

Design of Computer Methods for the Solution of Cervical Cancer Epidemic Model

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Received: 04 April 2021; Accepted: 15 May 2021

Abstract: Nonlinear modelling has a significant role in different disciplines of sciences such as behavioral, social, physical and biological sciences. The structural properties are also needed for such types of disciplines, as dynamical consistency, positivity and boundedness are the major requirements of the models in these fields. One more thing, this type of nonlinear model has no explicit solutions. For the sake of comparison its computation will be done by using different computational techniques. Regrettably, the aforementioned structural properties have not been restored in the existing computational techniques in literature. Therefore, the construction of structural preserving computational techniques are needed. The nonlinear model for cervical cancer is constructed by parametric perturbation technique. Well-known computer methods are considered for the computation of cervical cancer dynamics. The well-known existing methods in literature are Euler Maruyama, Euler and Runge Kutta. Nonstandard finite difference method or Implicitly driven explicit method is first time considered for aforesaid model under the assumptions given by Mickens in a stochastic way. Unfortunately, the aforementioned existing methods did not reinstate structural properties of cervical cancer dynamics in the human population. Our planned method is structural preserving and a powerful tool for all nonlinear models of biomedical engineering problems. We have verified that existing computational methods do not preserve dynamical properties. But, the implicitly driven explicit method is a good



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device for dynamical properties. In the support of assertions, convergence analysis of implicitly driven explicit method is presented.

Keywords: Cervical cancer model; differential equations; computer methods; convergence analysis

1 Introduction

Irregular development of cells into the cervix is the basic cause of cervical cancer which may grow to the nearby parts of the body. At early stages, cervical cancer has no specific sign. After some time, when the disease grows up violently, irregular vaginal bleeding, pelvic pain and having discomfort sex activity are realized as symptoms of the disease. Teenaged women may also suffer from cervical cancer. Commonly, 80% cases of this disease are found in developing states. Because of the disease, almost 200,000 deaths befell out of 500,000 in a year throughout the world. This slow-growing disease may have chances of prevention and treatment, if it is detected at early stages. Many women in the age of 20 s and 30 s are identified with precancerous variations, but mostly women in the age of mid-50 s are identified that they have cervical cancer [1,2]. This is the age difference in which precancerous changes are measured and detected. In this age, when cancer is explored, it can be prevented by specific diets. Body of every one is constructed by a number of various living cells. Ordinary body cells are produced and split into new cells in the early years of life. Cells split faster in the children than that of adults, as they require faster growth. Gradually, when any one gets adult; the cell division substitutes the dead cells and repairs the worn-out cells. When cells of any part of the body get out of appropriate development control, cancer occurs. There are numerous types of cancers. But every type of cancer twitches only due to irregular development of cells. Generally, the cancer is named by the part where it grows initially, later on it may spread to other parts of the body. Cervical cancer is an anomalous type of cancer which initiates in the uterine, the finishing lower side of the uterus that is linked with the upper vagina. Cervical cancer cases are occurred throughout the world, particularly, it is considered the second largest cancer world-widely in women. About 530,000 fresh belongings to cervical cancer are noticed and analyzed every year. According to modern global study, almost 275,000 women expire due to this cancer. Only in the U.K. almost 2,800 examples of the disease are reported per annum out of which about 1,000 are women expired. In 2004, the ratio was 282 cases and in 2005 due to this cancer 127 women expired. The existence rate in during five years in Scotland from 1997 to 2001 was 70.6%. It was reported from Canada in 2008 that almost 1,300 women were noticed and diagnosed from which 380 women were expired. The ratio of expiry in women is 80%, which regularly ensue in developed countries. The fourth utmost cancer in women in Pakistan is cervical carcinoma. In Pakistan, the cervical cancer is low as associated with the several western states, but the amount of mortality is higher. Nearly, all types of cervical cancer are because of long standing, associated with Human Papillomavirus Infection (HPV). There are over 100 types of HPVs and only certain types are caused by cancer [3–6]. Other HPV types develop warts on the skin or genital parts called “high risk.” HPV types cause cancer of cervix as well as cancer of the penis in men. HPVs may also cause the cancer of the mouth, throat and anus in people of both sexes. HPV is a very common sexually transmitted infection (STI) and epidemiology of this infection and the molecular studies show its etiologic management for cervical cancer. There are more than 40 kinds of HPV affecting the puberty parts of males and females, the outer skin of the penis, vulva (range external from vagina), anus, inside layer of vagina, rectum and cervix. Mostly people remain totally unaware that HPV has infected them. HPV-16 is one of the highly risky types of HPV (more than 56%) of all cervical cancers. Around 70% of all cancers

are because of HPV-16 and HPV-18. Nearby, 500,000 precancerous cells change of the vulva, vagina and cervix are identified. Even though all types of cervical cancers are associated with HPV, mostly genital infections may not be the source of cervical cancer in the United States. Almost twenty million American people are presently affected from HPV. At any time, at least half of sexually active (50%) males and females may attain genital HPV infection in their lives. Among the age of 15 to 25 years, nearly half of the infection is found. It was found that most of the genital HPV spreads through genital meeting during vaginal, oral and anal sex. It also turns over from the body fluid and mother-child transformation through birth. Having several sexual spouses or having intercourses by such males who have several sexual spouses, may increase the risk of the disease in women. In June 2006 a vaccine was introduced by the food and drugs administration (FDA) for the anticipation of protection from HPV-16 and HPV-18 (that causes 70% of all cervical cancers). These types of HPV infection after being detected at its initial stage having a pap smear or Papanicolaou test may be treated [7–12].

