

# New Stochastic Differential Equations Model for Controlling the Spread of Viral Diseases: HIV as an Example



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### **Abstract**

The main goal of presenting this manuscript is to study the dynamic activities of HIV spread and how to prevent it. The disease behavior was studied by proposing a new stochastic mathematical model. The solution's existence and uniqueness for the proposed mathematical model have been proven. We have also demonstrated the restrictions that must be met for society to rid itself of the scourge of the transmission of HIV by finding the basic reproduction rate  $R_0^s$ . If  $R_0^s < 1$ , This means the disease-free point is stable. Conversely, if  $R_0^s > 1$  this means the disease-free point is unstable. The theoretical results were proven numerically through computer simulation using MATLAB.

**Keywords:** HIV disease, Basic reproduction rate, Computer simulation, Disease-free point, Stochastic differential equations (SDIs).

### Introduction

Human immunodeficiency virus, abbreviated as (HIV) is a disease that targets the human body's immune system, As a result of this targeting, the person carrying HIV becomes susceptible to many chronic and serious diseases such as tuberculosis, viral hepatitis, and some cancers [1].



Understanding and predicting HIV has become very significant because of the enormous global health load, It has caused (42.3) million deaths to date, and the infection continues to spread worldwide, and some countries report an increase in new infections where they had previously been declining[1]. Mathematical models use the language of mathematics to describe a particular dynamic system. To interpret the experimental results and understand the basic mechanisms that influence the spread of HIV, mathematical models of the dynamics of HIV, the virus that causes AIDS, have been developed [2-7]. Ordinary differential equations (ODEs) have been the primary tool used by researchers to understand how AIDS spreads since its first appearance in 1981[8-12]. Stochastic differential equations (SDEs) have gained attention in the past years and have been used to model a number of infectious diseases such as viral hepatitis, coronavirus disease, and HIV [13-22]. In this manuscript, we will use the stochastic differential equations model to understand how HIV spreads and ways to prevent it. In this work, we relied on the stochastic differential equations model instead of the usual deterministic differential equations because the phenomenon of the spread of HIV is random and not deterministic, as it is not known with certainty how the disease spreads, and also the number of infected people is unknown. This is because no country can know the real number of people ill with HIV, and the reason is that those infected with HIV do not admit their infection for fear of isolation by society and the stigma that follows them, and for this reason there is difficulty in controlling this infectious disease. In our manuscript, we use environmental randomness in the deterministic equations model. We note that the random differential equations model is better than the deterministic equations model, since using the random differential equations system gives a distribution of expected results when used multiple times. For example, if we take the whole number of people ill with HIV at time t, while the deterministic equations model can provide us with a single probable value. This article aims to study the dynamic behavior of HIV transmission and highlight the role of prevention measures in controlling its spread. Prevention measures include antiretroviral drugs that prevent the progression of HIV to AIDS. Antiretroviral drugs can also be taken to prevent the transmission of HIV from infected mothers to their children, avoid illicit and suspicious relationships, and perform voluntary medical male



circumcision, as well as awareness and education campaigns about the seriousness of the disease. The manuscript was prepared as follows: The second section was dedicated to creating a new deterministic and stochastic model to comprehend and control the transmission of HIV. A table was also presented that explained the role of the parameters used in the new mathematical model. In The Third Section, we discuss the solution's existence and uniqueness for the proposed SDI model (2). The Fourth Section is devoted to the basic reproduction rate and how to derive the basic reproduction rate for the proposed model. Section five is devoted to discussing the existence of the disease-free points and their stability. The key outcomes are obtainable in Section Six. Finally, the Seventh Section is dedicated to the conclusion.

