

U.T. Mustapha<sup>1,2</sup>, T. Sanlidag<sup>1</sup>, E. Hincal<sup>1</sup>,  
B. Kaymakamzade<sup>1</sup>, S.M. Muhammad<sup>1</sup>, N. Gokbulut<sup>1,\*</sup>

<sup>1</sup>Near East University, TRNC, Nicosia, Turkey;

<sup>2</sup>Federal University Dutse, Nigeria;

(E-mail: umartasiustumustapha@yahoo.com, trsanlidag@yahoo.com, evren.hincal@neu.edu.tr,  
bilgen.kaymakamzade@neu.edu.tr, salisumuhammadmuhammad@gmail.com, nezihal.gokbulut@neu.edu.tr)

## Modelling the effect of horizontal and vertical transmissions of HIV infection with efficient control strategies

In this paper a mathematical model is developed to study the transmission dynamics of HIV infection and the effect of horizontal and vertical transmission in Turkey is analyzed. Model is fitted with the use of confirmed HIV cases of both vertical and horizontal transmission from 2011 to 2018. Using the next generation operator the basic reproduction number of the model is obtained, which shows whether the disease persists or dies out in time. Further analysis shows that the model is locally asymptotically stable when the basic reproduction number  $\mathcal{R}_0 < 1$  and is unstable when  $\mathcal{R}_0 > 1$ . The most sensitive parameters efficient for the control of the infection are obtained using forward normalized sensitivity index. Lastly, the results are obtained with the aid of mesh and contour plots, which show that decreasing the values of transmission rate diseases induced mortality rates and progression rates play a significant role in controlling the spread of HIV transmission.

*Keywords:* HIV, mathematical modelling, control strategies, sensitivity analysis.

### Introduction

Human Immune-deficiency Virus (HIV) reduces or destroys the human defense mechanisms, also known as the immune system, preventing fighting with infections or any other diseases and the progression of this virus occurs as a result of infecting the CD4+ T-cells of the organism [1, 2]. The number of these cells mainly shows how active and functioning the immune system is [3, 4]. The number of CD4+ T-cells must be in the range of 800 to 1200 cells/mm<sup>3</sup> for a healthy person. If this number of CD4+ T-cells goes down below 200 cells/mm<sup>3</sup> for any HIV patient, this patient is then considered to be an AIDS patient [5]. In other words, HIV is the virus that causes AIDS (Acquired Immune Deficiency Syndrome) which is the most advanced phase of the HIV infection [6].

HIV can be transmitted through direct contact with contaminated blood products, such as syringes or needles, contaminated transfusion, unprotected sexual intercourse, and breastfeeding or as a vertical transmission during birth [7]. However, not all HIV cases necessarily result in AIDS infection. It is clinically confirmed that an HIV patient may live a healthy life without progressing to severe stage (AIDS) [8].

HIV/AIDS was first discovered in the United States of America in the early 1980s in two homosexual men and it continues to progress with time [9]. 2003 was the year with the greatest number increase in an epidemic, where approximately 5 million additional infected individuals were discovered, which raised the global prevalence of the virus to 38 million people living with HIV/AIDS, and in the same year approximately 3 million patients passed away [10]. This virus happened to be the death cause of almost 25 million people as of 2005 and became one of the most devastating epidemics in history [11]. According to the statistics taken from the World Health Organization (WHO), in 2013 2.1 million people were infected and approximately 1.5 million people died because of AIDS [12]. Furthermore, in 2014 it was reported that the number of people that were living with HIV was 35 million [6].

According to the data taken from WHO, 2.3 million children were living with HIV and about 380,000 children passed away because of HIV in 2005 and approximately 2.1 million children were living with HIV/AIDS in 2007. In 2015, with 150000 newly infected children, 1.8 million children were living with HIV according to the UNAIDS

\*Corresponding author.