2 Literature Survey

A qualitative study was conducted by Wal boomers et al. in 1999, with eight focus groups (having 102 number of samples) with eleven questions drawn from “the health belief model.” The findings from the focus groups were that here was a deficiency of factual information around cervical cancer. So, the womenfolk stood unaware of the causal features and protective approaches concerning cervical cancer. The results exposed that main structural fences were lack of information as well as time and financial aspects. Denied misunderstandings and anxiety are the main psychological barriers to undertaking Pap tests and medical education advice impacts on contributors. To increase inspiration and knowledge, and to lessen certain barriers, recommendations are made [13,14]. In 2004, Raley et al. [15] suggested that HPV infection is the contributing agent of genital warts and cervical neoplasia. To prevent infection with HPV, a vaccine has been developed recently that may become a part of gynaecology. A survey was conducted by the researcher of American college of obstetricians and Gynaecologists (ACOG) to explore approaches towards vaccination of HPV. In 2004, Allison Friedman recommended that human papillomavirus (HPV) infection is the utmost mutual sexually transmitted and causing cervical cell irregularities, genital warts and cervical cancer in females in U.S.A. to prevail information about this virus in the individuals of general public, 35 groups were made stratified by, gender, race and location. The groups enhanced their information, thoughts and ideas about hypothetical HPV and HPV vaccines. In 2005 Sheila and Twin took a model of 467 Chinese women respectively to determine information about cervical cancer. The results revealed that female’s information around cervical cancer and precautionary or protective approaches play an important role in their screening practices. In 2006, the infectious disease obstetric Gynaecology journal recommended that in the near future, vaccines for cervical cancer (HPV 16 and HPV 18 types) will be available in the market. HPV vaccines being prophylactic, would be directed earlier to the detection of the virus at the age of adolescence or before or preadolescence. In 2007, Kwan et al. supervised a study to seek Chinese women’s vision about the HPV vaccine and their will to be vaccinated. The results showed that only 38% of the members had ever heard of HPV cancer while 50% had some knowledge of its vaccination and only 88% of them showed their concern about the cancer and wanted to be vaccinated. In 2007, Nubia Munoz et al. took an experimental group of 17,622 women of 15 to 26 years of age. These all were registered in one of the two placebo-controlled efficiency tribunals used for HPV (6, 11, 16 and 18) vaccine (first patient on 28 December 2001 and studies accomplished on 31 July 2007). The vaccine was dosed on day 1st, 2nd month and 6th month. Cervical vaginal sampling from the women was collected for pap testing at day one of every month up to 12 months regularly.

The result showed 100% effectiveness of the vaccine to lessen the dangers of HPV (16 and 18) as well as HPV-11 and genital warts [16–18]. Chao et al. presented in 2007, the learning amongst 34,193 women who were using HPV vaccination. The results showed that the completion rate was 47.1% amongst the patients of age 18 to 26 years and 41.9% amongst the patients of age from 9 to 17 years. In 2008, Caron et al, took college women to conduct a cross-sectional study. The results showed HPV as the main cause of cervical cancer and second ordinary source of cancer related mortality among the women. Foremost objective behind this analysis is to find out the alertness of HPV and its vaccine named Gardasil, along with reorganization accessed by the college women. The results showed that 14% of them identified as precancerous and got necessary treatment. 24% of them did not pay heed during primarily three months after receiving the results. To localize the cancer about 96% of the women were not aware. For the females, by helpful pap test, these results may be useful to select treatment modalities or fitness activities in developing intervention strategies to assist them. A study on HPV vaccination was supervised by Licht et al. in 2009. Main purpose of the analysis was to observe hazards and perceptions, linked in the direction of HPV which concerned the use of HPV vaccine amongst girls who studied in colleges. A concluded self-administered review was made amongst 406 enrolled women from the age of 18 to 26 years in two public universities by cross-sectional design [19–24]. Mathematical modelling is a comprehensive way to study the dynamics of diseases in the human population. That is the reason, we have done stochastic modelling with structure preserving analysis of cervical cancer due Papillomavirus infection in humans. After this analysis, we will be capable of predicting its extinction and persistence scenarios. There are different mathematical models of cervical cancer disease that have been considered in [25]. The all-explicit schemes for ODE's and SDE's have produced non-physical oscillation for certain parameters So, these schemes are less reliable and unstable. It is notable that stochastic schemes do not protect the dynamical properties in stochastic context. Here a question arises: Can we get protection of dynamical properties for stochastic dynamical systems?

This is our real idea to construct the implicitly driven explicit scheme namely as S-NSFD for stochastic cervical cancer model. The strategy of our paper is as follows: In Section 3, we shall call deterministic and stochastic cervical cancer models and their equilibria. In Section 4, we shall describe the numerical methods for stochastic models and results are related with deterministic solutions. In Section 5, we shall discuss the covariance among the sub populaces. Finally, we shall discuss the future framework of our research.

3 Deterministic Cervical Cancer Model

In this part, we consider the cervical cancer model due to human papillomavirus infection. Let at any time t , the variables are described as $S_P(t)$ represent susceptible number of women's, $I_{HPV}(t)$ represents infected number of women with HPV, $I_{CC}(t)$ represents infectious numeral of HPV female's populace who are affected from cervical cancer and $I_{UC}(t)$ represents infectious numeral of HPV female's populace who are uninfected from cervical cancer. The dynamics of cervical cancer model is shown in Fig. 1.

Note that R_0 is cervical cancer generation number. The cervical cancer generation number has imperative role in the learning of cervical cancer subtleties.

3.2 Stochastic Cervical Cancer Model

Now, we study the vector $C = [S_p, I_{HPV}, I_{cc}]^T$, the probable variations in the cervical cancer model as tracks in [Tab. 1](#):

Table 1: Probable variations in the cervical cancer model

T_i = Transitions	P_i = Probabilities
$T_1 = [1, 0, 0]^T$	$P_1 = \mu_h \Delta t$
$T_2 = [-1, 1, 0]^T$	$P_2 = P_v S_p I_{HPV} \Delta t$
$T_3 = [-1, 0, 0]^T$	$P_3 = \mu_h S_p \Delta t$
$T_4 = [0, -1, 1]^T$	$P_4 = P_c I_{HPV} \Delta t$
$T_5 = [0, -1, 0]^T$	$P_5 = \mu_h I_{HPV} \Delta t$
$T_6 = [0, 0, -1]^T$	$P_6 = \mu_h I_{cc} \Delta t$