### Formulating the Deterministic and Stochastic Mathematical Model

To control the spread of HIV in our communities, different mathematical models have been used. In our manuscript, we have presented a mathematical model consisting of four equations, which are as follows: The equation of healthy people exposed to infection will be denoted by p(t). The equation of males with HIV is I(t). The equation of women with HIV is w(t). The equation of people who are infected and receive antiretroviral therapy and adhere to all preventive measures do not transmit the infection to their sexual partners denoted as A(t). Therefore, the proposed mathematical model will be as follows:

$$\frac{dp(t)}{dt} = M - (\rho_1 I(t) + \rho_2 w(t)) p(t) - \beta_1 p(t),$$

$$\frac{dI(t)}{dt} = (\rho_1 I(t) + \rho_2 w(t)) p(t) - (\beta_2 + q + n) I(t),$$

$$\frac{dW(t)}{dt} = qI(t) - (\beta_3 + y) w(t),$$

$$\frac{dA(t)}{dt} = nI(t) + yw(t) - \beta_4 A(t).$$
(1)

We can control the spread of HIV and protect our society from this dangerous epidemic by placing restrictions that prevent the transmission of the infection from HIV-infected people to healthy people. These restrictions will be symbolized by the symbol (1-r). These restrictions are as follows: taking antiviral medications that prevent HIV infection from turning into AIDS,

testing for HIV and other sexually transmitted diseases before marriage, voluntary medical circumcision for males, and media campaigns to raise awareness of the seriousness of the disease and how to prevent it. Restrictions on transmission of the disease from infected mothers to their children during pregnancy and breastfeeding can also be imposed, and these restrictions are symbolized by the symbol (1-t), these restrictions are formed by taking antiretroviral drugs that protect children from infection. Because the exact number of people infected with HIV, both men and women, is not known, and the number of people who do not adhere to HIV prevention measures is also not known for these reasons, we will add a random part to the proposed stochastic mathematical model as follows:

$$dp(t) = [M - (1 - r)(\rho_1 I(t) + \rho_2 w(t)) \ p(t) - \beta_1 p(t)] \ dt - (1 - r)(\sigma_1 I(t) + \sigma_2 w(t)) \ p(t) dB(t),$$

$$dI(t) = [(1-r)(\rho_1 I(t) + \rho_2 w(t)) p(t) - (\beta_2 + q + n)I(t)]dt + (1-r)(\sigma_1 I(t) + \sigma_2 w(t)) p(t)dB(t),$$

$$dw(t) = [(1-t)qI(t) - (\beta_3 + y)w(t)] dt + (1-t)\sigma_3 I(t)dB(t),$$
(2)

$$dA(t) = [nI(t) + yw(t) - \beta_4 A(t)]dt - \sigma_4 A(t)dB(t).$$

with initial conditions

$$p(0) \ge 0, I(0) \ge 0, w(0) \ge 0 \& A(0) \ge 0.$$
 (3)

To illustrate the role of each parameter used in mathematical models 1 and 2, we will present Table 1.

Table 1: Model States and Model Parameters

Parameter	Description
p(t)	People at risk of contracting HIV.
I(t)	Males infected with HIV.
W(t)	Females infected with HIV.
A(t)	Infected persons who adhere to all preventive measures do not transmit the infection
	to their sexual partners.
M	It represents the increase in population per unit time.
(1-r)	It represents the possibility of restrictions that prevent people with AIDS from
	transmitting the infection.
$ ho_1$	The rate of spread of the ill from infected men to susceptible people.
$ ho_2$	The rate of spread of the infected from infected women to susceptible people.
$eta_1$	It denotes the death rate of uninfected people per unit of time.



$\sigma_1, \sigma_2, \sigma_3$ and $\sigma_4$	It represents the random parameters in the proposed stochastic model.
$\beta_2$	It represents the mortality rate of HIV-infected men per unit time.
q	It is the average number of children with the disease that infected women have in
	the absence of antiretroviral medications.
n	The rate of transformation of infected men into people who adhere to all measures
	to prevent disease transmission.
(1-t)	It represents the possibility of taking antiretroviral drugs to prevent the transmission
	of HIV from mothers to their children.
$\beta_3$	It represents the mortality rate of HIV-infected women per unit time.
у	The rate of transformation of infected women into people who adhere to all
	measures to prevent disease transmission.
$\beta_4$	It represents the death rate of infected people adhering to preventive measures per
	unit of time.
B(t)	It symbolizes independent standard Brownian motions.