E-mail: nezihal.gokbulut@neu.edu.tr

and 110000 children died because of AIDS-related diseases [13]. This data shows that AIDS has become one of the major death causes. Each day about 1500 children get newly infected [14].

Several mathematical models have been developed and used to gain insight into the transmission dynamics of HIV in human population (see, for instance, [2, 6, 11, 13, 15] and some of the references therein). However, none of these studied the dynamics of HIV transmission with effect of both vertical and horizontal transmission. The purpose of the current study is to design and analyse a new realistic model (which extends some of the aforementioned studies in the literature) for HIV transmission dynamics.

This paper is organized as follows. The epidemic model is developed and analyzed in sections 2 and 3, respectively. Model fitting is presented in section 4. Section 5 contains sensitivity analysis and numerical simulation while section 6 presents the conclusions.

#### Model formulation

In this section a mathematical model is proposed to monitor the dynamics of both vertical and horizontal transmissions of HIV infections at time  $t$ . The total population  $N(t)$  is divided into four different classes; susceptible adults,  $S(t)$ , infected adults,  $I(t)$ , newborn children with no HIV infection,  $C(t)$ , and newborn children with HIV infection,  $I_c(t)$ . That is,  $N(t) = S(t) + I(t) + C(t) + I_c(t)$ . Flow diagram of the model is presented in Fig. 1.

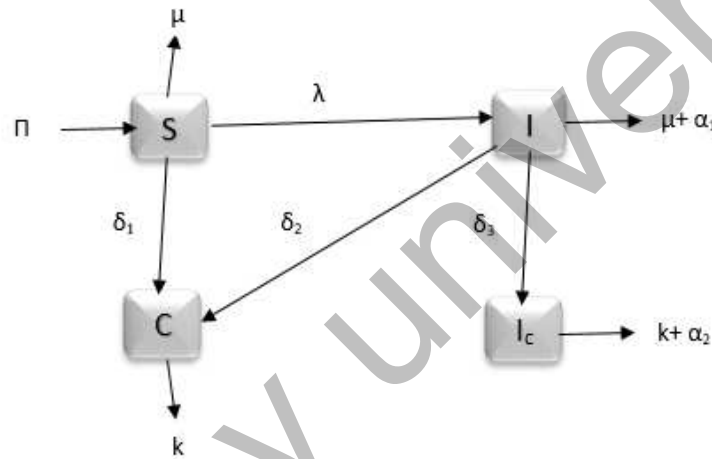


Figure 1. Flow diagram of the model.

By using the constructed model, system of ODE's is obtained as

$$\begin{aligned}
 \frac{dS}{dt} &= \Pi - \lambda S - (\delta_1 + \mu)S, \\
 \frac{dI}{dt} &= \lambda S - (\mu + \alpha_1 + \delta_2 + \delta_3)I, \\
 \frac{dC}{dt} &= \delta_1 S + \delta_2 I - kC, \\
 \frac{dI_c}{dt} &= \delta_3 I - (k + \alpha_2)I_c,
 \end{aligned} \tag{1}$$

where  $\lambda = \frac{\beta I}{N}$  is the force of infection.

Table 1

Interpretation of the State Variables Used in the Model (1).

Variables	Descriptions
$N$	Total human population
$S$	Susceptible adults (both male and female)
$I$	Infected adults with HIV (both male and female)
$C$	Newborn children
$I_c$	Newborn children with HIV infection

**Interpretation of the State Parameters Used in the Model (1).**

Parameters	Descriptions
$\phi$	Recruitment rate of both adults and new born children
$\beta$	Transmission or successful contact rate
$\alpha_1$	HIV induced mortality rate of adults
$\alpha_2$	HIV induced mortality rate of newborn children
$\mu$	Natural death for adults
$k$	Natural death for newborn children
$\delta_j (j = 1, 2, 3)$	Progression rates

*Fundamental properties of the model*

This section will highlight the quantitative analysis of HIV model (1) and briefly explain the relationship between the horizontal and vertical transmission dynamics. The persistence or elimination of HIV, which is determined by the threshold parameters, are studied. Thus, at first, the positivity and boundedness of the solutions of the model are verified for  $t \geq 0$ , and then the invariant region is studied.