The expectancy of the model (5)–(7) is given as:

$$E^*[\Delta C] = \sum_{i=1}^6 P_i, \quad E^*[\Delta C] = \begin{bmatrix} (\mu_h - P_v S_p I_{HPV} - \mu_h S_p) \\ (P_v S_p - P_c - \mu_h) I_{HPV} \\ (P_c I_{HPV} - \mu_h I_{cc}) \end{bmatrix} \Delta t.$$

$$E^*[CC^T] = \sum_{i=1}^6 P_i [(T)_i][(T)_i]^T, \quad E^*[\Delta C \Delta C^T] = \begin{bmatrix} U_{11} & U_{12} & U_{13} \\ U_{21} & U_{22} & U_{23} \\ U_{31} & U_{32} & U_{33} \end{bmatrix} \Delta t.$$

$$U_{11} = (\mu_h + P_v S_p I_{HPV} + \mu_h S_p), \quad U_{12} = -P_v S_p I_{HPV}, \quad U_{13} = 0.$$

$$U_{21} = -P_v S_p I_{HPV}, \quad U_{22} = (P_v S_p + P_c + \mu_h) I_{HPV}, \quad U_{23} = -P_c I_{HPV}.$$

$$U_{31} = 0, \quad U_{32} = -P_c I_{HPV}, \quad U_{33} = (P_c I_{HPV} + \mu_h I_{cc}).$$

The drift and diffusion of model is as follows:

$$\text{drift} = \chi_1(C(t), t) = \frac{E^*[\Delta C]}{\Delta t}, \quad \text{diffusion} = \chi_2(C(t), t) = \sqrt{\frac{E^*[\Delta C \Delta C^T]}{\Delta t}},$$

So, we can easily write the SDE as:

$$dC(t) = \chi_1(C(t), t) dt + \chi_2(C(t), t) dB(t). \quad (8)$$

with terms $C(0) \geq 0$ and GBM (Geometric Brownian motion) is symbolized by $B(t)$.

3.3 Euler-Murayama Method

For the solution of SDE (8), we shall use the fundamental technique, Euler Maruyama and selected values of parameters are presented in Tab. 2.

$$C_{n+1} = C_n + \chi_1(C_n, t) \Delta t + \chi_2(C_n, t) \Delta B_n. \quad (9)$$

where the step size is represented ' Δt ' and $\Delta B_n \sim N(0, 1)$. The cancer free equilibrium of model i.e., CFE is $D = (1, 0, 0)$ and the cervical cancer generation number $R_0 = 0.7500 < 1$ this situation assists us to control the cancer in population. The cancer endemic equilibrium i.e., CEE is $E = (0.5, 0.0625, 0.4375)$ and the cervical cancer generation number $R_0 = 2.0000 > 1$ means the cervical cancer is endemic in population.

Table 2: Parameter values

Parameters	Values (years)	Source
μ_h	0.1	[26]
P_v	CFE = 0.6 CEE = 1.6	[26]
P_c	0.7	[26]
σ	0.2	Estimated

4 Parametric Perturbation Model

We shall introduce the stochastic environmental factors $P_v dt = P_v dt + \sigma dB$ in the given model as follows:

$$dS_p = (\mu_h - P_v S_p I_{HPV} - \mu_h S_p) dt - \sigma S_p I_{HPV} dB. \quad (10)$$

$$dI_{HPV} = (P_v S_p - P_c - \mu_h) I_{HPV} dt + \sigma S_p I_{HPV} dB. \quad (11)$$

$$dI_{cc} = (P_c I_{HPV} - \mu_h I_{cc}) dt. \quad (12)$$

where, $B(t)$ is called the geometric Brownian motion (GBM) and σ is unpredictability of system (10)–(12). For the solution of system (10)–(12), we shall use numerical techniques in next section.

4.1 Stochastic Euler Method

The system (10)–(12) can be written as follows:

$$S_p^{n+1} = S_p^n + h \left[\mu_h (1 - S_p^n) - P_v S_p^n I_{HPV}^n - \sigma S_p^n I_{HPV}^n \Delta B_n \right] \quad (13)$$

$$I_{HPV}^{n+1} = I_{HPV}^n + h [P_v S_p^n I_{HPV}^n - P_c I_{HPV}^n - \mu_h I_{HPV}^n + \sigma S_p^n I_{HPV}^n \Delta B_n] \quad (14)$$

$$I_{cc}^{n+1} = I_{cc}^n + h [P_c I_{HPV}^n - \mu_h I_{cc}^n] \quad (15)$$

where “h” is called the time step size and ΔB_n : ($n = 1, 2$), i.e., $\Delta B_n \sim N(0, 1)$.

4.2 Stochastic Runge-Kutta Method

The system (10)–(12) can be written as follows.

First Stage

$$k_1 = h[\mu_h(1 - S_p^n) - P_v S_p^n I_{HPV}^n - \sigma S_p^n I_{HPV}^n \Delta B_n].$$

$$m_1 = h[P_v S_p^n I_{HPV}^n - P_c I_{HPV}^n - \mu_h I_{HPV}^n + \sigma S_p^n I_{HPV}^n \Delta B_n].$$

$$n_1 = h[P_c I_{HPV}^n - \mu_h I_{cc}^n].$$

Second Stage

$$k_2 = h \left[\mu_h \left(1 - \left(S_p^n + \frac{k_1}{2} \right) \right) - P_v \left(S_p^n + \frac{k_1}{2} \right) \left(I_{HPV}^n + \frac{m_1}{2} \right) - \sigma \left(S_p^n + \frac{k_1}{2} \right) \left(I_{HPV}^n + \frac{m_1}{2} \right) \Delta B_n \right].$$

$$m_2 = h \left[P_v \left(S_p^n + \frac{k_1}{2} \right) \left(I_{HPV}^n + \frac{m_1}{2} \right) - P_c \left(I_{HPV}^n + \frac{m_1}{2} \right) - \mu_h \left(I_{HPV}^n + \frac{m_1}{2} \right) + \sigma \left(S_p^n + \frac{k_1}{2} \right) \left(I_{HPV}^n + \frac{m_1}{2} \right) \Delta B_n \right]$$

$$n_2 = h \left[P_c \left(I_{HPV}^n + \frac{m_1}{2} \right) - \mu_h \left(I_{cc}^n + \frac{n_1}{2} \right) \right].$$

Third Stage

$$k_3 = h \left[\mu_h \left(1 - \left(S_p^n + \frac{k_2}{2} \right) \right) - P_v \left(S_p^n + \frac{k_2}{2} \right) \left(I_{HPV}^n + \frac{m_2}{2} \right) - \sigma \left(S_p^n + \frac{k_2}{2} \right) \left(I_{HPV}^n + \frac{m_2}{2} \right) \Delta B_n \right].$$

$$m_3 = h \left[P_v \left(S_p^n + \frac{k_2}{2} \right) \left(I_{HPV}^n + \frac{m_2}{2} \right) - P_c \left(I_{HPV}^n + \frac{m_2}{2} \right) - \mu_h \left(I_{HPV}^n + \frac{m_2}{2} \right) + \sigma \left(S_p^n + \frac{k_2}{2} \right) \left(I_{HPV}^n + \frac{m_2}{2} \right) \Delta B_n \right]$$

$$n_3 = h \left[P_c \left(I_{HPV}^n + \frac{m_2}{2} \right) - \mu_h \left(I_{cc}^n + \frac{n_2}{2} \right) \right].$$