### **Existence and Uniqueness**

This section will discuss the solution's existence and uniqueness for the proposed stochastic differential equation model 2.

**Theorem 1**. The solution (p(t), I(t), w(t), A(t)) of the proposed stochastic epidemiological model 2 of the spread of HIV is unique on  $t \ge 0$  for every primary value  $(p(0), I(0), w(0), A(0)) \in R_+^4$ . Also, a solution must stay in  $R_+^4$  with a possibility of one, i.e.  $(p(t), I(t), w(t), A(t)) \in R_+^4$  for any  $t \ge 0$ .

**Proof**. Because all coefficient of the new proposed model are global Lipschitz continuous, for any  $(p(0), I(0), w(0), A(0)) \in R_+^4$  for this reason, there is a single local solution (p(t), I(t), w(t), A(t)) on  $t \in [0, \tau_e)$ , where  $\tau_e$  is the greatest time. To clarify that the solution is global, we need only to prove  $\tau_e = \infty$  a.s. suppose that  $L_0 \ge 0$  is big enough, so we will get p(0), I(0), w(0) and  $A(0) \in \left[\frac{1}{L_0}, L_0\right]$  for all integers  $\ge L_0$ . The ideal downtime is defined as follows:

$$\tau_{L} = \left\{ t \in [0, \tau_{e}) : \min\{p(t), I(t), w(t), A(t)\} \le \frac{1}{L} \text{ or } \max\{p(t), I(t), w(t), A(t)\} \right.$$

$$\ge L \right\}. \tag{4}$$



In this manuscript. We will consider  $\inf \emptyset = \infty$  and  $\emptyset$  means the empty set. It is clear that,  $\tau_L$  is increasing when  $L \to \infty$ .

Let us define  $\tau_{\infty} = \lim_{L \to \infty} \tau_L$ , whence  $\tau_{\infty} \le \tau_e$  a.s. To end the proof, we must prove that  $\tau_{\infty} = \infty$  a.s, then  $\tau_e = \infty$  and  $(p(0), I(0), w(0), A(0)) \in R_+^4$  a.s for all  $t \ge 0$ . If this assertion is untrue, then there are two numbers T > 0 and  $\varepsilon \in (0,1)$  such that

$$P\{\tau_{\infty} \le T\} > \epsilon. \tag{5}$$

So, there is a number  $L_1 \ge L_0$  s.t

$$P\{\tau_L \le T\} \ge \epsilon \text{ For any } L \ge L_1.$$
 (6)

We take a function and define it as follows:  $v: R_+^4 \to R_+$  by  $v(p(t), I(t), w(t), A(t)) = p + I + w + A + 1 - (\log p(t) + \log I(t) + \log w(t) + \log A(t))$ .

This function's non-negativity is evident from  $u + 1 - \log(u) \ge 0$ ,  $\forall u > 0$ . Using Ito's formula [13], we will get

$$\begin{split} dv(p(t),I(t),w(t),A(t)) &= \\ & \left[ \left( 1 - \frac{1}{p(t)} \right) \left[ [M - (1-r)(\rho_1 I(t) + \rho_2 w(t)) p(t) - \beta_1 p(t)] \right] + \\ & \left( 1 - \frac{1}{I(t)} \right) \left[ (1-r)(\rho_1 I(t) + \rho_2 w(t)) p(t) - (\beta_2 + q + n) I(t) \right] + \\ & \left( 1 - \frac{1}{w(t)} \right) \left[ (1-t) q I(t) - (\beta_3 + y) w(t) \right] + \left( 1 - \frac{1}{A(t)} \right) \left[ n I(t) + y w(t) - \beta_4 A(t) \right] + \\ & \frac{1}{2} (1-r)^2 \left( \sigma_1 I(t) + \sigma_2 w(t) \right)^2 + \frac{1}{I(t)^2} 0.5 (1-r)^2 \left( \sigma_1 I(t) + \sigma_2 w(t) \right)^2 p(t)^2 \\ & + \frac{1}{w(t)^2} 0.5 (1-t)^2 \sigma_3^2 I(t)^2 + 0.5 \sigma_4^2 \right] dt \\ & + \left[ (1-r) \left( \sigma_1 I(t) + \sigma_2 w(t) \right) - (1-r) \left( \sigma_1 I(t) + \sigma_2 w(t) \right) p(t) \right. \\ & + (1-r) \left( \sigma_1 I(t) + \sigma_2 w(t) \right) p(t) - \frac{(1-r)(\sigma_1 I(t) + \sigma_2 w(t)) p(t)}{I(t)} + (1-t) \sigma_3 I - \frac{(1-t)\sigma_3 I(t)}{w(t)} \\ & + \sigma_4 - \sigma_4 A(t) \right] dB(t). \end{split}$$



