beta_1^2 H_1^* X^*; Y_{13} = 0$$

$$X_{14} = Y_{14} = X_{15} = Y_{15} = X_{16} = Y_{16} = X_{17} = Y_{17} = X_{18} = Y_{18} = 0; X_{19} = \omega^4 - \omega^2(\beta_1\beta_2 H_1^* H_2^* + \beta_1^2 H_1^* X^*)$$

$$Y_{19} = X_{20} = Y_{20} = X_{21} = Y_{21} = X_{22} = Y_{22} = X_{23} = Y_{23} = X_{24} = Y_{24} = 0;$$
$$X_{25} = \omega^4 - \omega^2(\beta_1\beta_2 H_1^* H_2^* + \beta_1^2 H_1^* X^*); Y_{25} = 0$$

Thus, the population variances are given by

$$\sigma_{u_1}{}^2 = \frac{1}{2\pi}\left\{\int_{-\infty}^{\infty} \frac{1}{R^2(\omega)+I^2(\omega)}\begin{bmatrix}\alpha_1\left(X_1{}^2+Y_1^2\right)+\alpha_2\left(X_2{}^2+Y_2^2\right)+\alpha_3\left(X_3{}^2+Y_3^2\right)\\+\alpha_4\left(X_4{}^2+Y_4^2\right)+\alpha_5\left(X_5{}^2+Y_5^2\right)\end{bmatrix}d\omega\right\}$$

$$\sigma_{u_2}{}^2 = \frac{1}{2\pi}\left\{\int_{-\infty}^{\infty} \frac{1}{R^2(\omega)+I^2(\omega)}\begin{bmatrix}\alpha_1\left(X_6{}^2+Y_6^2\right)+\alpha_2\left(X_7{}^2+Y_7^2\right)+\alpha_3\left(X_8{}^2+Y_8^2\right)\\+\alpha_4\left(X_9{}^2+Y_9^2\right)+\alpha_5\left(X_{10}{}^2+Y_{10}{}^2\right)\end{bmatrix}d\omega\right\}$$

$$\sigma_{u_3}{}^2 = \frac{1}{2\pi}\left\{\int_{-\infty}^{\infty} \frac{1}{R^2(\omega)+I^2(\omega)}\begin{bmatrix}\alpha_1\left(X_{11}{}^2+Y_{11}{}^2\right)+\alpha_2\left(X_{12}{}^2+Y_{12}{}^2\right)+\alpha_3\left(X_{13}{}^2+Y_{13}{}^2\right)\\+\alpha_4\left(X_{14}{}^2+Y_{14}{}^2\right)+\alpha_5\left(X_{15}{}^2+Y_{15}{}^2\right)\end{bmatrix}d\omega\right\}$$

$$\sigma_{u_4}{}^2 = \frac{1}{2\pi}\left\{\int_{-\infty}^{\infty} \frac{1}{R^2(\omega)+I^2(\omega)}\begin{bmatrix}\alpha_1\left(X_{16}{}^2+Y_{16}{}^2\right)+\alpha_2\left(X_{17}{}^2+Y_{17}{}^2\right)+\alpha_3\left(X_{18}{}^2+Y_{18}{}^2\right)\\+\alpha_4\left(X_{19}{}^2+Y_{19}{}^2\right)+\alpha_5\left(X_{20}{}^2+Y_{20}{}^2\right)\end{bmatrix}d\omega\right\}$$

$$\sigma_{u_5}{}^2 = \frac{1}{2\pi}\left\{\int_{-\infty}^{\infty} \frac{1}{R^2(\omega)+I^2(\omega)}\begin{bmatrix}\alpha_1\left(X_{21}{}^2+Y_{21}{}^2\right)+\alpha_2\left(X_{22}{}^2+Y_{22}{}^2\right)+\alpha_3\left(X_{23}{}^2+Y_{23}{}^2\right)\\+\alpha_4\left(X_{24}{}^2+Y_{24}{}^2\right)+\alpha_5\left(X_{25}{}^2+Y_{25}{}^2\right)\end{bmatrix}d\omega\right\}$$

If we are now interested for the dynamics of the system (2.1)-(2.3) with either $\alpha_1 = 0$ or $\alpha_2 = 0$, or $\alpha_3 = 0$, or $\alpha_4 = 0$ or $\alpha_5 = 0$ then the population variances are given by

For $\alpha_1 = \alpha_2 = \alpha_3 = \alpha_4 = 0$ and then $\sigma_{u_1}{}^2 = \sigma_{u_2}{}^2 = \sigma_{u_3}{}^2 = \sigma_{u_4}{}^2 = 0$;

$$\sigma_{u_5}{}^2 = \frac{\alpha_5}{2\pi}\int_{-\infty}^{\infty} \frac{(\omega^4 - \omega^2(\beta_1\beta_2 H_1^* H_2^* + \beta_1^2 H_1^* X^*))^2}{R^2(\omega)+I^2(\omega)}d\omega;$$

For $\alpha_1 = \alpha_2 = \alpha_3 = \alpha_5 = 0$ and then $\sigma_{u_1}{}^2 = \sigma_{u_2}{}^2 = \sigma_{u_3}{}^2 = \sigma_{u_5}{}^2 = 0$

$$\sigma_{u_4}{}^2 = \frac{\alpha_4}{2\pi}\int_{-\infty}^{\infty} \frac{(\omega^4 - \omega^2(\beta_1\beta_2 H_1^* H_2^* + \beta_1^2 H_1^* X^*))^2}{R^2(\omega)+I^2(\omega)}d\omega;$$

For $\alpha_1 = \alpha_2 = \alpha_4 = \alpha_5 = 0$ and then $\sigma_{u_4}{}^2 = \sigma_{u_5}{}^2 = 0$

$$\sigma_{u_1}{}^2 = \frac{\alpha_3}{2\pi}\int_{-\infty}^{\infty} \frac{(-\omega^2\beta_1\beta_2 H_1^* X^*)^2}{R^2(\omega)+I^2(\omega)}d\omega;$$

$$\sigma_{u_2}{}^2 = \frac{\alpha_3}{2\pi}\int_{-\infty}^{\infty} \frac{(\omega^3\beta_2 H_1^*)^2}{R^2(\omega)+I^2(\omega)}d\omega$$

$$\sigma_{u_3}{}^2 = \frac{\alpha_3}{2\pi}\int_{-\infty}^{\infty} \frac{(\omega^4 - \omega^2\beta_1^2 H_1^* X^*)^2}{R^2(\omega)+I^2(\omega)}d\omega$$