Fourth Stage

$$k_4 = h[\mu_h(1 - (S_p^n + k_3)) - P_v(S_p^n + k_3)(I_{HPV}^n + m_3) - \sigma(S_p^n + k_3)(I_{HPV}^n + m_3)\Delta B_n].$$

$$m_4 = h[P_v(S_p^n + k_3)(I_{HPV}^n + m_3) - P_c(I_{HPV}^n + m_3) - \mu_h(I_{HPV}^n + m_3) + \sigma(S_p^n + k_3)(I_{HPV}^n + m_3)\Delta B_n]$$

$$n_4 = h[P_c(I_{HPV}^n + m_3) - \mu_h(I_{cc}^n + n_3)].$$

Final Stage

$$\left. \begin{aligned} S_p^{n+1} &= S_p^n + \frac{1}{6}[k_1 + 2k_2 + 2k_3 + k_4] \\ I_{hpv}^{n+1} &= I_{hpv}^n + \frac{1}{6}[m_1 + 2m_2 + 2m_3 + m_4] \\ I_{cc}^{n+1} &= I_{cc}^n + \frac{1}{6}[n_1 + 2n_2 + 2n_3 + n_4] \end{aligned} \right\} \quad (16)$$

where “ h ” is called the time step size and $\Delta B_n: (n = 1, 2)$, i.e., $\Delta B_n \sim N(0, 1)$.

4.3 Stochastic NSFD Method

The system (10)–(12) can be written by using this method and presented in [27]:

$$S_p^{n+1} = \frac{S_p^n + h\mu_h}{1 + h(\mu_h + P_v I_{HPV}^n + \sigma I_{HPV}^n \Delta B_n)}. \quad (17)$$

$$I_{HPV}^{n+1} = \frac{I_{HPV}^n}{1 + h[P_c + \mu_h - P_v S_p^n - \sigma S_p^n \Delta B_n]}. \quad (18)$$

$$I_{cc}^{n+1} = \frac{I_{cc}^n + hP_c I_{HPV}^n}{1 + h\mu_h}. \quad (19)$$

where “h” is called the time step size and ΔB_n : ($n = 1, 2$), i.e., $\Delta B_n \sim N(0, 1)$.

4.4 Convergence Analysis

Theorem 1: Let for $(S_p^n(0), I_{hpv}^n(0), I_{cc}^n(0)) \in R_+^3$, the system (17)–(19) has a unique positive solution $(S_p^n, I_{hpv}^n, I_{cc}^n) \in R_+^3$ on $n \geq 0$, nearly sure.

Theorem 2: The set $\Omega = \{(S_p^n, I_{hpv}^n, I_{cc}^n) \in R_+^3 : S_p^n \geq 0, I_{hpv}^n \geq 0, I_{cc}^n \geq 0, S_p^n + I_{hpv}^n + I_{cc}^n \leq 1\}$ for all $n \geq 0$ and the system (17)–(19) has a positive invariant set.

Proof: The system (17)–(19) may be written as:

$$\frac{S_p^{n+1} - S_p^n}{h} = \left[\mu_h (1 - S_p^n) - P_v S_p^n I_{HPV}^n - \sigma S_p^n I_{HPV}^n \Delta B_n \right].$$

$$\frac{I_{HPV}^{n+1} - I_{HPV}^n}{h} = [P_v S_p^n I_{HPV}^n - P_c I_{HPV}^n - \mu_h I_{HPV}^n + \sigma S_p^n I_{HPV}^n \Delta B_n].$$

$$\frac{I_{cc}^{n+1} - I_{cc}^n}{h} = [P_c I_{HPV}^n - \mu_h I_{cc}^n].$$

By adding all the equations, we have

$$(S_p^{n+1} + I_{HPV}^{n+1} + I_{cc}^{n+1} - (S_p^n + I_{hpv}^n + I_{cc}^n)) \leq \mu_h - \mu_h (S_p^n + I_{hpv}^n + I_{cc}^n).$$

$$S_p^{n+1} + I_{HPV}^{n+1} + I_{cc}^{n+1} \leq 1.$$

nearly sure.

Theorem 3: The system (17)–(19) will be stable, if all eigen values of given system should lie in a unit circle.

For this we consider F, G and H as follows:

$$F = \frac{S_p + h\mu_h}{1 + h(\mu_h + P_v I_{HPV} + \sigma I_{HPV} \Delta B)}, \quad G = \frac{I_{HPV}}{1 + h(P_c + \mu_h - P_v S_p - \sigma S_p \Delta B)}, \quad H = \frac{I_{cc} + hP_c I_{HPV}}{1 + h\mu_h}.$$

The Jacobian matrix J defined as

$$J = \begin{bmatrix} \frac{\partial F}{\partial S_p} & \frac{\partial F}{\partial I_{HPV}} & \frac{\partial F}{\partial I_{cc}} \\ \frac{\partial G}{\partial S_p} & \frac{\partial G}{\partial I_{HPV}} & \frac{\partial G}{\partial I_{cc}} \\ \frac{\partial H}{\partial S_p} & \frac{\partial H}{\partial I_{HPV}} & \frac{\partial H}{\partial I_{cc}} \end{bmatrix}.$$

$$\text{where, } \frac{\partial F}{\partial S_p} = \frac{1}{1 + h(\mu_h + P_v I_{HPV} + \sigma I_{HPV} \Delta B)}, \quad \frac{\partial F}{\partial I_{hpv}} = -\frac{h(S_p + h\mu_h)(P_v + \sigma \Delta B)}{[1 + h(\mu_h + P_v I_{HPV} + \sigma I_{HPV} \Delta B)]^2},$$

$$\frac{\partial F}{\partial I_{cc}} = 0.$$

$$\frac{\partial G}{\partial S_p} = \frac{h I_{HPV} (P_v + \sigma \Delta B)}{[1 + h(P_c + \mu_h - P_v S_p - \sigma S_p \Delta B)]^2}, \quad \frac{\partial G}{\partial I_{hpv}} = \frac{1}{1 + h(P_c + \mu_h - P_v S_p - \sigma S_p \Delta B)}, \quad \frac{\partial G}{\partial I_{cc}} = 0.$$

$$\frac{\partial H}{\partial S_p} = 0, \quad \frac{\partial H}{\partial I_{hpv}} = \frac{h P_c}{1 + h\mu_h}, \quad \frac{\partial H}{\partial I_{cc}} = \frac{1}{1 + h\mu_h}.$$