*Positivity of the solutions and boundedness*

In this study to say that the model (1) is epidemiologically meaningful we need to verify the positivity of all the state variables of the model at  $t > 0$ . This means that every solution of the system (1) together with the positive initial conditions shall remain positive at any time  $t > 0$ .

*Theorem 1.* Suppose that we have initial data  $S(0) > 0, I(0) > 0, C(0) > 0, I_c(0) > 0$ . Then, the solutions of the model  $(S, I, C, I_c)$  are positive for all time  $t > 0$ .

*Proof.* It can easily be seen from the first equation of system (1) that

$$\begin{aligned} \frac{dS}{dt} &= \Pi - [\lambda(t) + \delta_1 + \mu]S \\ &\geq -[\lambda(t) + \delta_1 + \mu]S(t). \end{aligned}$$

Applying integrating factor method to the obtained inequality it is found that

$$S(t) \geq S_0 e^{-\int_0^t (\lambda(u) + \delta_1 + \mu) du} \geq 0.$$

By using the equations given in (1) and applying the same method to the equations it can be easily seen that  $I(t) \geq 0, C(t) \geq 0$  and  $I_c(t) \geq 0$  whenever  $t > 0$ .

*The invariant region*

To obtain the region the following theorem is considered.

*Theorem 2.* The solutions of the system (1) are said to be feasible for all  $t \geq 0$  whenever they enter the invariant region  $\Omega$ . That is,

$$\Omega = \left\{ (S, I, C, I_c) \in R_+^4 : S + I + C + I_c \leq \frac{\Pi}{\mu} \right\}, \text{ where } N = S + I + C + I_c.$$

*Proof.* Let  $\Omega = \{(S, I, C, I_c) \in R_+^4 : S + I + C + I_c \leq \frac{\Pi}{\mu}\}$  be the solutions of the system and assume that initial conditions are all non-negative. Then, the sum of equations of the system (1) gives

$$\frac{dN}{dt} = \Pi - \mu S - (\mu + \alpha_1)I - kC - (k + \alpha_2)I_c.$$

From the above equation it is clear that  $\frac{dN}{dt} \leq \Pi$  and integrating both sides it is obtained that  $Ne^t \leq \Pi e^t + c$ , for some arbitrary constant  $c$ . With the use of Rota and Birkhoff [16] it can be seen that  $0 \leq N \leq \frac{\Pi}{\mu}$  as  $t \rightarrow \infty$ .

This reveals that all the solutions together with the initial conditions in  $\Omega$  stay inside the region for all cases when  $t > 0$  (i.e., the set happen to be positively invariant). It is consequently adequate enough to study the dynamics of the generated flow by system (1) within the region  $\Omega$ , which guarantees the mathematical and epidemiological well-posedness of the model [2, 15, 17].

*Disease-free equilibrium (DFE) and local stability*

Let  $\chi^0 = (S_0, I_0, C_0, I_{c,0})$  be the disease-free equilibrium (DFE) of the model (1). DFE exists when the disease dies out. So, at this point there is no infection and hence, no infected individuals, i.e.,  $I_0(t) = I_{c,0}(t) = 0$ . Here, it is enough to show  $\chi^0$  attraction on the region

$$\chi^0 = \{(S_0, I_0, C_0, I_{c,0}) \in \chi^0 : I_0 = I_{c,0} = 0\}.$$

$S_0$  and  $C_0$  are obtained by equating the right hand side of the first and third equations in the system (1), and plugging 0 instead of  $I_0$  and  $I_{c,0}$ . Therefore,

$$S_0 = \frac{\Pi}{\delta_1 + \mu}$$

and

$$C_0 = \frac{\delta_1 S_0}{k} = \frac{\Pi \delta_1}{k(\delta_1 + \mu)}.$$

The DFE point of the constructed system is

$$\chi^0 = \left( \frac{\Pi}{\delta_1 + \mu}, I_0, \frac{\Pi \delta_1}{k(\delta_1 + \mu)}, I_{c,0} \right).$$

