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Hepatit-B İçin Yeni Bir Matematiksel Model ve Modelde Dikey Bulaşın Etkisi

Mehmet YAVUZ [1,2*] Naime Büşra BAYRAKTAR [1] Kübra AKYÜZ [1] Feyza Nur ÖZDEMİR [3]

[1] Necmettin Erbakan University, Faculty of Science, Department of Mathematics and Computer Sciences, Konya, Türkiye

[2] Kyrgyz-Turkish Manas University, Faculty of Science, Department of Applied Mathematics and Informatics, Bishkek, Kyrgyzstan

[3] Necmettin Erbakan University, Graduate School of Natural and Applied Sciences, Department of Computer Engineering, Konya, Türkiye

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Hepatit-B virüsü,
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Göç faktörü,
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ÖZET

Bu makalede, Hepatit virüsünün bulaşma dinamiklerini araştırmak için Hepatit B'nin yeni bir matematiksel modeli oluşturulmuştur. Model, dikey bulaşmayı dikkate alarak geliştirilmiştir. Modelde, duyarlı, latent, akut, taşıyıcı, iyileşen ve aşılanmış popülasyonlar dikkate alınmıştır. Ayrıca, pozitiflik ve hastalıksız denge noktası belirlenmiştir. Son olarak, sayısal sonuçlar elde edilmiş ve hastalığın gelecekteki seyrini tahmin etmek için biyolojik yorumları yapılmıştır.

A New Mathematical Model for Hepatitis-B and the Effect of Vertical Transmission in the Model

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ABSTRACT

In this paper, a new mathematical model of Hepatitis B is constructed to investigate the dynamics of the transmission of the Hepatitis virus. The model is developed by considering the vertical transmission. In the model, susceptible, latent, acute, carrier, recovered, and vaccinated populations are taken into account. Moreover, positivity is performed, and disease-free equilibrium point is calculated. Finally, the numerical results and their biological interpretations are performed to estimate the future directions of the disease.

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***Sorumlu Yazar:** Mehmet Yavuz, *mehmetyavuz@erbakan.edu.tr*

INTRODUCTION

Infectious diseases have caused problems for humanity throughout history. From past to present, infectious diseases that have caused social, economic and cultural losses have also caused mass deaths. In the historical process, it is known that infectious diseases such as plague, smallpox, typhus, typhoid, cholera, influenza, malaria and Hepatitis-B cause the death of many people. Various solutions and effects on the transmission routes and course of these diseases, which pose problems for humanity, are being investigated. For this reason, in recent years, especially with a major process such as a pandemic, studies on infectious disease research have begun to appear widely in the literature [54–57]. One of the infectious diseases that has been a problem throughout history is Hepatitis-B disease. Hepatitis-B is a liver infection caused by Hepatitis-B virus (HBV). It poses a global health problem because it causes many deaths [1, 2].

HBV disease, which causes elevated liver enzymes, is an infectious disease that is transmitted through body fluids, blood and mucosal contact. In some individuals infected with HBV, the disease can survive silently in the body. The virus can also manifest itself in some individuals. Hepatitis-B disease is divided into acute and chronic. Acute HBV is a short-term illness that occurs within the first 6 months after exposure to the Hepatitis-B virus. Even if the virus does not cause any symptoms in these people, the risk of being a carrier and transmitting the infection continues. Among the symptoms observed in individuals, complaints such as fever, fatigue, loss of appetite, nausea or vomiting, muscle, joint and stomach pain are observed. Chronic HBV infection is the form of the Hepatitis-B virus that cannot be eliminated and can cause bad consequences such as liver damage (cirrhosis), liver cancer and death [3]. While there is no known treatment for acute HBV, the disease can be controlled with the help of various medications in chronic HBV. HBV can be transmitted horizontally or vertically between individuals. Transmission between individuals through blood or water, sexual contact, or reuse of unsafe syringes is called horizontal transmission, while transmission of the virus from an HBV-infected mother to her newborn baby is called vertical transmission.

Mathematical modeling in infectious diseases is an important method to examine the course of diseases. Mathematical models provide information about the estimated number of cases of infectious diseases and the estimated number of deaths from the disease [4]. Moreover, mathematical modeling also helps determine parameters and solution methods to alleviate the disease [5]. Therefore, mathematical modeling of the disease has become a highly preferred subject to determine the course of infectious diseases.