Put, $D = (1, 0, 0)$ and $R_o < 1$, we have

$$J = \begin{bmatrix} \frac{1}{1 + h\mu_h} & -\frac{h(1 + h\mu_h)(P_v + \sigma \Delta B)}{[1 + h\mu_h]^2} & 0 \\ 0 & \frac{1}{1 + h(P_c + \mu_h - P_v - \sigma \Delta B)} & 0 \\ 0 & \frac{h P_c}{1 + h\mu_h} & \frac{1}{1 + h\mu_h} \end{bmatrix}$$

$$\text{The eigen values are } \lambda_1 = \frac{1}{1 + h\mu_h} < 1, \quad \lambda_2 = \frac{1}{1 + h(P_c + \mu_h - P_v - \sigma \Delta B)} < 1 \text{ and}$$

$$\lambda_3 = \frac{1}{1 + h\mu_h} < 1.$$

Thus, all eigen values of discrete dynamical system for cancer free equilibria (CFE) lies in unit circle. So, the system (17)–(19) is stable.

5 Computer Experiments

In this unit, we shall discourse the simulations of above methods and the values of constants are presented in [26].

5.1 Euler Maruyama Method

The simulations of system (9) are presented as follows:

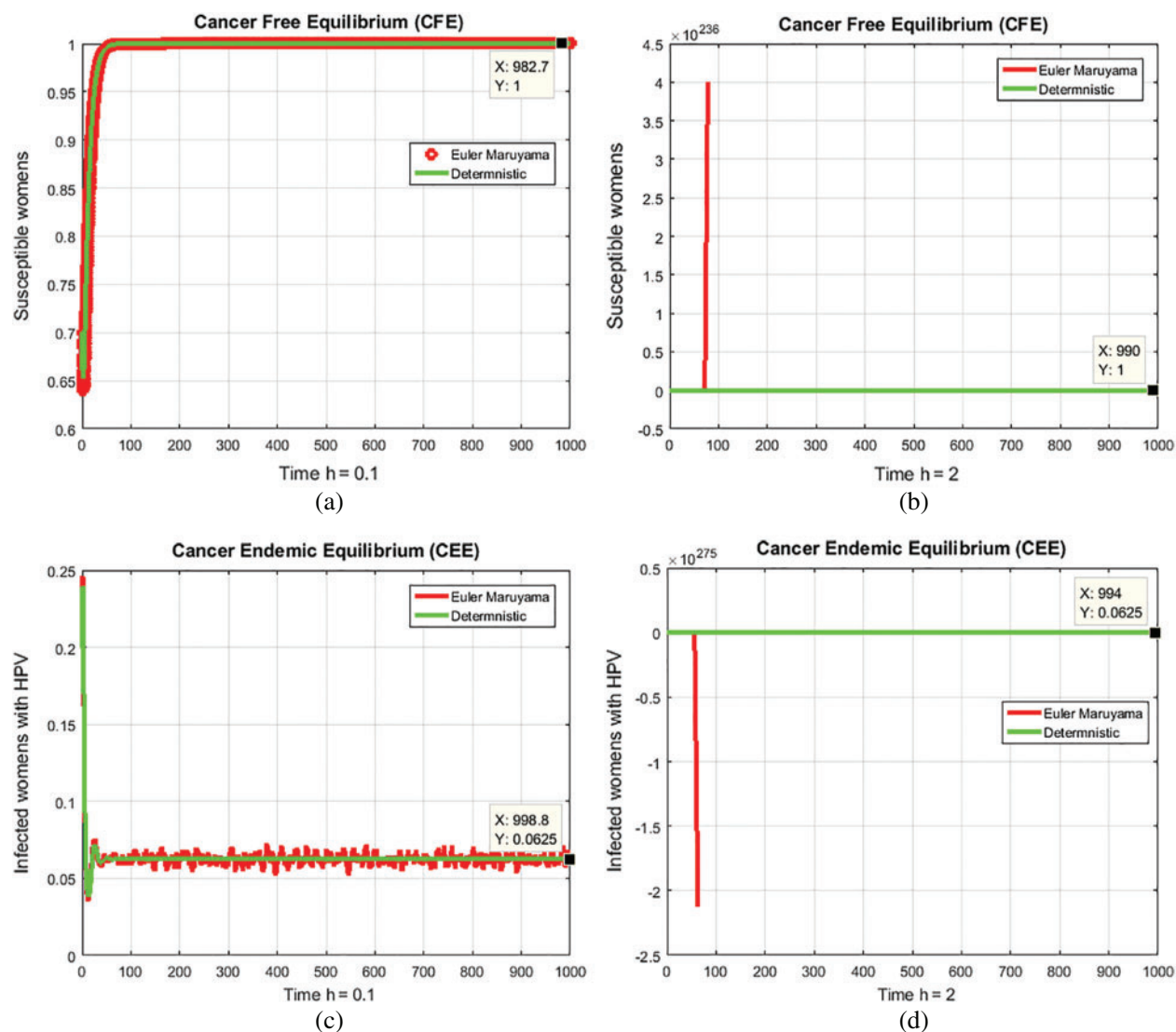


Figure 2: (a) Susceptible women's at CFE for $h = 0.1$ (b) Susceptible women's at CFE for $h = 2$ (c) Infected women at CEE for $h = 0.1$ (d) Infected women at CEE for $h = 2$

Remarks: The aforesaid method converges for parameter $h = 0.1$ in Figs. 2a and 2c But diverges in Figs. 2b and 2d.