Using the next generation matrix method [18], the basic reproduction number of the HIV model (1) (denoted by  $\mathcal{R}_0 = \rho(FV^{-1})$ ,  $\rho$  is the spectral radius of the next generation matrix,  $FV^{-1}$ ) is obtained, where  $F$  stands for the matrix of new infection terms and  $V$  stands for the matrix containing the remaining transition terms of the model. Thus,

$$f = \begin{bmatrix} \frac{\beta I}{N} S_0 \\ 0 \\ 0 \end{bmatrix}, \quad v = \begin{bmatrix} (\alpha_1 + \delta_2 + \delta_3 + \mu)I \\ -\delta_2 I + kC \\ -\delta_3 I + (\alpha_2 + k)I_c \end{bmatrix},$$

$$F = \begin{bmatrix} \beta & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, \quad V = \begin{bmatrix} \alpha_1 + \delta_2 + \delta_3 + \mu & 0 & 0 \\ -\delta_2 & k & 0 \\ -\delta_3 & 0 & k + \alpha_2 \end{bmatrix}.$$

Then,  $V^{-1}$  is obtained as

$$V^{-1} = \begin{bmatrix} (\alpha_1 + \delta_2 + \delta_3 + \mu)^{-1} & 0 & 0 \\ \frac{\delta_2}{(\alpha_1 + \delta_2 + \delta_3 + \mu)k} & k^{-1} & 0 \\ \frac{\delta_3}{(\alpha_1 + \delta_2 + \delta_3 + \mu)(\alpha_2 + k)} & 0 & (\alpha_2 + k)^{-1} \end{bmatrix} \quad \text{and} \quad FV^{-1} = \begin{bmatrix} \frac{\beta}{\alpha_1 + \delta_2 + \delta_3 + \mu} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}.$$

Thus,  $\mathcal{R}_0 = \rho(FV^{-1})$  the basic reproduction number is given by

$$\mathcal{R}_0 = \frac{\beta}{\alpha_1 + \delta_2 + \delta_3 + \mu}.$$

*Endemic equilibrium*

The endemic equilibrium (EE) of the model exists only when  $I \neq 0$ ,  $C \neq 0$ , and  $I_c \neq 0$ . This means that there is a persistence of the HIV infection in the populace, and it is denoted by  $\chi^* = (S^*, I^*, C^*, I_c^*) : (S^*, I^*, C^*, I_c^*) > 0$ . Thus, the endemic equilibrium point is derived by solving the system (1) in terms of  $(\lambda) = \beta \frac{I}{N}$ , where  $(\lambda)$  is the force of infection. Then,

$$S^* = \frac{\pi}{\lambda + \mu + \delta_1},$$

$$I^* = \frac{\lambda \pi}{(\mu + \delta_3 + \delta_2 + \alpha_1)(\lambda + \mu + \delta_1)},$$

$$C^* = \frac{\pi(\lambda \delta_2 + \mu \delta_1 + \alpha_1 \delta_1 + \delta_1 \delta_2 + \delta_1 \delta_3)}{k(\mu + \delta_3 + \delta_2 + \alpha_1)(\lambda + \mu + \delta_1)},$$

$$I_c^* = \frac{\delta_3 \lambda \pi}{(\mu + \delta_3 + \delta_2 + \alpha_1)(k + \alpha_2)(\lambda + \mu + \delta_1)}.$$

*Model fitting*

This section explains the fitting of parameters involved in the proposed HIV model based upon the real cases of HIV (CD4+) in Turkey for both vertical and horizontal cases. Yearly cases are taken from 2011 to 2018 while preparing this research paper. The objective function yields to relatively small error value  $9 * 10^{-6}$ . The Fig. 2 shows the real HIV (CD4+) cases by black cycles whereas the best fitted curve of the model is shown by the black solid line. The biological parameters included in the model are listed in Table 3 along with their best estimated values obtained via least-squares technique. These parameters have finally produced the value of the basic reproduction number equivalent to  $\mathcal{R}_0 = 1.23$ .