Many studies have been conducted in the literature on the mathematical modeling of HBV, one of the infectious diseases. Bashir and Umar [6] established a new mathematical model by combining three control strategies known as treatment, vaccination and media campaign in order to reduce the spread of HBV. Combining the three interventions was shown to improve the outcome of the study as much as possible. Kiemtore et al. [7] conducted a study in Africa, one of the places in the world where HBV is most likely to occur. They developed a model that included vaccination and treatment of HBV in the Burkina Faso population. They estimated the disease parameters using Gray Wolf Optimization (GWO). Li and Chai [8], developed a mathematical model that models the drug resistance treatment of HBV. Their aim was to investigate the reason for the decrease in drug resistance of the disease as a result of the mutation of HBV over time. As a result, it has been revealed that if the virus resistance of the disease is high in the body, combined drug treatment shortens the clearance time of the resistant HBV virus.

Liu et al. [9] designed a fractional model for Hepatitis-B. They created a non-linear epidemic model by investigating the stages of transmission of HBV. They formulated the model with the vaccine effect using the Atangana-Baleanu derivative (AB derivative). As a result, it has been demonstrated that

vaccination is a method that can end the HBV epidemic process. Elaiw et al. [10] pointed out that there may be model inaccuracies that may arise when modeling HBV disease, and they modeled the disease with a non-linear ordinary differential equation. Possible disruptions and uncertainties were reflected in the model as social limited disruptions. They expanded their model by adding two types of drug therapies used to prevent new infections. Khan et al. [11] established an HBV model and conducted a sensitivity analysis of the model by examining its existence and positivity. They performed numerical simulations to analyze parameter sensitivities. de Villiers et al. [12] compared two deterministic HBV models, the Imperial HBV model and CDA. Additionally, the effect of the birth dose of HBV vaccine was investigated. They contributed to the literature by revealing some differences between the two models.

Preventing the HBV epidemic in Ghana and the Brong Ahafo region is a major challenge due to limited resources in these regions. Otoo et al. [13] drew attention to this problem and formulated a model that explains the spread of HBV. They proposed an intervention that would minimize the impact of the epidemic. As a result, they have shown that it is possible to combat the spread of the disease by vaccinating susceptible people and treating infected people. Cardoso et al. [14] explained the dynamics of HBV with a fractional model. They presented the main results of this model. They calculated the equilibrium point and basic reproduction number. Finally, by performing numerical simulations, they demonstrated that the model converged to an equilibrium point. Farman et al. [15] created a time-spanning and non-linear model of HBV. They used fractional parameters to develop the system. Finally, they applied numerical simulation. In this way, they investigated the effect of the system parameter on the spread of the disease. Reinharz et al. [16] developed a multicompartmental model that included infected human hepatocytes and total intracellular HBV DNA per infected human hepatocyte. They also modeled HBV kinetics during 14-day treatment of Humanized Chimeric Mice. As a result of the study, they obtained new information about HBV DNA dynamics in infected human hepatocytes.

There are many models in the form of in vivo model systems to demonstrate the progression of HBV in different animal species. Ortega-Priet et al. [17] drew attention to this situation and conducted a study examining in vivo model systems to examine the HBV life cycle. They examined models such as the chimpanzee model, tree mouse model and carrier animal models of HBV. Khan et al. [18] considered various stages of HBV using generalized saturated incidence. They created a model to demonstrate HBV dynamics and control strategies. They investigated the time dynamics and stability conditions of the model. Finally, they updated their models to increase the number of the recovered population and minimize the infected population. Friedman and Siewe [19], discussed the treatment of chronic HBV with the combination of IFN-alpha and adefovir. In their study, they investigated what the optimal ratio between IFN-alpha and adefovir should be for the best results. They designed a model of HBV pathogenesis using a partial differential equation system.

Oludoun et al. [20] formulated a model to examine the impact of testing and treatment on HBV and to analyze the transmission process. They used the Next Generation Matrix method for the basic reproduction number. As a result, they revealed that testing in cases of acute HBV and chronic unconsciousness will contribute to controlling the disease. Moreover, a number of illustrative applications have been performed in terms of the investigations on HBV, COVID-19 [21–25], HIV-AIDS [26–28], SARS-CoV [29–32], tuberculosis [33–36], Malaria [37–40], other infectious diseases [41–46] and prey-predator competition research that successfully explain mathematical modelling [47–50].

In this study, mathematical modeling was used to examine the effect of vertical transmission and migration parameters of HBV on the course of HBV infectious disease. In the mathematical model consisting of 6 compartments, susceptible population, latent population, acute population, carrier

population, protected infected (in the sense of recovered) and vaccinated population were used.

The rest of the study is organized as follows: Numerical results and discussion are presented in Section 4, and the conclusion is explained in Section 5.

DERIVATION OF THE MATHEMATICAL MODEL FOR HBV INFECTION

In this section, the basic SLACPV mathematical model for the effect of HBV on vertical transmission and migration factor is proposed and analyzed. Total population, susceptible population $S(t)$, latent population $L(t)$, acute population $A(t)$, carrier population $C(t)$, protective rescued $P(t)$ and vaccinated population $V(t)$ is divided into six mutually exclusive parts.