$$= [M - (1 - r)(\rho_{1}I(t) + \rho_{2}w(t))p(t) - \beta_{1}p(t) + (1 - r)(\rho_{1}I(t) + \rho_{2}w(t))p(t) - (\beta_{2} + q + n)I(t) + (1 - t)qI(t) - (\beta_{3} + y)w(t) + nI(t) + yw(t) - \beta_{4}A(t) - \frac{M}{p(t)} + (1 - r)(\rho_{1}I(t) + \rho_{2}w(t) + \beta_{1} - \frac{(1 - r)(\rho_{1}I(t) + \rho_{2}w(t))p(t)}{I(t)} + (\beta_{2} + q + n) - \frac{(1 - t)qI(t)}{w(t)} + (\beta_{3} + y) - \frac{nI(t)}{A(t)} - \frac{yw(t)}{A(t)} + \beta_{4} + \frac{1}{2}(1 - r)^{2}(\sigma_{1}I(t) + \sigma_{2}w(t))^{2} + \frac{1}{I(t)^{2}}0.5(1 - r)^{2}(\sigma_{1}I(t) + \sigma_{2}w(t))^{2}p(t)^{2} + \frac{1}{w(t)^{2}}0.5(1 - t)^{2}\sigma_{3}^{2}I(t)^{2} + 0.5\sigma_{4}^{2}dt + [(1 - r)(\sigma_{1}I(t) + \sigma_{2}w(t)) - (1 - r)(\sigma_{1}I(t) + \sigma_{2}w(t))p(t) + (1 - r)(\sigma_{1}I(t) + \sigma_{2}w(t))p(t) - \frac{(1 - r)(\sigma_{1}I(t) + \sigma_{2}w(t))p(t)}{I(t)} + (1 - t)\sigma_{3}I - \frac{(1 - t)\sigma_{3}I(t)}{w(t)} + \sigma_{4} - \sigma_{4}A(t)]dB(t).$$

Hence,

$$dv(p(t), I(t), w(t), A(t))$$

$$\leq \left[ M + \beta_1 + \beta_2 + q + n + \beta_3 + y + \beta_4 + \sigma_4 + (1 - r)(\rho_1 I(t) + \rho_2 w(t)) p(t) + (1 - t)qI(t) + nI(t) + yw(t) + (1 - r)(\rho_1 I(t) + \rho_2 w(t)) + \frac{1}{2}(1 - r)^2 (\sigma_1 I(t) + \sigma_2 w(t))^2 + \frac{1}{I(t)^2} 0.5(1 - r)^2 (\sigma_1 I(t) + \sigma_2 w(t))^2 p(t)^2 + \frac{1}{w(t)^2} 0.5(1 - t)^2 \sigma_3^2 I(t)^2 + 0.5\sigma_4^2 dt + \left[ (1 - r)(\sigma_1 I(t) + \sigma_2 w(t)) + (1 - r)(\sigma_1 I(t) + \sigma_2 w(t)) + (1 - r)(\sigma_1 I(t) + \sigma_2 w(t)) p(t) + (1 - t)\sigma_3 I - \frac{(1 - t)\sigma_3 I(t)}{w(t)} dB(t) \right]$$

$$= K.$$
(7)