For $\alpha_1 = \alpha_3 = \alpha_4 = \alpha_5 = 0$ and then $\sigma_{u_4}{}^2 = \sigma_{u_5}{}^2 = 0$

$$\sigma_{u_1}^{\ 2} = \frac{\alpha_2}{2\pi}\int_{-\infty}^{\infty}\frac{(\omega^3\beta_1 X^*)^2}{R^2(\omega)+I^2(\omega)}d\omega;$$

$$\sigma_{u_2}^{\ 2} = \frac{\alpha_2}{2\pi}\int_{-\infty}^{\infty}\frac{(\omega^4)^2}{R^2(\omega)+I^2(\omega)}d\omega$$

$$\sigma_{u_3}^{\ 2} = \frac{\alpha_2}{2\pi}\int_{-\infty}^{\infty}\frac{(-\omega^3\beta_1 H_2^{\ *})^2}{R^2(\omega)+I^2(\omega)}d\omega$$

For $\alpha_2=\alpha_3=\alpha_4=\alpha_5=0$ and then $\sigma_{u_4}^{\ 2}=\sigma_{u_5}^{\ 2}=0$

$$\sigma_{u_1}^{\ 2} = \frac{\alpha_1}{2\pi}\int_{-\infty}^{\infty}\frac{(\omega^4-\omega^2\beta_1\beta_2 H_1^{\ *}H_2^{\ *})^2}{R^2(\omega)+I^2(\omega)}d\omega;$$

$$\sigma_{u_2}^{\ 2} = \frac{\alpha_1}{2\pi}\int_{-\infty}^{\infty}\frac{(-\omega^3\beta_1 H_1^{\ *})^2}{R^2(\omega)+I^2(\omega)}d\omega$$

$$\sigma_{u_3}^{\ 2} = \frac{\alpha_1}{2\pi}\int_{-\infty}^{\infty}\frac{(-\omega^2\beta_1\beta_2 H_1^{\ *}H_2^{\ *})^2}{R^2(\omega)+I^2(\omega)}d\omega$$

Analytical evaluation of the population variances is very difficult to calculate, but it can be evaluated numerically for a different set of values of certain proposed parameters.

4. Numerical simulations:

In this section, the above analytical results are validated in terms numerical simulations with the tabulated values from table – A The following simulations allows us to draw some remarkable conclusion on the proposed structure with population classes (X(t), H_1(t), H_2(t), Y(t) and Z(t) along with other parameter values in the system(1-5) which are best fitted from table-1 .

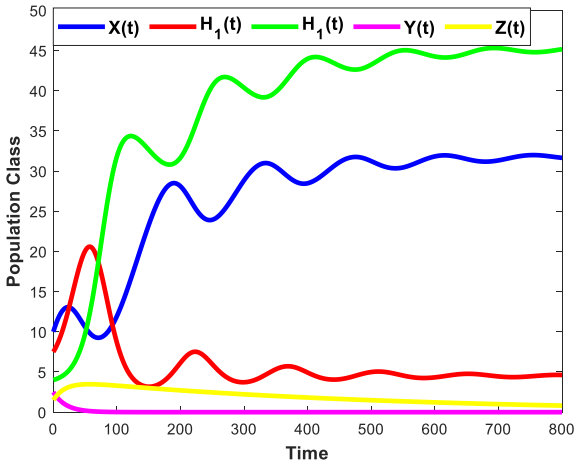

Figure-1

Figure-1 represents the time series evaluation of population with the initial values $X(0)=10, H_1(0)=7.5; H_2(0)=4; Y(0)=2.5; Z(0)=1.5$ and with table –1 values

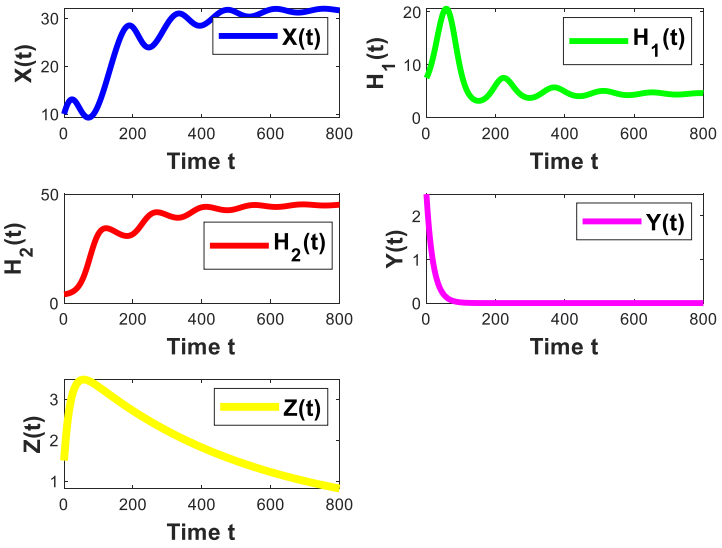

Figure-1a

Figure-1a represents the time series evaluation of different classes of population with the initial values $X(0)=10, H_1(0)=7.5; H_2(0)=4; Y(0)=2.5; Z(0)=1.5$ and values with table-1

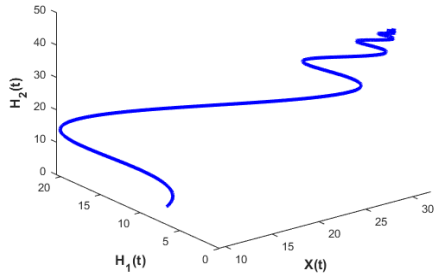

Figure 1(b)