The parameters provided constitute elements of a mathematical model describing the transmission dynamics of Hepatitis-B virus (HBV). Λ denotes the recruitment rate of individuals into the susceptible population, while ς represents the rate of immunity decrease due to vaccination. μ stands for the natural mortality rate, and β signifies the transmission rate. ϕ indicates reduced transmission compared to acute infection, and ω represents the rate of babies born infected from mothers. ϑ signifies the proportion of infected mothers transmitting HBV to their offspring. ϖ is the vaccination rate, ξ is the recovery rate from acute infection, and f is the recovery rate of carriers. p denotes the transition rate from acute to carrier status, and μ_o is the HBV-related death rate. g represents the rate of new individuals joining the community through migration. b signifies the transition rate from susceptible to latent infection, l is the vaccine leakage rate, and k is the transition rate from latent to acute infection. $\wp$ is the rate of babies born infected. These parameters collectively depict the interplay of various processes in HBV transmission dynamics, aiding in understanding and predicting disease spread and control measures. The same situation is represented by a schematic diagram and stated in Section 2. The equation system of the given schematic diagram is given in Eq. (1)

Hence, by considering this assumption the rate of change of susceptible, latent, acute, carrier, protected infected (protective rescued) and vaccinated population is described in the following nonlinear system of differential equations.

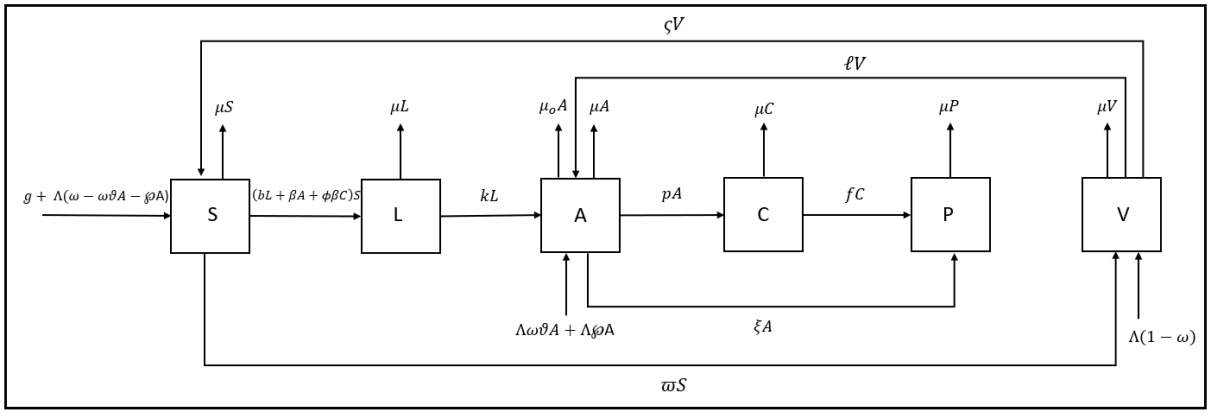

Figure 1
Schematic diagram of the SLACPV model.

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda\omega(1-\vartheta A) - (bL)S + \varsigma V - (\mu + \beta A + \phi\beta C + \varpi)S + g - \Lambda\wp A,\\
\frac{dL}{dt} &= (\beta A + \phi\beta C)S - \mu L + (bL)S - kL,\\
\frac{dA}{dt} &= \Lambda\omega\vartheta A - \mu A - \mu_o A - pA - \xi A + kL + \ell V + \Lambda\wp A,
\end{aligned} \tag{1}$$

$$\begin{aligned}
\frac{dC}{dt} &= pA - fC - \mu C,\\
\frac{dP}{dt} &= \xi A - \mu P + fC,\\
\frac{dV}{dt} &= \Lambda(1-\omega) + \varpi S - \mu V - \varsigma V - \ell V,
\end{aligned} \tag{1}$$

with initial conditions $S(0) = S_0 \geq 0$, $L(0) = L_0 \geq 0$, $A(0) = A_0 \geq 0$, $C(0) = C_0 \geq 0$, $P(0) = P_0 \geq 0, V(0) = V_0 \geq 0$. The used model parameters and their biological description are listed in Table 1.