Table 3

**Values of the Parameters of the Proposed HIV Model**

Parameter	Values	Source
$\Pi$	35	Estimated
$\beta$	0.0071	Estimated
$\alpha_1$	0.000129	Fitted
$\alpha_2$	0.000234	Fitted
$\mu$	0.0052	[2]
$k$	0.0092	[13]
$\delta_1$	0.00011	Fitted
$\delta_2$	0.00000011	Fitted
$\delta_3$	0.00044	Fitted

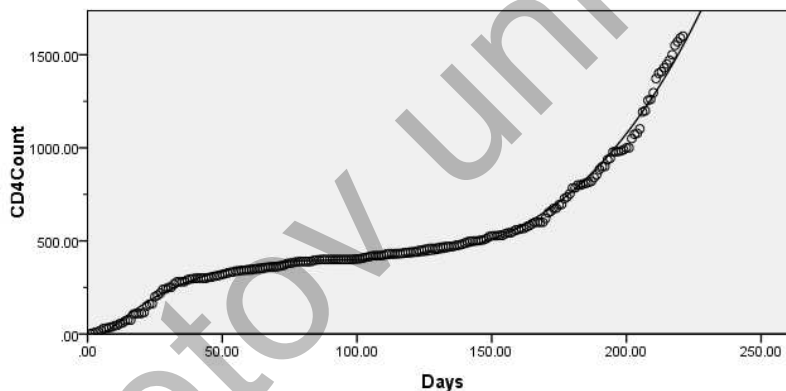


Figure 2. Data fitting for the real cases of TB (CD4+) in Turkey for both vertical and horizontal cases from 2011 to 2018

*Sensitivity analysis*

In this section the local sensitivity analysis method is used to outline the sensitivity of the basic reproduction number  $\mathcal{R}_0$  to certain key associated parameters of the proposed HIV model. The basic reproduction number was obtained and described as a parameter-dependent output of the model and the severity indicator of the HIV infection, the main way of curtailing and spreading the HIV infection in the population is to lower this reproduction number below unity.

Therefore it became crucially important to investigate the relationship between the parameters of the model and the basic reproduction number. Our main concern here is to explain the sensitivity of the basic reproduction number with respect to the significant parameters used in the model. The set of input parameters relative to  $\mathcal{R}_0$  is

$$\sigma = \{\beta, \mu, \delta_1, \delta_2, \delta_3, \alpha_1\}.$$

Typically, if a model has different parameters, variations in parameters might not always influence the outcome due to variance in the sensitivity of the parameters, those with positive sign are considered as highly and proportionally sensitive for increasing the value of  $\mathcal{R}_0$  while those with negative sign are sensitive for the

decrease of  $\mathcal{R}_0$  value and the other category are neutrally sensitive (with zero relative sensitivity) [19,20]. We denote by  $\Omega_\gamma^{R_0}$  the normalized local sensitivity index of the output  $R_0$  with respect to a parameter ( $\gamma$ ), where  $\gamma \in \sigma$ , and it is defined as [21–23]

$$\dot{\Psi}_\gamma = \Omega_\gamma^{R_0} = \frac{\gamma}{R_0} \frac{\partial R_0}{\partial \gamma} = \frac{\partial \ln(R_0)}{\partial \ln(\gamma)}.$$

Using the above definition, the following indices shown in Table 4 are computed for the output  $\mathcal{R}_0$  with respect to every parameter presented in Table 4.