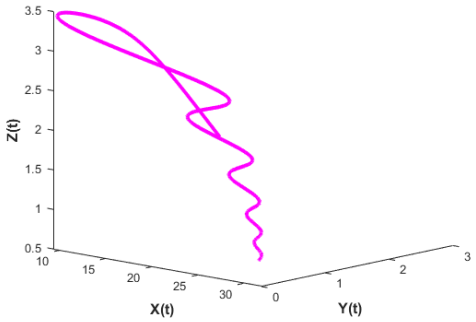

Figure 1(c)

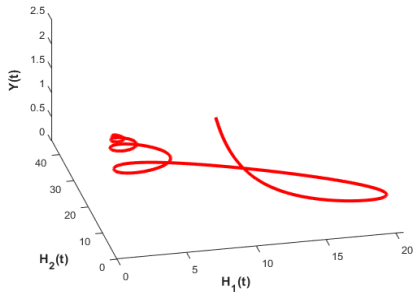

Figure 1(d)

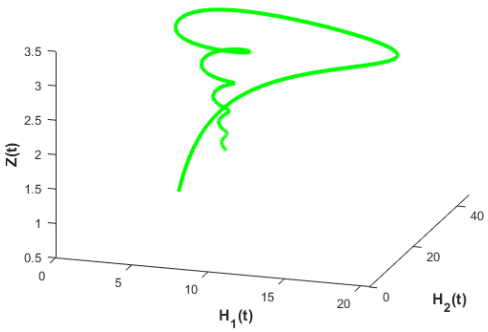

Figure 1(e)

Figures 1(b), 1(c), 1(d) and 1(e) represents the phase portrait projections of among populations(X(t), $H_1(t)$, $H_2(t)$, Y(t), Z(t)) with the attributes of table-1

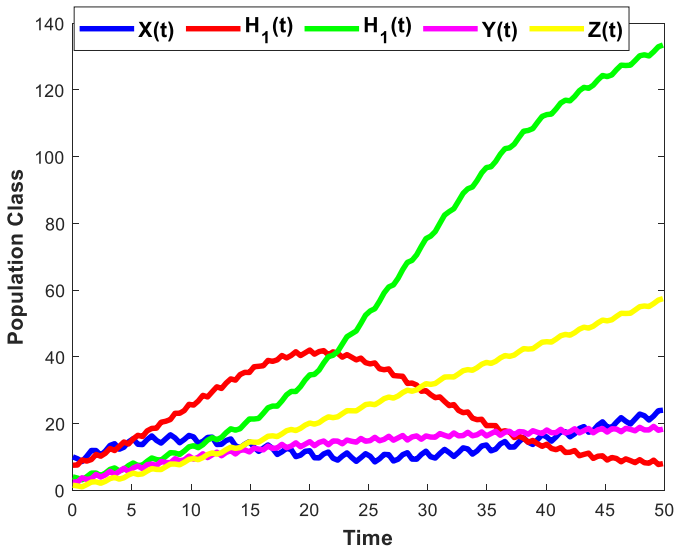

Figure 2a

Figure 2a shows the projection plot of population classes – X(t), $H_1(t)$, $H_2(t)$, Y(t) and Z(t) for the noise intensities $5;3;3;4;2$ and along with the other attributes of table A

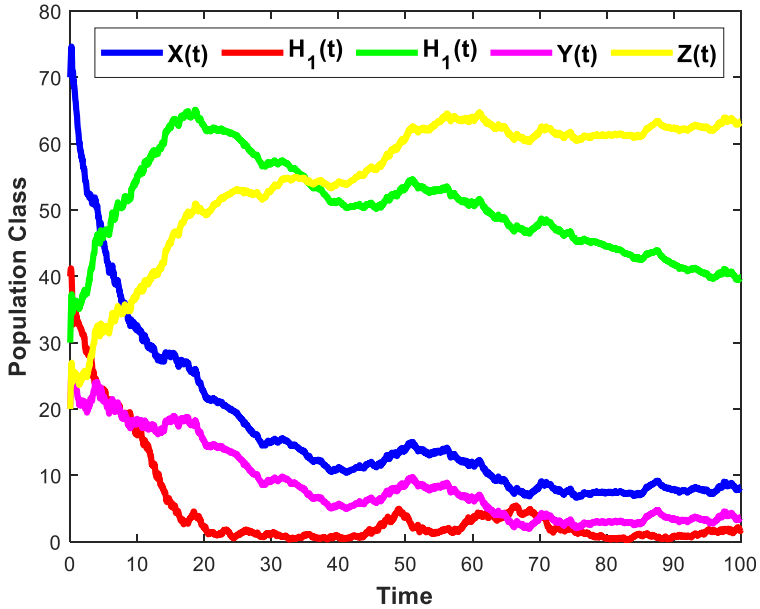

Figure 2b

Figure 2b shows the projection plot of population classes – X(t), $H_1(t)$, $H_2(t)$, Y(t) and Z(t) for the noise intensities $45;25;25;35;15$ and along with the other attributes of table-1.

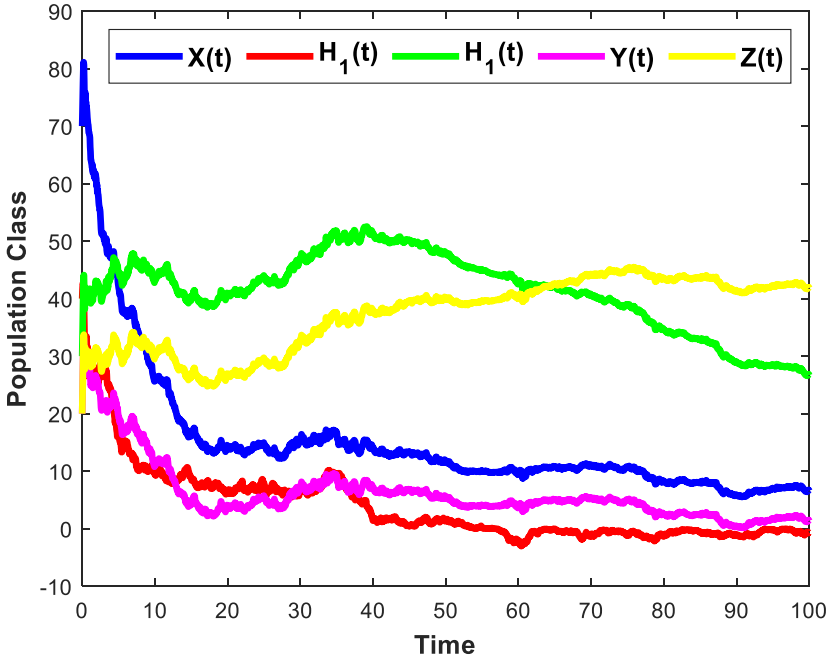

Figure 2c

Figure 2c shows the projection plot of population classes – X(t), $H_1(t)$, $H_2(t)$, Y(t) and Z(t) for the noise intensities $85;50;55;75;40$ and along with the other attributes of table-1.