QUALITATIVE ANALYSIS OF THE MODEL

Equilibria of the Model

In this subsection, we evaluate the disease-free equilibrium (DFE) and its stability to study the steady-state behaviour of the model constructed for the HBV disease model. In order to achieve this, we reconsider the following system of equations:

$$\begin{aligned}
&\Lambda\omega(1-\vartheta A) - (bL)S + \varsigma V - (\mu + \beta A + \phi\beta C + \varpi)S + g - \Lambda\wp A = 0,\\
&(\beta A + \phi\beta C)S - \mu L + (bL)S - kL = 0,\\
&\Lambda\omega\vartheta A - \mu A - \mu_o A - pA - \xi A + kL + \ell V + \Lambda\wp A = 0,\\
&pA - fC - \mu C = 0,\\
&\xi A - \mu P + fC = 0,\\
&\Lambda(1-\omega) + \varpi S - \mu V - \varsigma V - \ell V = 0,
\end{aligned} \tag{2}$$

By solving the last equation, we get the following DFE point:

$$\Psi^0 = (S_0, L_0, A_0, C_0, P_0, V_0) = (S_0, 0, 0, 0, 0, 0), \tag{3}$$

where

$$S_0 = \frac{\Lambda\omega + g}{\mu + \varpi}$$

or

$$S_0 = \frac{\Lambda(\omega - 1)}{\varpi}.$$

NUMERICAL RESULT AND DISCUSSION

In this study, we want to examine the effect of HBV on the course of the disease by establishing a new model that takes into account the vertical transmission and migration parameters. For this, we are building a mathematical model with 6 compartments: susceptible population $S(t)$, latent population $L(t)$, acute population $A(t)$, carrier population $C(t)$, protected infected (protective rescued) $P(t)$ and vaccinated population $V(t)$. We use the $4th$ order Runge-Kutta method to obtain numerical solutions of the model within these compartments. In general, m.-order Runge-Kutta method has the following advantages:

• To calculate the approximate value of the Y_{i+1} solution, it is found by calculating only the Y_i value. That's why the one-step method is the most important method of its kind.

• When $m = 4$, it turns into the method called classical Runge Kutta. This method is the most used method [51].

After performing the numerical solutions of the relevant model, we obtain some results. We use the literature [52, 53] for some of our parameters used in the model and estimate many of them. Our parameter estimates are shown in Table 1:

Table 1
Parameters used for the HBV model.

Par.	Biological description	Value	Sources
Λ	Recruitment rate	0.22996	fitted
ς	Rate of decrease in immunity due to the effect of the vaccine	0.1546	fitted
μ	Natural mortality rate	3.4857e-05	[52]
β	Transmission rate	0.001	fitted
ϕ	Reduced transmission rate relative to acute	0.21	fitted
ω	The rate of births without successful insemination (babies born sick after insemination from mothers)	0.003	fitted
ϑ	Infected rate of mothers with HBV acute virus	0.02456	fitted
ϖ	Vaccination rate	0.21	fitted
ξ	Recovery rate of individuals with acute infection	0.17	fitted
f	Recovery rate of individuals in the carrier class	0.2124	fitted
p	Transition rate from Acute to Carrier	0.1349	fitted
μ_o	Death rate from HBV disease	0.1025	fitted
g	Rate of community coming from outside	0.3899	fitted
b	Transition rate from susceptible to latent	0.002	fitted
ℓ	Vaccine leakage rate	0.1356	fitted
k	Transition rate from latent to acute	0.1174	fitted
$\wp$	Rate of babies born infected	0.2438	fitted
$S(0)$	Initial Susceptible population	6000	fitted
$L(0)$	Initial Latent population	500	fitted
$A(0)$	Initial Acute population	1497	[53]
$C(0)$	Initial Carrier population	100	fitted
$P(0)$	Initial protected infected population	150	fitted
$V(0)$	Initial Vaccinated population	300	fitted

In this investigation we have performed all numerical results and finding by benefiting from Matlab R2023b Software. Below, we examine the development and change of some populations in our model over time. In Figure 2, we examine the change and development of acute individuals, vaccinated individuals and recovered individuals over time.

In Figure 3, we examine the effect of individuals in the latent phase on acute individuals and recovering individuals. In addition, on these populations, the mortality rate from HBV disease in Figure 4, the recovery rate of individuals with acute infection in Figure 5, the transmission rate in Figure 6 and in Figure 7 we also examine the change in vaccination rate over time.

It has been observed that when the number of vaccinated individuals is kept at a minimum level, the number of infected individuals decreases, and the number of protected infected individuals reaches a maximum.

The Protected Infected population increased as a result of the decrease in Latent and Acute individuals towards the end of the process. This positively affected the course of the disease.

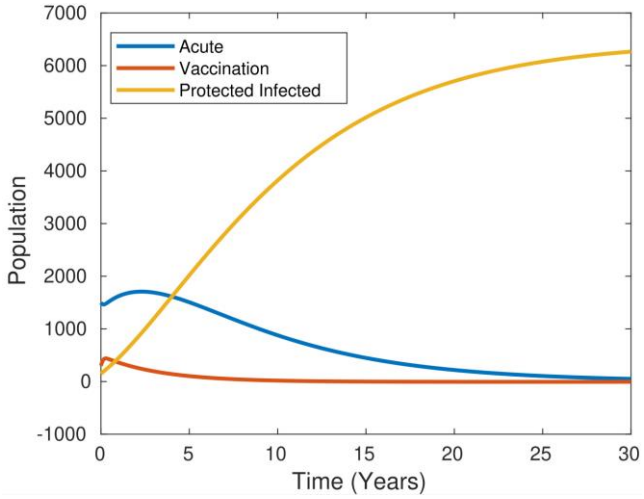

Figure 2
Status of Acute, Vaccination, Protected Infected populations relative to each other.