For this, we will get



 $E[v(p(t)(\tau_L \wedge T), I(t)(\tau_L \wedge T), w(t)(\tau_L \wedge T), A(t)(\tau_L \wedge T)]$ 

$$\leq v(p(0), I(0), w(0), A(0)) + E\left[\int_{0}^{\tau_{L} \wedge T} K dt\right]$$

$$\leq v(p(0), I(0), w(0), A(0))$$
+ KT. (8)

Assume that  $\Omega_L = \tau_L \leq T$  for  $L \geq L_1$  and by Equation (6)  $P(\Omega_L) \geq \epsilon$ . We find that for each  $\omega \in \Omega_L$ , there is one of these values  $p(t)(\tau_L, \omega)$ ,  $I(t)(\tau_L, \omega)$ ,  $w(t)(\tau_L, \omega)$ ,  $A(t)(\tau_L, \omega)$  that is equivalent L or  $\frac{1}{L}$ , and then  $v(p(t)(\tau_L), I(t)(\tau_L), w(t)(\tau_L), A(t)(\tau_L))$  is bigger than  $L - 1 - \log L$  or  $\frac{1}{L}$  and  $L - 1 - \log L$ . As a result of this

$$v((p(t)(\tau_L), I(t)(\tau_L), w(t)(\tau_L), A(t)(\tau_L)) \ge E(L - 1 - \log L) \wedge \left(\left(\frac{1}{L}\right) - 1 + \log L\right)$$

$$(9)$$

Therefore, Equation (6) and (8) indicate this

$$v(p(0), I(0), w(0), A(0)) + KT \ge E \left[ 1_{\Omega_{(\omega)}} v((p(t)(\tau_L), I(t)(\tau_L), w(t)(\tau_L), A(t)(\tau_L)) \right]$$

$$\ge \epsilon \left[ (L - 1) - \log L \right] \wedge \left( \left( \frac{1}{L} \right) - 1 + \log L \right). \tag{10}$$

Note that  $1_{\Omega_{(\omega)}}$  is a pointer function of  $\Omega$ . Letting  $L \to \infty$  we will get a contradiction

 $\infty > v(p(0), I(0), w(0), A(0)) + KT = \infty$  This means  $\tau_{\infty} = \infty$  a.s,



#### The Basic Reproduction Rate

To comprehend the activities of HIV, how it spreads, and whether society can eliminate this infectious disease, we will use the basic reproduction rate  $R_0^s$ , which may be defined as the total number of secondary infections created via one ill person in a population that is completely at risk of contracting HIV. We found the mathematical formula for the basic reproduction rate for the new stochastic model which as follows:

$$R_0^s = \frac{(1-r)\rho_1 + (1-r)\sigma_1}{(\beta_2 + q + n)} + \frac{((1-r)\rho_2 + (1-r)\sigma_2)((1-t)q + (1-t)\sigma_3)}{(\beta_2 + q + n)(\beta_3 + y)}.$$
 (11)

### **Existence of Equilibrium Points and Their Stability**

To calculate the stability of the new stochastic model, we will calculate the steady states of this model. The new model has an equilibrium point known as the "disease-free equilibrium,". This denotes that there are no infected men and no infected women (i.e.,  $I(t)^* = 0 \& w(t)^* = 0$ ). Therefore, when solving the equations in model 2, we find the disease-free point, which is as follows:

 $(p(t)^*, 0,0,0) = \left(\frac{M}{\beta_1}, 0,0,0\right)$ . To prove the stability of the disease- free point will present the following theory.

**Theorem 2**. The disease- free point of the stochastic model 2 is asymptotically stable if  $R_0^s < 1$ , and unstable if  $R_0^s > 1$ .