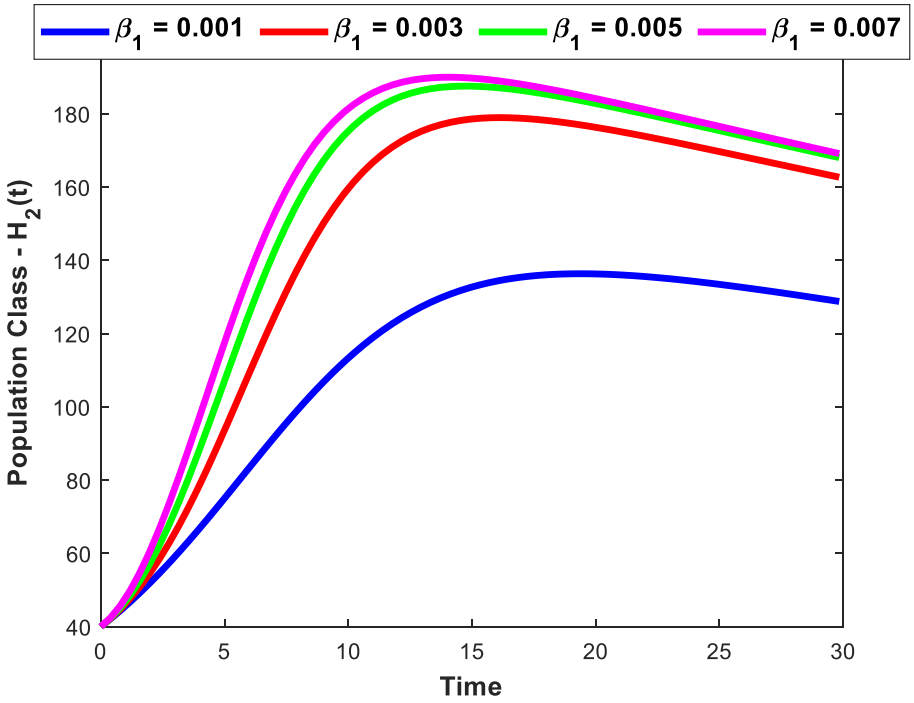

Figure 3c

Figure 3c represents the variations in time series evaluation of population class – $H_2(t)$ for various values of parameter β_1 and along with the other attributes of table-1.

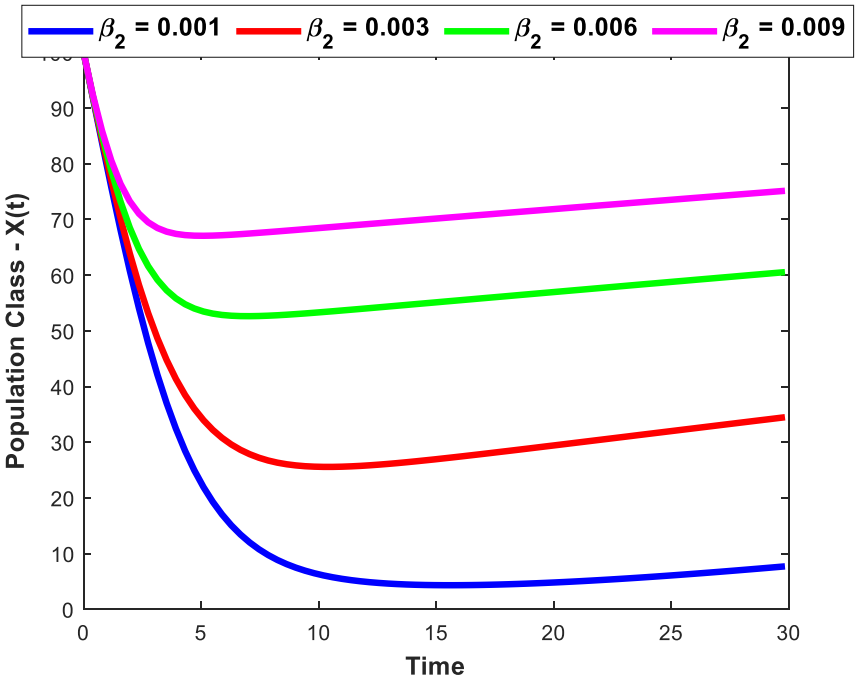

Figure 4a

Figure 4a represents the variations in time series evaluation of population class – X(t) for various values of parameter β_2 and along with the other attributes of table -1.

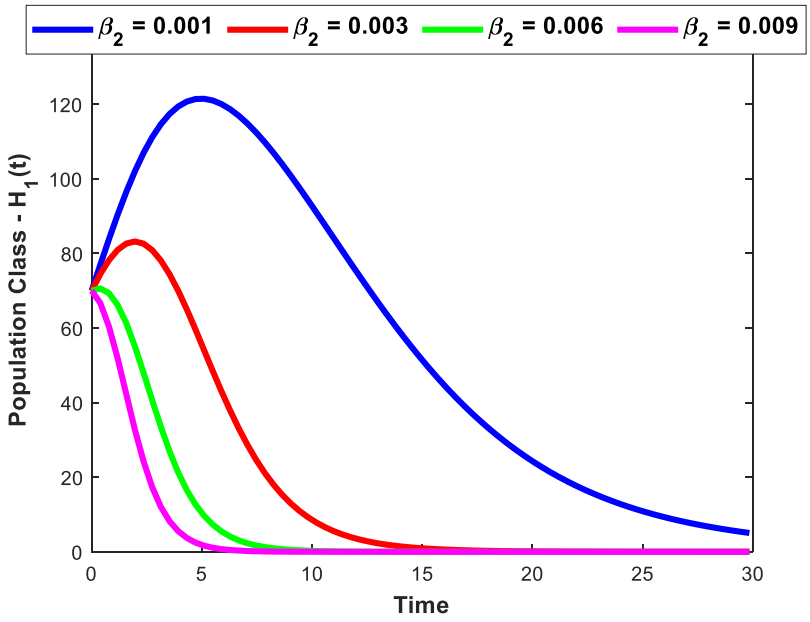

Figure 4b

Figure 4b represents the variations in time series evaluation of population class – $H_1(t)$ for various values of parameter β_2 and along with the other attributes of table-1.