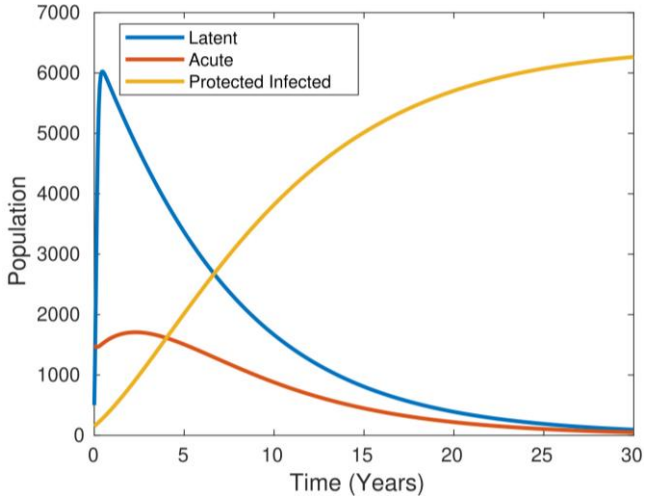

Figure 3
Latent Status of Latent, Acute, Protected Infected populations relative to each other.

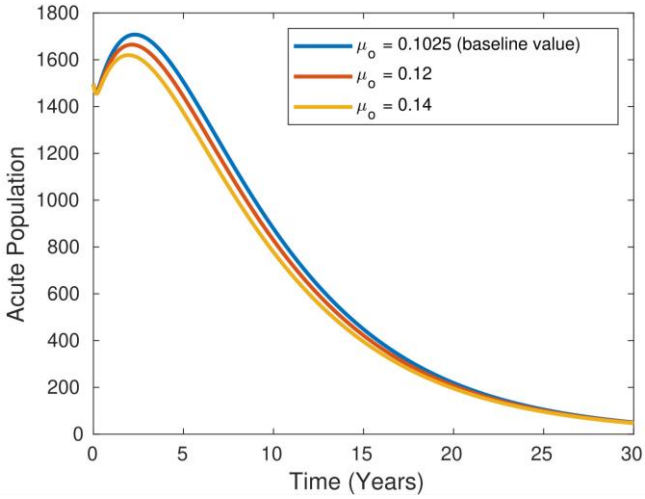

Figure 4
The mortality rate from HBV disease.

The graph in Figure 4 depicts the mortality rates attributable to HBV (Hepatitis B Virus) over a specific period of time (30 years). Trends in the graph indicate the change in death rates from HBV

disease over time. The reasons for sudden decreases or fluctuations in a certain period in the graph are; epidemics, changes in health policies or difficulties in access. A clear downward trend is observed in the death rates caused by HBV disease over time. This decline is generally due to factors such as the expansion of vaccination programs, improvements in treatment options, and effective implementation of health education.

The graph in Figure 5 shows the recovery rates of individuals with acute infectious disease over 30 years. Examining the changes in the trends in the graph over time is important for the management and treatment of the disease. It is necessary to determine the underlying reasons for a significant increase or decrease in recovery rates in a certain period. These reasons include factors such as the nature of the disease, treatment methods used, access to healthcare and patients' lifestyle. Additionally, information can be obtained about the change in recovery rates by demographic characteristics by examining the change in recovery rates. Evaluating the impact of factors such as age, gender, geographical location or underlying health condition on recovery rates is important to understand how the disease is affected among different groups. There is a clear trend in the graph, so appropriate strategies are being developed to understand the reasons for this trend and increase recovery rates. This is a critical step for improving health policies and health services and improving patients' quality of life.

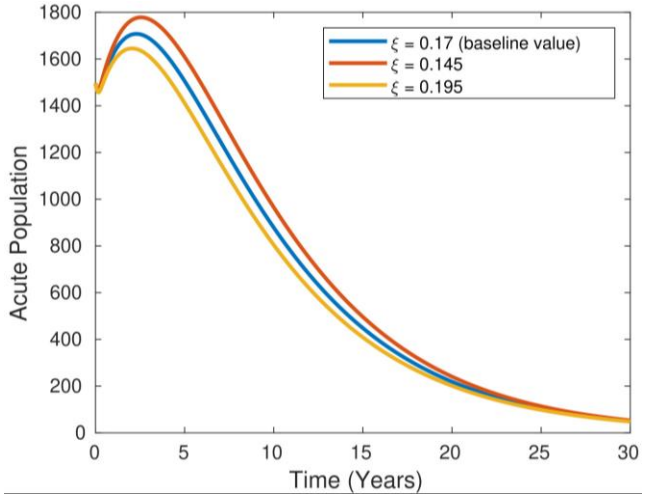

Figure 5
The recovery rate of individuals with acute infection disease.