**Proof.** The Jacobin matrix for the new model 2 at the disease-free point, is as follows:

$$J(p(t)^*,0,0,0) = \begin{bmatrix} -\beta_1, & -(1-r)\rho_1 p(t)^* - (1-r)\sigma_1 p(t)^*, -(1-r)\rho_2 p(t)^* - (1-r)\sigma_2 p(t)^*, & 0 \\ 0, (1-r)\rho_1 p(t)^* - (\beta_2 + q + n) + (1-r)\sigma_1 p(t)^*, (1-r)\rho_2 p(t)^* + (1-r)\sigma_2 p(t)^*, & 0 \\ 0, & (1-t)q + (1-t)\sigma_3, & -(\beta_3 + y), & 0 \\ 0, & n, & y, & -\beta_4 - \sigma_4 \end{bmatrix}$$



The characteristic equation about  $J(p(t)^*, 0, 0, 0)$  is  $|J(p(t)^*, 0, 0, 0) - \lambda I| = 0$ .

$$\begin{bmatrix} [-\beta_1] - \lambda, & -(1-r)\rho_1 p(t)^* - (1-r)\sigma_1 p(t)^*, -(1-r)\rho_2 p(t)^* - (1-r)\sigma_2 p(t)^*, 0 \\ 0, [(1-r)\rho_1 p(t)^* - (\beta_2 + q + n) + (1-r)\sigma_1 p(t)^*] - \lambda, (1-r)\rho_2 p(t)^* + (1-r)\sigma_2 p(t)^*, 0 \\ 0, & (1-t)q + (1-t)\sigma_3, & [-(\beta_3 + y)] - \lambda, & 0 \\ 0, & n, & y, & [(-\beta_4 - \sigma_4)] - \lambda \end{bmatrix} = 0.$$

The first two roots of the characteristic equation or the eigenvalues are  $\lambda_1 = -\beta_1 \& \lambda_2 = -\beta_4 - \sigma_4$ , and the other two eigenvalues can be determined via the following quadratic equation

$$P_{2}(\lambda) = \lambda^{2} - (b_{11} + b_{22})\lambda + (b_{11}b_{22} - b_{12}b_{21})$$

$$P_{2}(\lambda) = \lambda^{2} - (((1 - r)\rho_{1}p(t)^{*} - (\beta_{2} + q + n) + (1 - r)\sigma_{1}p(t)^{*}) - \beta_{4} - \sigma_{4})\lambda$$

$$-((\beta_{4} + \sigma_{4})((1 - r)\rho_{1}p(t)^{*} - (\beta_{2} + q + n) + (1 - r)\sigma_{1}p(t)^{*})$$

$$-((1 - t)q + (1 - t)\sigma_{3})((1 - r)\rho_{2}p(t)^{*} + (1 - r)\sigma_{2}p(t)^{*}))$$

$$P_{2}(\lambda) = \lambda^{2} + b_{1}\lambda + b_{2}.$$

With coefficients given by

$$\begin{split} b_1 &= - \Big( \big( (1-r)\rho_1 p(t)^* - (\beta_2 + q + n) + (1-r)\sigma_1 p(t)^* \big) - \beta_4 - \sigma_4 \Big) \\ b_2 &= - \Big( (\beta_4 + \sigma_4) \big( (1-r)\rho_1 p(t)^* - (\beta_2 + q + n) + (1-r)\sigma_1 p(t)^* \big) \\ &- \big( (1-t)q + (1-t)\sigma_3 \big) \big( (1-r)\rho_2 p(t)^* + (1-r)\sigma_2 p(t)^* \big) \Big) \end{split}$$

It is easy to prove that  $b_1 > 0$  &  $b_2 > 0$  if  $R_0^s < 1$ , then both eigenvalues have a negative real part via the Roth-Hurwitz principle. Further, since the first two eigenvalues also have, a negative real part, the disease- free point is asymptotically stable. On the contrary, if  $R_0^s > 1$ , leads to  $b_1 < 0$  &  $b_2 < 0$ , and then the eigenvalues have the non-negative real part. Hence the disease- free point is an unstable, which complete the proof



### **Main Results**

In this section, we conducted many computer simulations using the Matlab program to prove the theoretical results numerically. We found from Theorem 2 that the disease- free point of the stochastic model 2 is stable if  $R_0^s < 1$ , and unstable if  $R_0^s > 1$ . In another meaning I(t) and w(t) are exponentially stable and  $\lim_{t\to\infty} I(t) = 0$  &  $\lim_{t\to\infty} w(t) = 0$ , if  $R_0^s < 1$  While I(t) and w(t) are unstable if  $R_0^s > 1$ . we will use two examples to illustrate how antiretroviral drugs and preventive measures that prevent people with HIV from transmitting the disease contribute to stabilize the system and eliminating the spread of the HIV epidemic.