Table 4

Forward Normalized Sensitivity Indices

Parameter	Elasticity Indices	Values of the Elasticity Indices
$\beta$	$\Omega_\beta^{R_0}$	1.000
$\mu$	$\Omega_\mu^{R_0}$	-0.002
$\delta_2$	$\Omega_{\delta_2}^{R_0}$	-0.285
$\delta_3$	$\Omega_{\delta_3}^{R_0}$	-0.585
$\alpha_1$	$\Omega_{\alpha_1}^{R_0}$	-0.109

Numerical simulation

Some numerical simulation results were obtained with the use of mesh and contour plots for the reproductive number as a function of two different parameters chosen from the Table 3. The results given in Fig. 3, 4, and 5 show that the value of  $\mathcal{R}_0$  increases when the values of transmission rates increases.

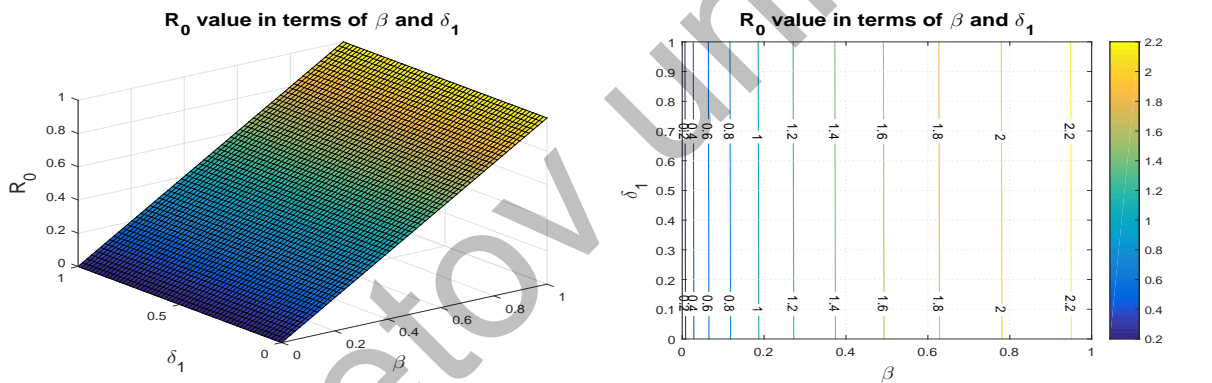


Figure 3. Profile of reproductive number in terms of transmission rate  $\beta$  and progression rate  $\delta_1$ .

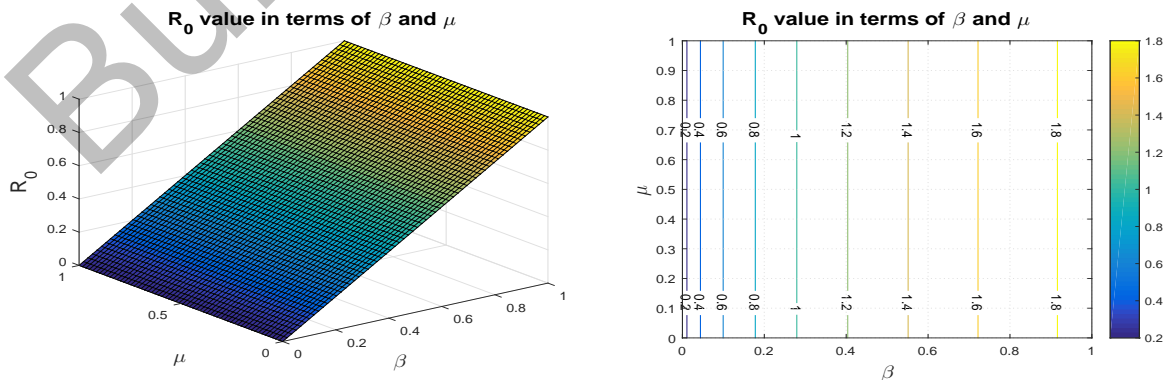


Figure 4. Profile of reproductive number in terms of transmission rate  $\beta$  and natural death rate for adults  $\mu$ .