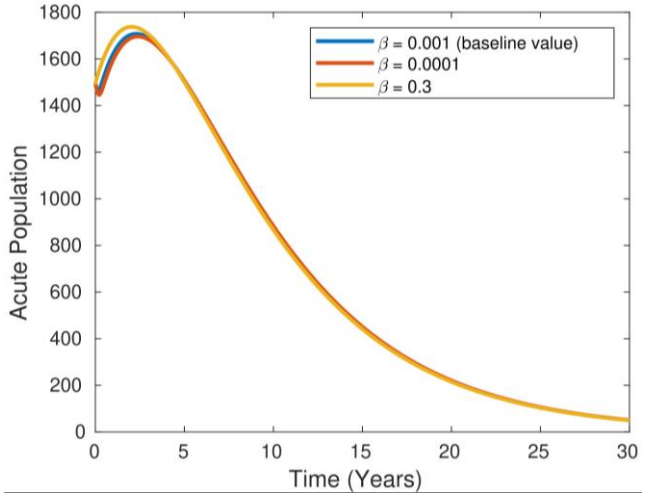

Figure 6
The transmission rate of the HBV virus.

The graph in Figure 6 depicts the change in the transmission rate of the HBV virus over a certain

period of time. An important point is that the continuous, graphical changes increase over time. For example, it is necessary to determine the underlying causes of sudden increases or decreases in transactions over a certain period. These include factors such as epidemics, changes in health policies, changes in the development of society, or a new treatment or vaccine. In addition, a clear formation is seen in the graph, and it is necessary to examine how the events progress. It should also be determined how long the increases or decreases continue over 30 years and what factors they depend on. The results of such analyzes should evaluate health policies and interventions. Finally, appropriate strategies are developed to understand the reasons for this trend and prevent similar situations from occurring in the future. These strategies control the spread of the disease.

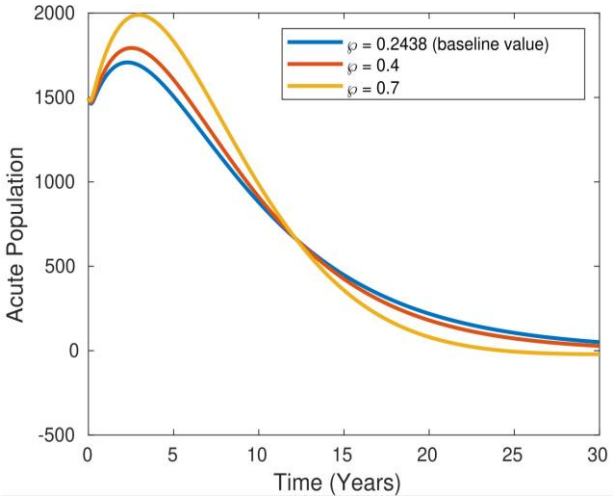

Figure 7
Rate of babies born infected.

The graph in Figure 7 shows the change in the rate of infected babies over time over 30 years. It is important to know the reasons behind the increase or decrease in the rates of babies born in certain periods of these data. The reason for the increase at the beginning of this process is due to the high vertical transmission rate of HBV. In the middle of the process, a clear intersection can be seen on the graph. The reason for this intersection is that the vertical transmission of HBV is under control. After this point, a significant decrease is observed in the graph. Appropriate strategies are developed to understand the reasons for this trend and prevent similar situations from occurring in the future. As a result of these strategies, vertical transmission of the disease is controlled. As a result of these analyses, the number of healthy babies is expected to increase.

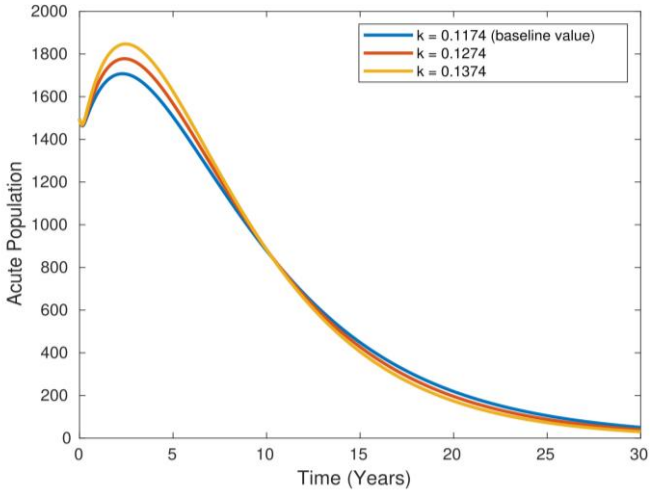

Figure 8
Acute population dynamics according to various values of k.