5.2 Stochastic Euler Method

The simulations of system (13)–(15) are as follows:

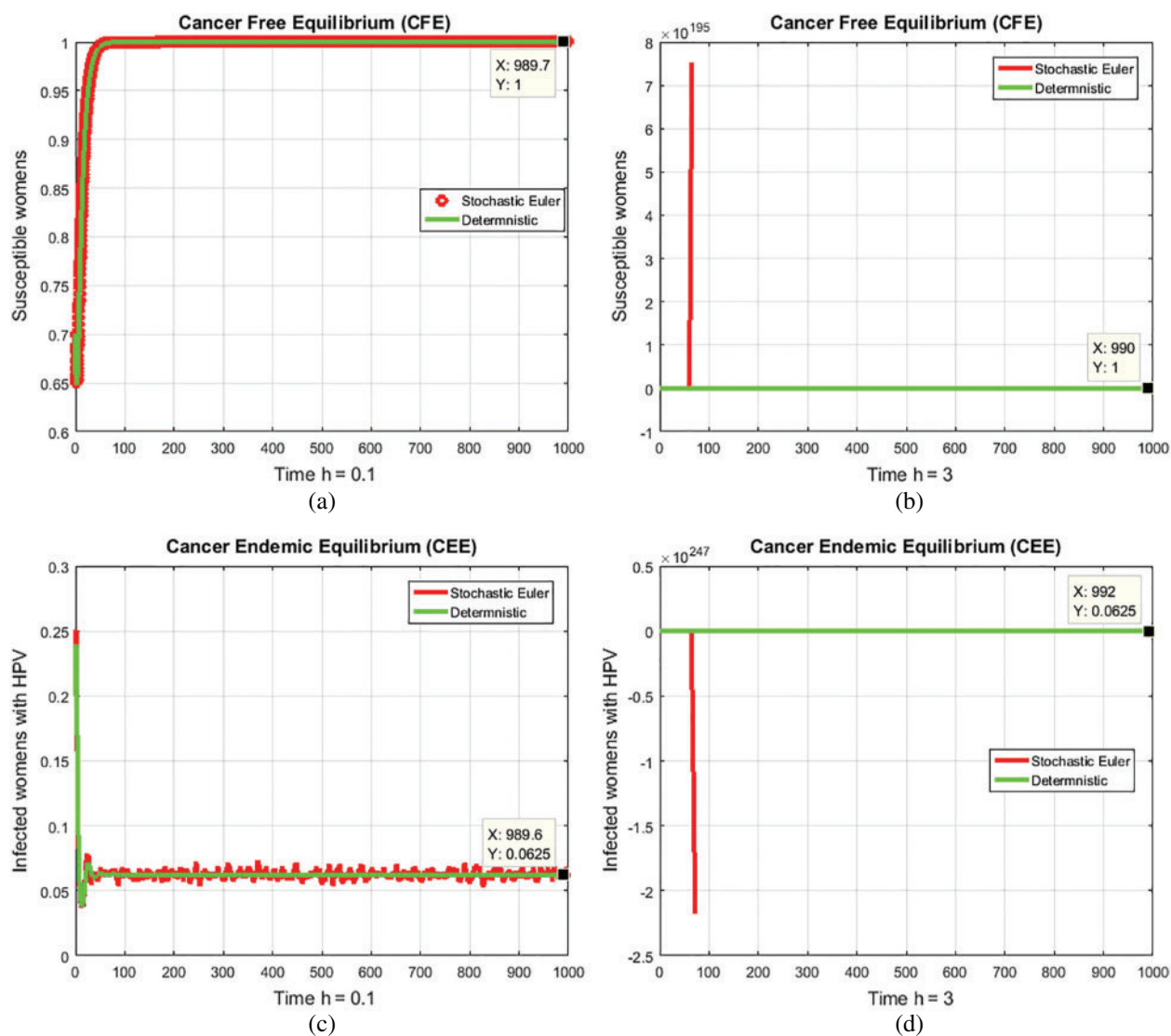


Figure 3: (a) Susceptible women's' at CFE for $h = 0.1$ (b) Susceptible women's at CFE for $h = 3$ (c) Infected women at CEE for $h = 0.1$ (d) Infected women at CEE for $h = 3$

Remarks: The aforesaid method converges for parameter $h = 0.1$ in Figs. 3a and 3c But diverges and shows negativity in Figs. 3b and 3d.

5.3 Stochastic Runge Kutta Method

The simulations of system (16) are described as follows:

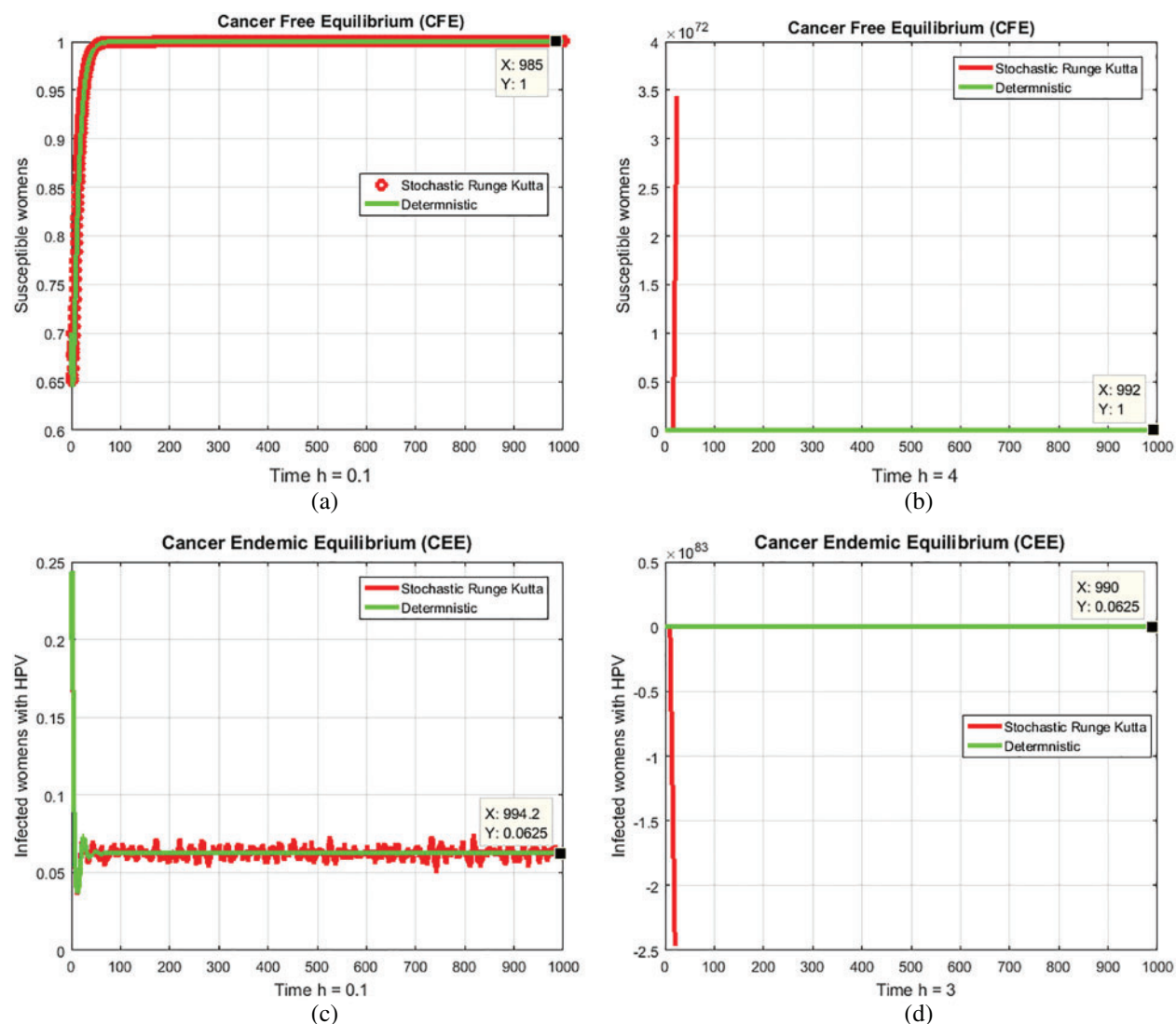


Figure 4: (a) Susceptible women's at CFE for $h = 0.1$ (b) Susceptible women's at CFE for $h = 4$ (c) Infected women at CEE for $h = 0.1$ (d) Infected women at CEE for $h = 3$

Notes: The aforesaid method converges for parameter $h = 0.1$ in Figs. 4a and 4c But diverges and shows negativity in Figs. 4b and 4d Its means the above stochastic explicit methods are time dependent. We can not observe long term qualitative behavior of system.

5.4 Stochastic NSFD Method

The simulations of system (17)–(19) are as follows:

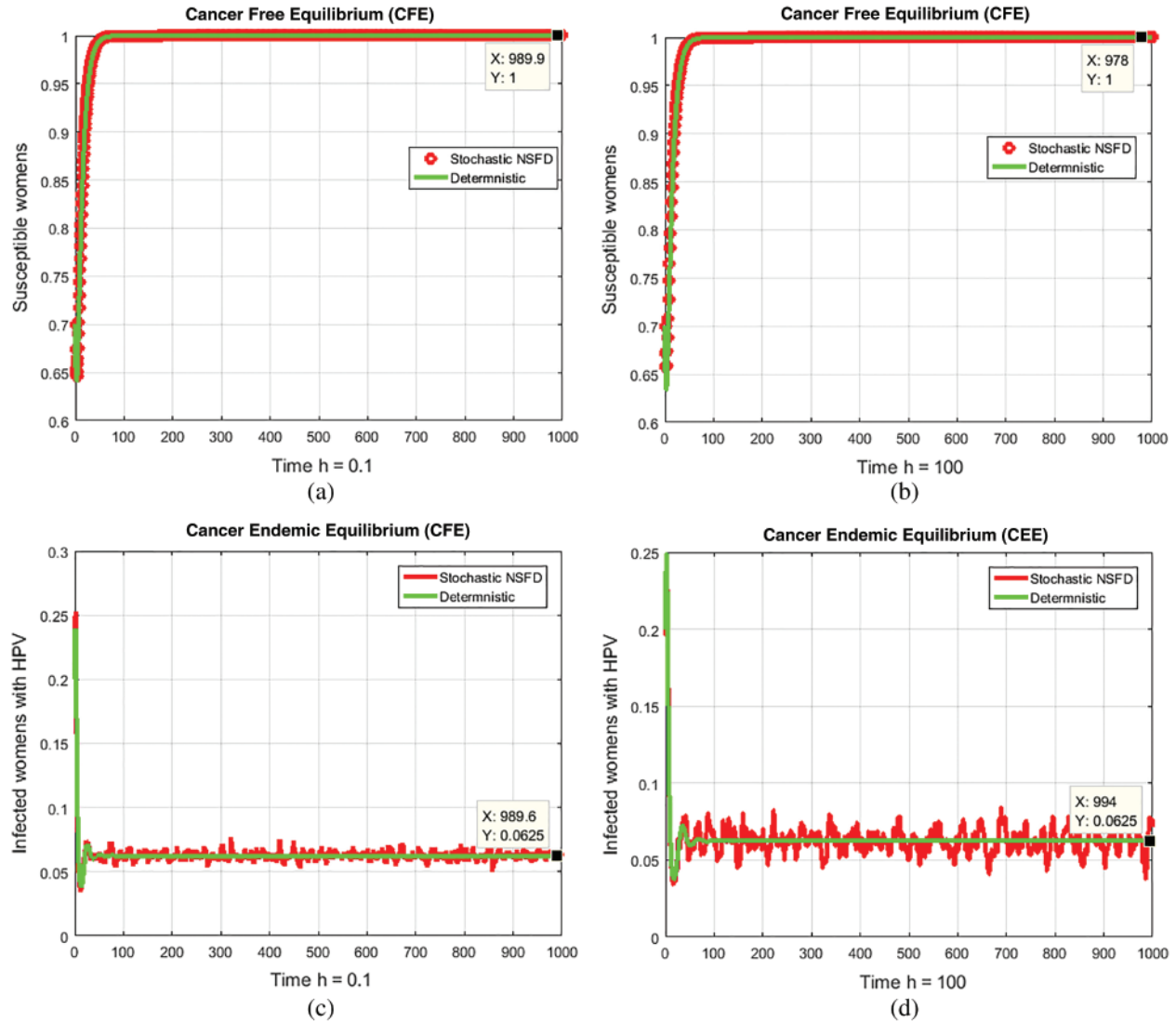


Figure 5: (a) Susceptible women's at CFE for $h = 0.1$ (b) Susceptible women's at CFE for $h = 100$ (c) Infected women at CEE for $h = 0.1$ (d) Infected women at CEE for $h = 100$

Notes: The aforesaid method converges at any step size of time in as shown Figs. 5a and 5d.