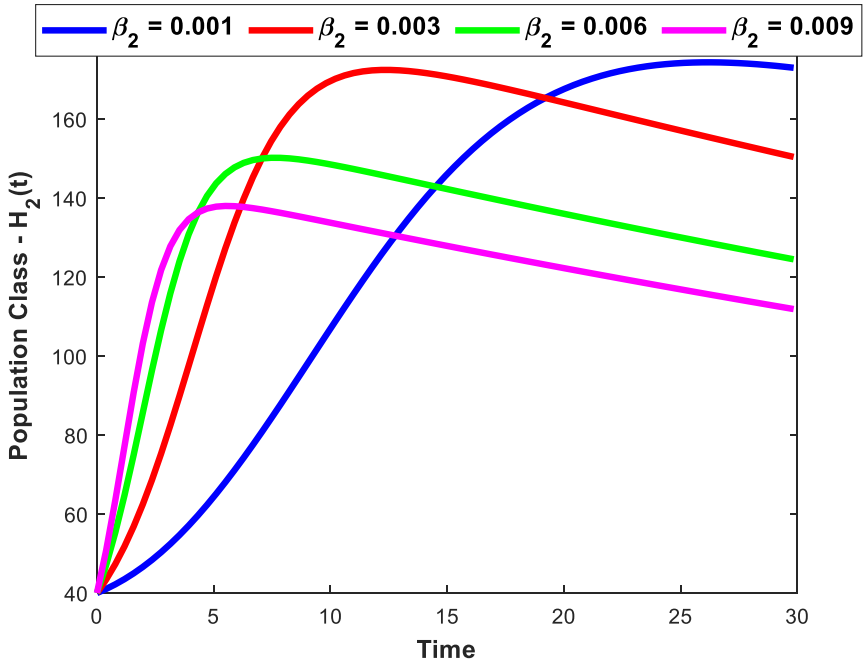

Figure 4c

Figure 4c represents the variations in time series evaluation of population class – $H_2(t)$ for various values of parameter β_2 and along with the other attributes of table-1.

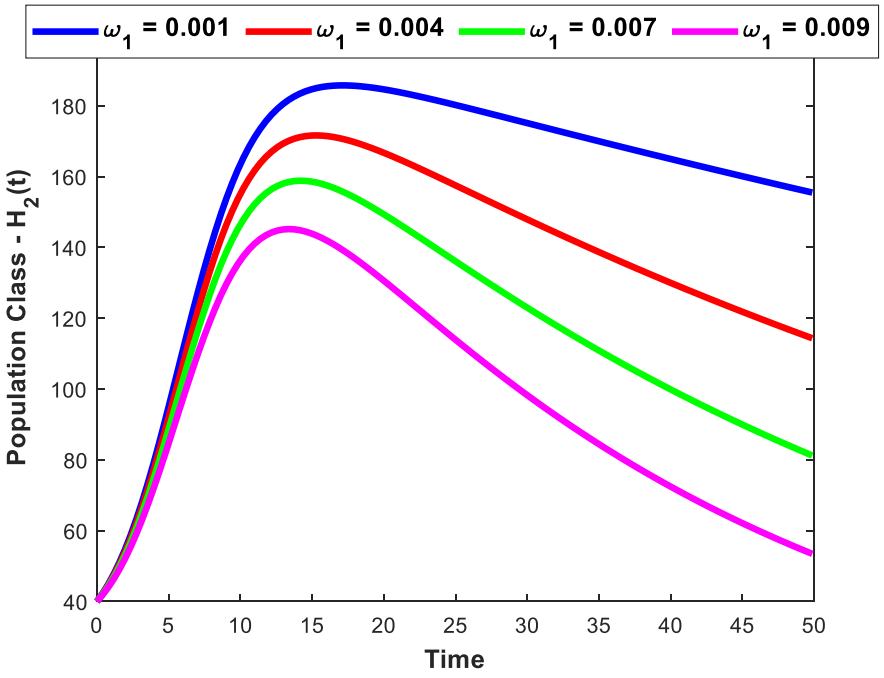

Figure 5a

Figure 5a represents the variations in time series evaluation of population class – $H_2(t)$ for various values of parameter ω_1 and along with the other attributes of table-1 .

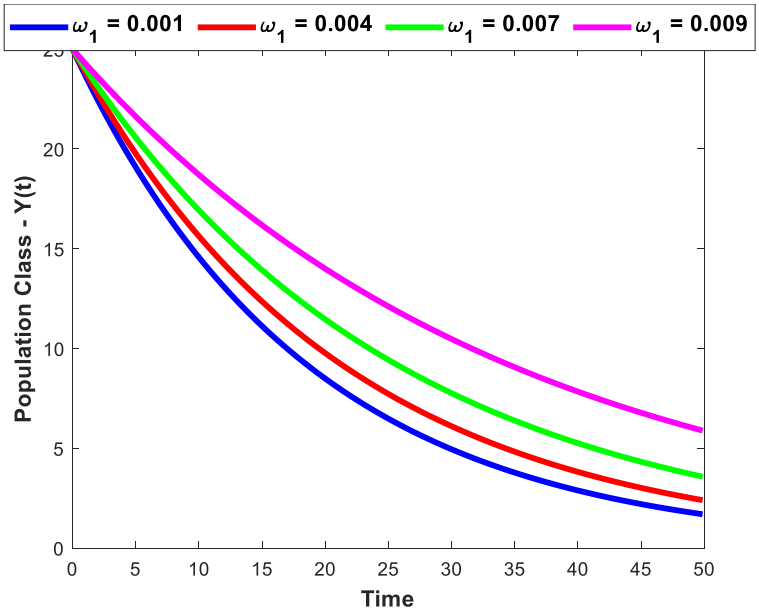

Figure 5b

Figure 5b represents the variations in time series evaluation of population class – Y(t) for various values of parameter ω_1 and along with the other attributes of table-1.