CONCLUSION

In this paper, we have developed a new Hepatitis-B mathematical model containing a vertical transmission from mothers to newborn babies. Also, we have evaluated the disease-free equilibrium point. In the numerical simulation section of the article presents the results obtained throughout our study process. The effectiveness of this model and its performance by developing a new expanding structure that models Hepatitis-B by selecting the appropriate effect for the model. In the modelling of the results we obtained, it is seen that the course of Hepatitis-B disease is modelled by predicting it and predictions about its process are obtained. Non-negative solutions have been obtained to ensure biological augmentation of our system of equations of the model.

Ethical Statement

This study is an original research article designed and developed by the authors.

Author Contributions

Research Design (CRediT 1) M.Y. (%60) – N.B.B. (%15) – K.A. (%15) – F.N.Ö. (%15)
Data Collection (CRediT 2) M.Y. (%10) – N.B.B. (%30) – K.A. (%30) – F.N.Ö. (%30)
Research - Data Analysis - Validation (CRediT 3-4-6-11) M.Y. (%25) – N.B.B. (%25) – K.A. (%25) – F.N.Ö. (%25)
Writing the Article (CRediT 12-13) M.Y. (%25) – N.B.B. (%25) – K.A. (%25) – F.N.Ö. (%25)
Revision and Improvement of the Text (CRediT 14) M.Y. (%25) – N.B.B. (%25) – K.A. (%25) – F.N.Ö. (%25)

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Conflict of Interest

The authors declare no conflict of interest.

Data Availability Statement

No Data associated in the manuscript.

Sustainable Development Goals (SDG)

Sustainable Development Goals: 3 Good health and well-being

REFERENCES

[1] S.M. Ciupe, R.M. Ribeiro, P.W. Nelson, G. Dusheiko, A.S. Perelson, The role of cells refractory to productive infection in acute hepatitis B viral dynamics, *Proceedings of the National Academy of Sciences*. 104(12) (2007), 5050-5055. doi: 10.1073/pnas.0603626104

[2] F.F. Chenar, Y.N. Kyrychko, K.B. Blyuss, Mathematical model of immune response to hepatitis B, *Journal of Theoretical Biology*. 447 (2018), 98-110. doi: 10.1016/j.jtbi.2018.03.025

[3] Acıbadem Yayın Kurulu, (2021). https://www.acibadem.com.tr/ilgi-alani/hepatit-b/ (erişim 10 Mayıs 2024).

[4] A. Çilli, K. Ergen, Salgın hastalıkların tahmininde kullanılan SI ve SIS modellerin uygulamaları, *Bitlis Eren Üniversitesi Fen Bilimleri Dergisi*. 8(3) (2019), 755-761. doi: 10.17798/bitlisfen.522533

[5] A. Costa, M. Pires, R. Resque, S.S.M.S. Almeida, Mathematical modeling of the infectious diseases: key concepts and applications, *Journal of Infectious Diseases and Epidemiology*. 7(5) (2021), 209. doi: 10.23937/2474-3658/1510209

[6] U.S. Bashir, A. Umar, Mathematical analysis of hepatitis B virus model with interventions in Taraba state, Nigeria. *International Journal of Development Mathematics (IJDM)*. 1 (1) (2024). doi: 10.62054/ijdm/0101.14

[7] A. Kiemtore, W.O. Sawadogo, I. Zangré, P.O.F. Ouedraogo, I. Mouaouia, Estimation of parameters for the mathematical model of the spread of hepatitis B in Burkina Faso using grey wolf optimizer, *International Journal of Analysis and Applications*. 22 (2024), 48-48. doi: 10.28924/2291-8639-22-2024-48

[8] D.M. Li, B. Chai, A dynamic model of hepatitis B virus with drug-resistant treatment, *AIMS Mathematics.* 5(5) (2020), 4734-4753. doi: 10.3934/math.2020303

[9] P. Liu, A. Din, R. Zarin, Numerical dynamics and fractional modeling of Hepatitis B virus model with non-singular and non-local kernels, *Results in Physics*. 39 (2022), 105757. doi: 10.1016/j.rinp.2022.105757

[10] A.M. Elaiw, M.A. Alghamdi, S. Aly, Hepatitis B virus dynamics: modeling, analysis, and optimal treatment scheduling, *Discrete Dynamics in Nature and Society*. 1 (2013). doi: 10.1155/2013/712829