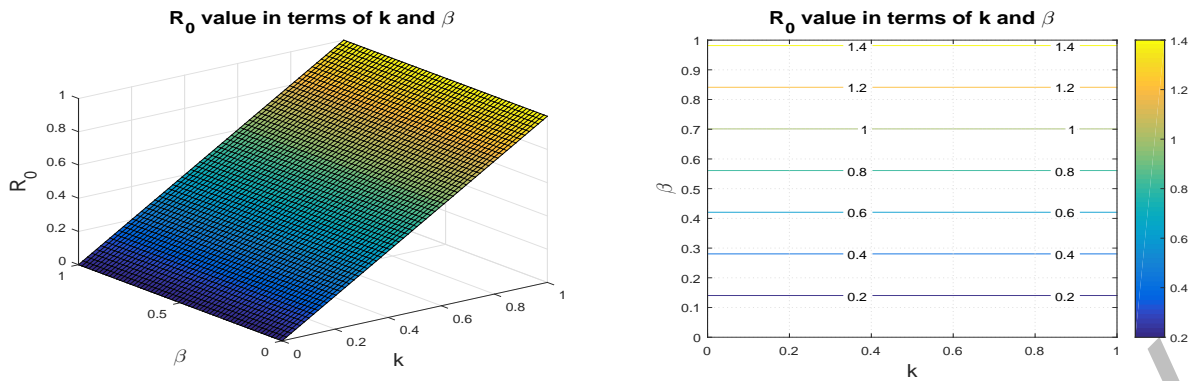


Figure 5. Profile of reproductive number in terms of transmission rate  $\beta$  and natural death rate for newborn children  $k$ .

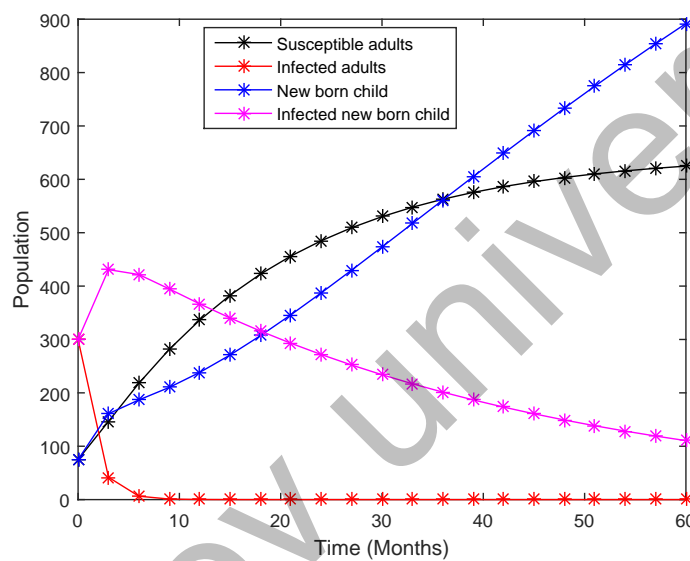


Figure 6. Profile of the total population dynamics with the respect to the parameter values in Table 2.

### Conclusions

Human Immune-deficiency Virus (HIV) reduces or destroys the human defense mechanisms known as the immune system to prevent it fighting infections and any other diseases. In this study a mathematical model is developed to study the transmission dynamics of HIV infection and the effect of horizontal and vertical transmission in Turkey is analyzed. The model is fitted with the use of confirmed HIV cases of both vertical and horizontal transmission from 2011 to 2018. Using the next generation matrix method, the basic reproduction number of the model is obtained, which shows whether disease persists or dies out in time.