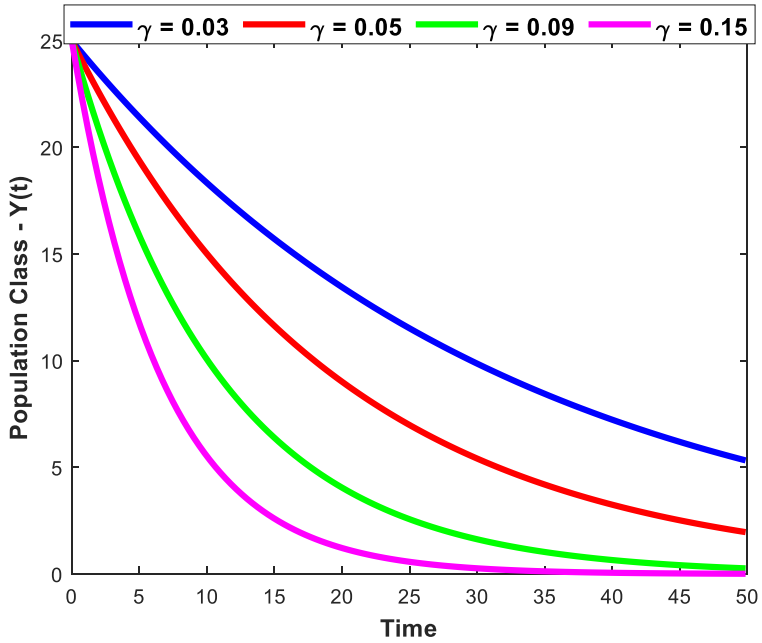

Figure 6a

Figure 6a represents the variations in time series evaluation of population class – Y(t) for various values of parameter γ and along with the other attributes of table-1.

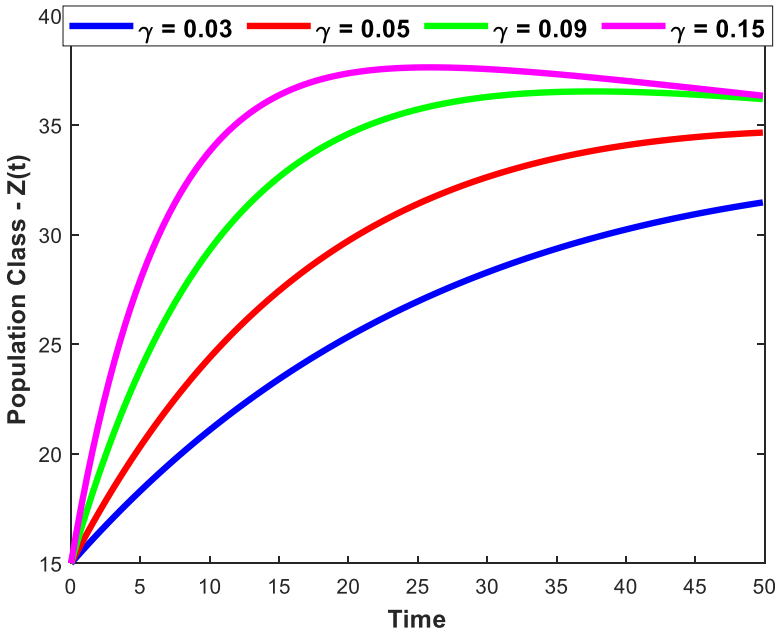

Figure 6b

Figure 6b represents the variations in time series evaluation of population class – Z(t) for various values of parameter γ and along with the other attributes of table-1.

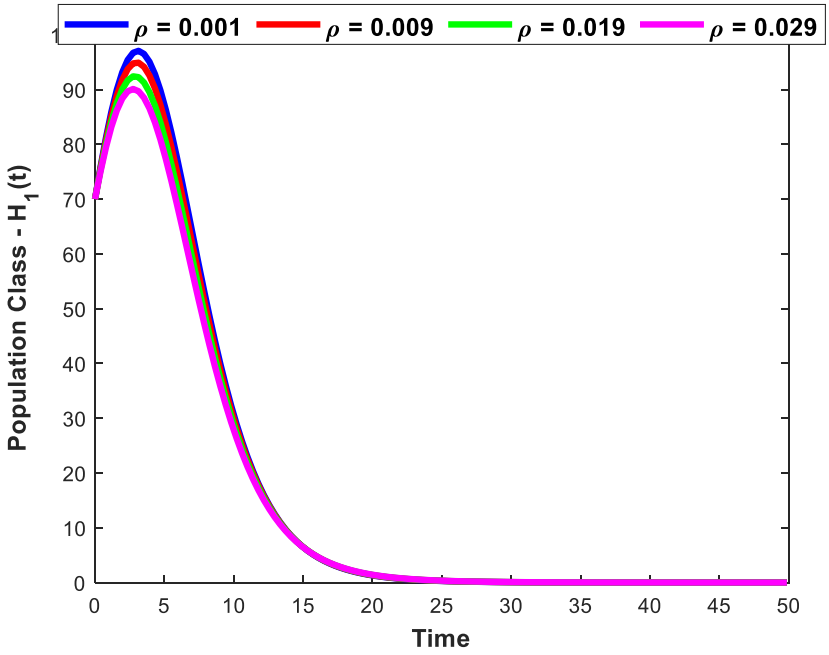

Figure 7a

Figure 7a represents the variations in time series evaluation of population class – $H_1(t)$ for various values of parameter ρ and along with the other attributes of table-1.