5.5 Comparison Section

We shall draw a comparison of aforesaid techniques as presented in Fig. 6 as follows:

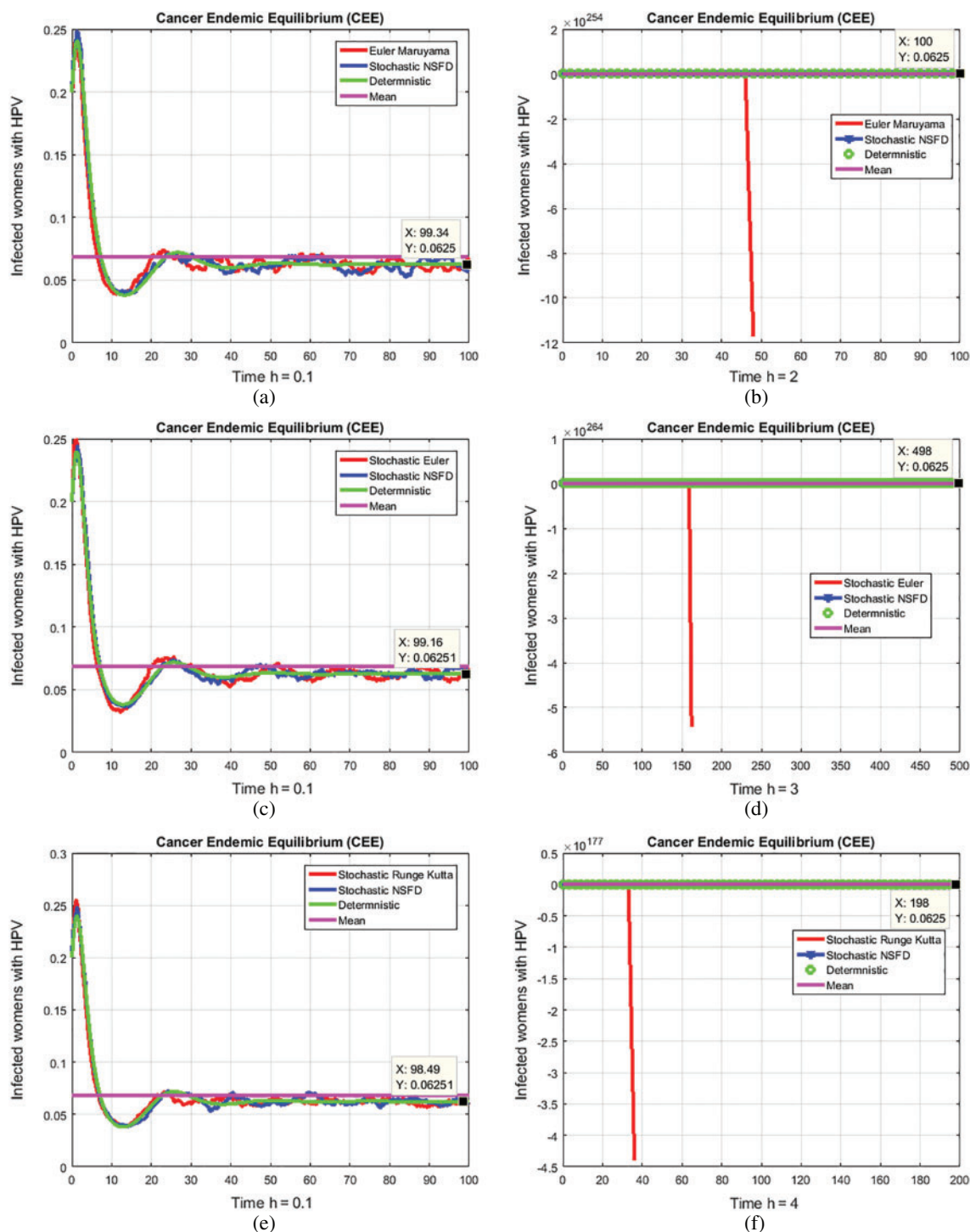


Figure 6: (a) Comparison of infected women with HPV compartment at $h = 0.1$ (b) Comparison of infected women with HPV compartment at $h = 2$. Euler Maruyama produce negativity. (c) Comparison of infected women with HPV compartment at $h = 0.1$ (d) Comparison of infected women with HPV compartment at $h = 3$ and stochastic Euler produce negativity. (e) Comparison of infected women with HPV compartment at $h = 0.1$ (f) Comparison of infected women with HPV compartment at $h = 4$ and stochastic Runge Kutta produce negativity

5.6 Covariance of Model

The covariance of population for nonlinear cervical cancer model due to human papillomavirus infection is studied here. Now, we shall calculate the correlation coefficients for checking the covariance of the model and results are described below.

Table 3: Relationship Coefficient (RC)

Sub-populations	Correlation coefficient (ρ)	Relationship
(S_P, I_{HPV})	-0.3080	Inverse
(I_{HPV}, I_{cc})	-0.3408	Inverse
(I_{cc}, S_P)	-0.7870	Inverse

In [Tab. 3](#), we can observe the terms among the compartmental population. The inverse relation holds in susceptible women and the other two populations. The population will be cancer free if there is an increase in susceptible women and decrease in remaining compartments.

6 Conclusion and Future Frame Work

We have claimed that SDEs analysis of models is most effective and real as compared to ODE's analysis of models. No doubt they are connected to each other. The stochastic explicit methods are conditionally convergent and depend on time step size. But, the NSFD method is independent of time step size. This scheme is an unconditionally convergent technique as compared to other explicit stochastic methods. This scheme preserves all dynamical properties of stochastic models such as consistency, stability, positivity and boundedness in the stochastic framework. In future, we shall extend our work in all disciplines of stochastic calculus. Moreover, we shall extend this idea in stochastic fractional models and stochastic fuzzy models as presented in [\[28–30\]](#).

Acknowledgement: We always warmly, thanks to anonymous referees.

Funding Statement: The authors received no specific funding for this study.

Conflicts of Interest: The authors declare that they have no conflicts of interest to report regarding the present study.

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