Further analysis showed that the model is locally asymptotically stable when the basic reproduction number  $\mathcal{R}_0 < 1$  and is unstable when  $\mathcal{R}_0 > 1$ . The most sensitive parameters efficient for the control of the infection are obtained using forward normalized sensitivity index. The results obtained with the aid of mesh and contour plots showed that decreasing the values of transmission rate, disease induced mortality rates and progression rates play a significant role in controlling the spread of HIV transmission.

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У.Т. Мустафа<sup>1,2</sup>, Т. Санлидаг<sup>1</sup>, Э. Хинжал<sup>1</sup>, Б. Каймакамзаде<sup>1</sup>,  
С.М. Мухаммад<sup>1</sup>, Н. Гокбулут<sup>1,\*</sup>

<sup>1</sup>Таяу Шығыс университеті, Никосия, Түркия;

<sup>2</sup>Дуце федералды университеті, Нигерия

## Тиімді бақылау стратегияларының көмегімен АИТВ-инфекциясының көлденең және тік берілу әсерін модельдеу

Мақалада АИТВ-инфекциясының таралу динамикасын зерттеу үшін математикалық модель әзірленді және Түркияда инфекцияның көлденең және тік берілуінің әсері талданды. Модель 2011 жылдан бастап 2018 жылға дейін АИТВ-ның тік және көлденең берілуінің расталған жағдайларын пайдалана отырып, зерттелген. Келесі буын операторының көмегімен аурудың сақталатындығын немесе уақыт өте келе жоғалатынын көрсететін модельдің негізгі репродуктивті нөмірі алынады. Қосымша талдау көрсеткендей, базалық репродуктивті санында  $\mathcal{R}_0 < 1$  моделі локалды асимптотикалық тұрақты және  $\mathcal{R}_0 > 1$  кезінде тұрақсыз. Инфекциямен күресу үшін тиімді ең сезімтал параметрлер тікелей қалыпқа келтірілген сезімталдық индексін қолдану арқылы алынады. Торлы және контурлық графиктер арқылы алынған нәтижелер берілу жылдамдығының, аурудың, өлім-жітімнің және прогрессия көрсеткіштерінің төмендеуі АИТВ-ның таралуын бақылауда маңызды рөл атқаратынын көрсетеді.

*Кілт сөздер:* АИТВ, математикалық модельдеу, басқару стратегиялары, сезімталдықты талдау.

У.Т. Мустафа<sup>1,2</sup>, Т. Санлидаг<sup>1</sup>, Э. Хинжал<sup>1</sup>, Б. Каймакамзаде<sup>1</sup>,  
С.М. Мухаммад<sup>1</sup>, Н. Гокбулут<sup>1,\*</sup>

<sup>1</sup>Ближневосточный университет, Никосия, Турция;

<sup>2</sup>Федеральный университет Дуце, Нигерия

## Моделирование эффекта горизонтальной и вертикальной передачи ВИЧ-инфекции с помощью эффективных стратегий контроля

В статье разработана математическая модель для изучения динамики передачи ВИЧ-инфекции, и проанализировано влияние горизонтальной и вертикальной передачи инфекции в Турции. Модель адаптирована с использованием подтвержденных случаев как вертикальной, так и горизонтальной передачи ВИЧ с 2011 по 2018 годы. С помощью оператора следующего поколения получается базовый репродуктивный номер модели, который показал, сохраняется ли болезнь или исчезает со временем. Дальнейший анализ выявил, что модель локально асимптотически устойчива при базовом воспроизводственном числе  $\mathcal{R}_0 < 1$  и нестабильна при  $\mathcal{R}_0 > 1$ . Наиболее чувствительные параметры, эффективные для борьбы с инфекцией, получены с использованием прямого нормализованного индекса чувствительности. Наконец, результаты, полученные с помощью сетчатых и контурных графиков, показывают, что снижение значений скорости передачи, показателей смертности от болезней и прогрессирования играет важную роль в контроле распространения передачи ВИЧ.

*Ключевые слова:* ВИЧ, математическое моделирование, стратегии управления, анализ чувствительности.