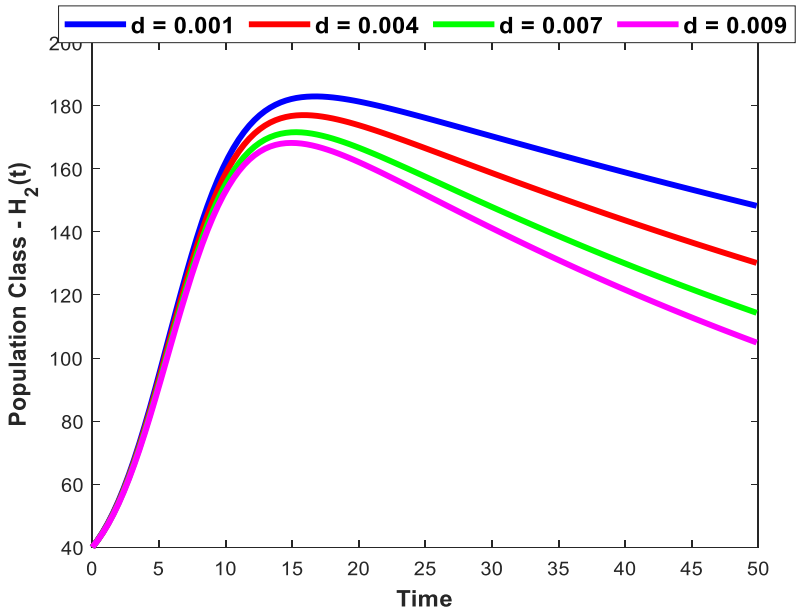

Figure 8a

Figure 8a represents the variations in time series evaluation of population class – $H_2(t)$ for various values of parameter d and along with the other attributes of table-1.

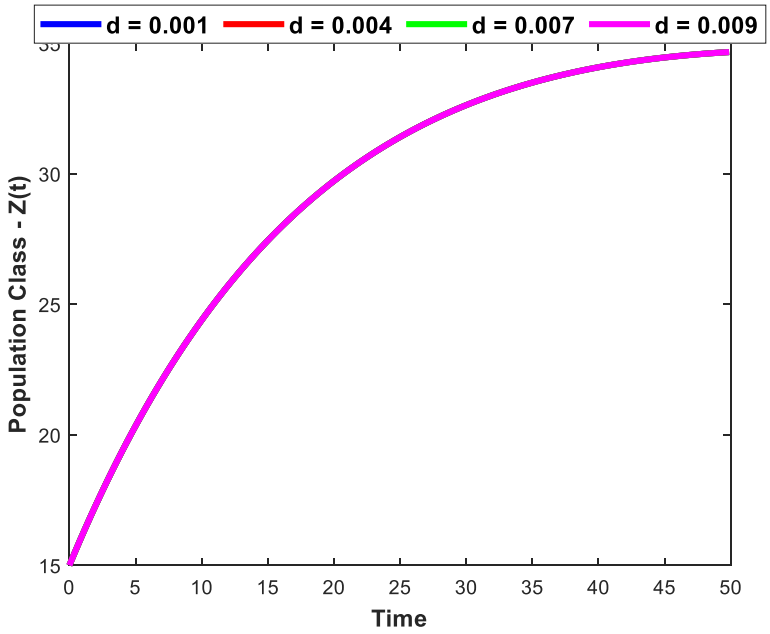

Figure 8b

Figure 8b represents the variations in time series evaluation of population class – Z(t) for various values of parameter d and along with the other attributes of table -1.

Observations and Discussions on Numerical Simulation:

Figure 1 is the projection plot for the deterministic structure framed using five compartmental population classes (susceptible smoker's class, snuffing class, irregular smokers, regular smokers and quit smokers) along with time t as X(t), $H_1(t)$, $H_2(t)$, Y(t) and Z(t) with the suitable attributed values from Table-1.

Figure 1a is the projection plot for the deterministic structure framed using five

compartmental population classes along with time t as X(t), $H_1(t)$, $H_2(t)$, Y(t) and Z(t) individually projected graphically with the suitable attributed values from Table- 1.

Figure 1b, 1c and 1d are the phase portrait projection plot for the deterministic structure framed using five compartmental population classes (susceptible smoker's class, snuffing class, irregular smokers, regular smokers and quit smokers) along with time t as X (t), H_1 (t), $H_2(t)$, Y(t) and Z(t) projected among them (with different combination) graphically with the suitable attributed values from Table- 1.

Figures 2(a), 2(b) 2(c) are stochastic graphs for the proposed model with additive noise on structured frame on smoking addiction.

Figure 2a represents the time series evaluation of populations for the values of noise intensities of 5, 3, 3, 4 and 2 respectively along with the attributes of table A. Figure 2a clearly shows the oscillatory behaviour exhibited by all populations classes X(t), $H_1(t)$, $H_2(t)$, Y(t) and Z(t), which says model system starts to undergone influence at these noise intensities(5, 3, 3, 4 and 2) System influenced by additive noise is notable in this Figure.

Figure 2b represents the time series evaluation of populations for the values of noise intensities of 45, 25, 25, 35 and 15 respectively along with the attributes of table 1. Figure 2b also clearly shows good oscillatory behaviour exhibited by all populations classes X(t), $H_1(t)$, $H_2(t)$, Y(t) and Z(t), which says model system undergone good amount of influence at these noise intensities(45, 25, 25, 35 and 15). System exhibits its remarkable dynamics with additive noise is quite interesting in this Figure.

Figure 2c represents the time series evaluation of populations for the values of noise intensities of 85, 50, 55, 75 and 40 respectively along with the attributes of table 1. Figure 2c also clearly shows the high oscillatory behaviour exhibited by all populations classes X(t), $H_1(t)$, $H_2(t)$, Y(t) and Z(t), which says model system undergone high influence at these noise intensities(85, 50, 55, 75 and 40). System exhibits high and rich dynamics with these values of additive noise are good interesting in this Figure.

Figure 3a, 3b and 3c shows the variations in population classes X(t), $H_1(t)$ and $H_2(t)$ for various values of β_1 along with the other rest of the values as best fitted from Table-1.