**Example 6.1**: Let us choose the parameter values as follows:

Parameter	The value
r	0.8 unit less
$ ho_1$	$10 \ day^{-1}$
$\sigma_1$	0.02
$ ho_2$	$0.1 \ day^{-1}$
$\sigma_2$	0.02
t	0.9 unit less
q	$1*10^0 day^{-1}$
$\sigma_3$	0.03
$eta_2$	$0.2  day^{-1}$
n	$1.5*10^0 day^{-1}$
$\beta_3$	$0.05*10^0 day^{-1}$
y	$4*10^0 day^{-1}$

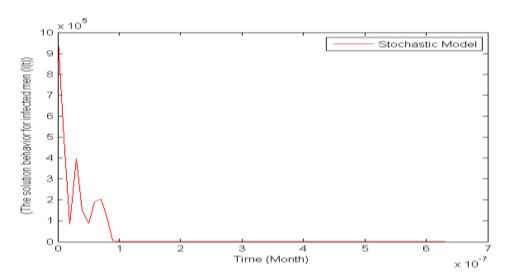
First, let's calculate a value of  $R_0^s$ . This is done by substituting the parameter values into the Equation (11), we will find its value as  $R_0^s = 0.7424500563 < 1$ . Since the value of  $(R_0^s)$  is less than one according to Theory 2 the disease-free point is stable. To prove this result numerically, we will take the equation of men infected with HIV.

$$dI = [(1-r)(\rho_1 I + \rho_2 w)p(t) - (\beta_2 + q + n)I]dt + (1-r)(\sigma_1 I + \sigma_2 w)p(t)dB(t),$$



When we substitute the values, of the parameters into the equation for injured men and use the Ito's formula [13] to solve the resulting equation, we will find that

 $I(t) = 1000e^{-(29980002.7)t}$ , so the number of men with HIV I(t) decreases and goes to zero exponentially in case  $t\rightarrow\infty$ , This is illustrated using MATLAB, as shown in Figure 1.



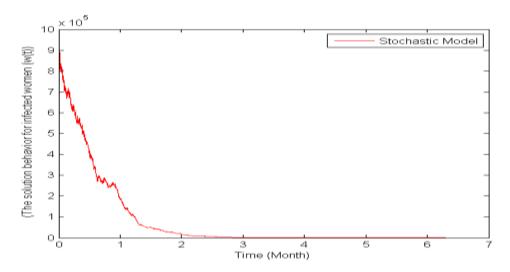
**Figure 1**: Computer simulations show that the number of men infected with HIV keeps decreasing until it reaches zero in the case  $R_0^s < 1$ .

If we take the equation of women infected with the disease

$$dw = [(1-t)qI - (\beta_3 + y)w]dt + (1-t)\sigma_3 IdB(t).$$

When we substitute the values of the parameters into the equation for women with HIV and use Ito's formula [13]. To solve the resulting equation, will find that the solution is as follows:  $w(t) = 1000e^{-3.95t}$ , this means that the number of infected women goes to zero in case  $t\rightarrow\infty$ . This is illustrated using MATLAB, as shown in Figure 2.





**Figure 2:** Computer simulations show that the number of women infected with HIV keeps decreasing until it reaches zero in the case  $R_0^s < 1$ ,

**Example 6.2**: In contrast to the first example, we assume that preventive measures (1 - r) and treatment with antiviral drugs (1 - t) are both weak and that random variance  $(\sigma_1)$ ,  $(\sigma_2)$ , and  $(\sigma_3)$  are also weak, therefore, the parameters will be as shown in the table below.