As the rate at which population moves from susceptible class to snuffing class, β_1 increases, susceptible smokers population class X(t), which is quite natural that as susceptible moves to another class then, there will be a gradual decrease in X(t) which is clearly presented in the graph and labelled as Figure 3a.

As the rate at which population moves from susceptible class to snuffing class, β_1 increases, snuffing population class $H_1(t)$ increases. Which is quite natural that as susceptible moves to another class, which is snuffing population class then, there will be a gradual increase in Y(t) which is clearly presented in the graph and labelled as Figure 3b.

As the rate at which population moves from susceptible class to snuffing class, β_1 increases, irregular smoker's population class $H_2(t)$ increases. Which is an indirect effect or interrelation between snuffing class and irregular smoker's class is identified and presented in this graph and which is labelled as Figure 3c. Mathematically modelled and structured frame (like the model which is considered in this current work) can enable us to find out the interrelations between the different behaviour classes and their impact can be identified easily with the help of graphical simulation with the suitable and best fitted values which can validate the analytical results.

Figure 4a, 4b and 4c shows the variations in population classes X(t), $H_1(t)$ and $H_2(t)$ for various values of β_2 along with the other rest of the values as best fitted from Table-1.

As the rate at which population class become another class, i.e, snuffing class become irregular smokers class as β_2 increases, susceptible smokers population class X(t) increases. Which is again an interrelation between the susceptible and smokers (irregular or regular) is always a proportional relation between them. As irregular smokers increases (β_2 increases means snuffing class becomes irregular smokers class gradually) more people are prone to become susceptible, the same result which is clearly exhibited by X(t)(gradual increase). The clear and gradual increase in X(t) is presented in the graph and labelled as Figure 4a.

As the rate at which population class become another class, i.e, snuffing class become irregular smokers class as β_2 increases, snuffing population class $H_1(t)$ decreases. Which is quite natural that as snuffing class become another class, which is irregular smokers population class then, there will be a gradual decrease in snuffing population class $H_1(t)$ which is clearly presented in the graph and labelled as Figure 4b.

As the rate at which population class become another class, i.e, snuffing class become irregular smokers class as β_2 increases, irregular population class $H_2(t)$ decreases. Which is the reflexion of interrelated complex dynamics. There is a decrease in irregular smokers population class states that snuffing decrease rate bigger than increase rate of β_2 which is clearly presented in the graph and labelled as Figure 4c.

Which is again an interrelation between the susceptible and smokers (irregular or regular) is always a proportional relation between them. As irregular smokers increases (β_2 increases means snuffing class becomes irregular smokers class gradually) more people are prone to become susceptible, the same result which is clearly exhibited by X(t)(gradual increase). The clear and gradual increase in X(t) is presented in the graph and labelled as Figure 4b.

Figure 5a and 5b shows the variations in population classes $H_2(t)$ and Y(t) for various values of ω_1 along with the other attributes of Table-1.

As the rate at which population class become another class, i.e, irregular class become regular smokers class as ω_1 increases, irregular smokers population class $H_2(t)$ decreases. Which is quite natural that as irregular population class become another class, which is regular smokers population class then, there will be a gradual decrease in irregular population class $H_2(t)$ which is clearly presented in the graph and labelled as Figure 5a.

As the rate at which population class become another class, i.e, irregular class become regular smokers class as ω_1 increases, regular smokers population class Y(t) increases. Which is quite natural that as irregular population class become another class, which is regular smokers population class then, there will be a gradual increase in regular population class Y(t) which is clearly presented in the graph and labelled as Figure 5b.

Figure 6a and 6b shows the variations in population classes Y(t) and Z(t) for various values of γ along with the other attributes of Table -1.

As quitting rate of population γ increases regular smokers population class Y(t) decreases i.e. The effect of γ on Y(t) shows an inverse proportionality between them, as they are two different and opposite behaviour population classes. So as γ increases there will be a gradual decrease in regular population class Y(t), which is clearly presented in the graph and labelled as Figure 6a.

As quitting rate of population γ increases, quit smokers population class Z(t) increases. So as γ increases there is a definite increase in quitting population class Z(t), which is clearly presented in the graph and labelled as Figure 6b.

Figure 7a shows the variations in snuffing population class $H_1(t)$ for various values of ρ along with the other attributes of Table 1.

As the death rate of snuffing population class ρ increases snuffing population class $H_1(t)$ is decreasing, which is shown in the figure 7a

Figure 8a and 8b shows the variations in irregular population class $H_2(t)$ and quit population class for various values of d along with the other attributes of Table 1.

As the rate of the death due to tobacco related disease d increases, irregular population class $H_2(t)$ is decreasing, which is shown in the figure 8a. In this graphical simulation, death rate plays as physiological effect on irregular population class $H_2(t)$, which results in a decrease in irregular population class $H_2(t)$(there may be interrelated decrease in regular population class).

As the rate of the death due to tobacco related disease d increases, quit smoking population class Z(t) is increasing, which is shown in the figure 8b. In this graphical simulation, death rate plays a major role and an instant effect on quit smoking population class Z(t), which results in an increase in quit smoking population class Z(t).

5. Concluding remarks:

We conducted our research using a stochastic tobacco smoking model that included snuffing users. By considering the effect of noise on the smoking model and analysing stability with the corresponding driving factors, its dynamics are investigated in terms of stochasticity. The effect of additive white noise on the proposed system are presented in the form of environmental fluctuations close to the positive equilibrium was examined and justified with numerical simulation also. The population variances are calculated and checked for stability in MATLAB. These population variances play a crucial role in analysing the stability of the system, according to the numerical simulation and analytical results of the cigarette smoking model noise system model. Large oscillation variations around the equilibrium point are produced by noise on the equation in a noisy environment, suggesting that our system is periodic. Numerical replications show that the system's trajectories oscillate arbitrarily with a wide range of amplitudes, with the noise strength initially growing but eventually oscillating, as seen in the graphs (1–8). We come to the conclusion that stochastic perturbation results in a large shift in the intensity of the dynamical scheme under consideration, leading to significant environmental variations.

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