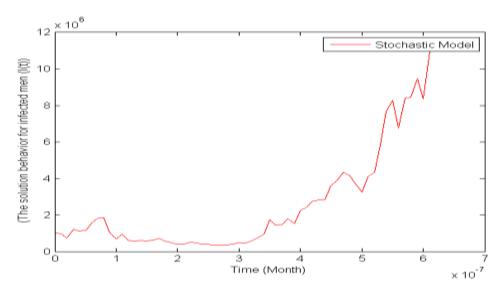
Parameter	The value
r	0.1 unit less
$ ho_1$	$10 \ day^{-1}$
$\sigma_1$	0.001
$ ho_2$	$0.1 \ day^{-1}$
$\sigma_2$	0.001
t	0.1 unit less
q	$1*10^0 day^{-1}$
$\sigma_3$	0.01
$eta_2$	$0.2 \ day^{-1}$
n	$1.5*10^0 day^{-1}$
$eta_3$	$0.05*10^0 day^{-1}$
у	$0.07*10^{0} day^{-1}$

If calculating a value of  $R_0^s$ , this is done by substituting the parameter values into Equation 11, will find its value as  $R_0^s = 3.58 > 1$ , since the value of  $(R_0^s)$  is greater than one if we apply

Theory2 the disease-free point is unstable. To prove this result numerically, will take the equation of men infected with HIV.

$$dI = [(1-r)(\rho_1 I + \rho_2 w)p(t) - (\beta_2 + q + n)I]dt + (1-r)(\sigma_1 I + \sigma_2 w)p(t)dB(t),$$

When substituting the values of the parameters into the equation for infected men and using Ito's formula [13] to solve the resulting equation, we will find the solution as follows  $I(t) = 1000e^{7469997.3t}$ , as a result, I(t) keeps increasing and does not go to zero when  $t\rightarrow\infty$ . This is illustrated using MATLAB, as in Figure 3.



**Figure 3:** Computer simulations show that the number of men infected with HIV keeps increasing and does not go to zero in the case  $R_0^s > 1$ .

If we take the equation of women infected with the disease

$$dw = [(1-t)qI - (\beta_3 + y)w]dt + (1-t)\sigma_3 IdB(t).$$

When substituting the values of the parameters into the equation for women with HIV and using Ito's formula [13] to solve the resulting equation, will find that the solution as follows  $w(t) = 1000e^{0.7799595t}$ . This means that the number of infected women does not go to zero in case  $t\rightarrow\infty$ . This is illustrated using MATLAB, as shown in Figure 4.



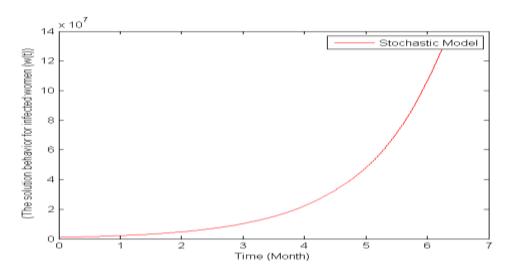


Figure 4: Computer simulations show that the number of women infected with HIV keeps increasing and does not go to zero in the case  $R_0^s > 1$ .

### **Conclusions**

What is new in our article is that we studied the behavior of HIV and its prevention by proposing a new stochastic differential equations model. This was achieved by proving the solution's existence and uniqueness for the proposed mathematical model. We have also proved that if preventive measures (1-r) and treatment with antiviral drugs (1-t) are both strong and that random variance  $(\sigma_1)$ ,  $(\sigma_2)$ , and  $(\sigma_3)$  are also strong, this gives us  $R_0^s < 1$ , this means according to Theory 2 the disease-free point is stable. Computer simulations shown in Figures1and 2 support the result. On the contrary, if preventive measures (1-r) and treatment with antiviral drugs (1-t) are both weak and that random variance  $(\sigma_1)$ ,  $(\sigma_2)$ , and  $(\sigma_3)$  are also weak this gives us  $R_0^s > 1$ . This means according to Theory 2 the disease-free point is unstable the result was confirmed by using computer simulations as shown in Figures 3 and 4.

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