[11] T. Khan, S. Ahmad, G. Zaman, Modeling and qualitative analysis of a hepatitis B epidemic model, *Chaos: An Interdisciplinary Journal of Nonlinear Science*. 29(10) (2019). doi: 10.1063/1.5111699

[12] M.J. de Villiers, I. Gamkrelidze, T.B. Hallett, S. Nayagam, H. Razavi, D. Razavi-Shearer, Modelling hepatitis B virus infection and impact of timely birth dose vaccine: a comparison of two simulation models, *PLoS One*. 15 (8) (2020). e0237525 doi: 10.1371/journal.pone.0237525

[13] D. Otoo, I.O. Abeasi, S. Osman, E.K. Donkoh, Mathematical modeling and analysis of the dynamics of hepatitis b with optimal control, *Communications in Mathematical Biology and Neuroscience.* (2021). doi: 10.28919/cmbn/5733

[14] L.C. Cardoso, R.F. Camargo, F.L.P. dos Santos, J.P.C. Dos Santos, (2021). Global stability analysis of a fractional differential system in hepatitis B. Chaos, *Solitons & Fractals*. 143 (2021). 110619. doi: 10.1016/j.chaos.2020.110619

[15] M. Farman, A. Ahmad, H. Muslim, M.O. Ahmad, (2019). Dynamical behavior of hepatitis B fractional-order model with modeling and simulation, *Journal of Biochemical Technology*. 10(3) (2019), 11-17.

[16] V. Reinharz, Y. Ishida, M. Tsuge, K. Durso-Cain, T.L. Chung, C. Tateno, et al. Understanding hepatitis B virus dynamics and the antiviral effect of interferon alpha treatment in humanized chimeric mice, *Journal of Virology*. 95(14) (2021), 10-1128. doi: 10.1128/jvi.00492-20

[17] A.M. Ortega-Prieto, C. Cherry, H. Gunn, M. Dorner, In vivo model systems for hepatitis B virus research, *ACS Infectious Diseases*. 5(5) (2018), 688-702. doi: 10.1021/acsinfecdis.8b00223

[18] T. Khan, F.A. Rihan, M. Ibrahim, S. Li, A.M. Alamri, S.A. AlQahtani, Modeling different infectious phases of hepatitis B with generalized saturated incidence: An analysis and control, *Mathematical Biosciences and Engineering*. 21(4) (2024), 5207-5226. doi: 10.3934/mbe.2024230

[19] A. Friedman, N. Siewe, Chronic hepatitis B virus and liver fibrosis: A mathematical model, *PLoS One*. 13(4) (2018), e0195037. doi: 10.1371/journal.pone.0195037

[20] O. Oludoun, O. Adebimpe, J. Ndako, M. Adeniyi, O. Abiodun, B. Gbadamosi, The impact of testing and treatment on the dynamics of Hepatitis B virus, *F1000Research*. 10 (2021). doi: 10.12688/f1000research.72865.1

[21] R. Ikram, A. Khan, M. Zahri, A. Saeed, M. Yavuz, P. Kumam, Extinction and stationary distribution of a stochastic COVID-19 epidemic model with time-delay, *Computers in Biology and Medicine.* 141 (2022), 105115. doi: 10.1016/j.compbiomed.2021.105115

[22] I.U. Haq, N. Ali, and K. S. Nisar, An optimal control strategy and Grünwald-Letnikov finite-difference numerical scheme for the fractional-order COVID-19 model, *Mathematical Modelling and Numerical Simulation with Applications*. *2*(2) (2022), 108-116. doi: 10.53391/mmnsa.2022.009

[23] H. Joshi, B.K. Jha, and M. Yavuz, Modelling and analysis of fractional-order vacci-nation model for control of COVID-19 outbreak using real data, *Mathematical Biosciences and Engineering*. 20(1) (2022), 213-240. doi: 10.3934/mbe.2023010

[24] H. Joshi, M. Yavuz, S. Townley, and B. K. Jha, Stability analysis of a non-singular fractional-order covid-19 model with nonlinear incidence and treatment rate, *Physica Scripta*. 98(4) (2023), 045216. doi: 10.1088/1402-4896/acbe7a

[25] M. Yavuz, F.Ö. Coşar, F. Günay, F.N. Özdemir, A new mathematical modeling of the COVID-19 pandemic including the vaccination campaign, *Open Journal of Modelling and Simulation*. 9(3) (2021), 299-321. doi: 10.4236/ojmsi.2021.93020

[26] A.S. Waziri, E.S. Massawe, O.D. Makinde, Mathematical modelling of HIV/AIDS dynamics with treatment and vertical transmission, *Journal Applied Mathematics*. 2(3) (2012), 77-89. doi: 10.5923/j.am.20120203.06

[27] D. Wodarz, M.A. Nowak, Mathematical models of HIV pathogenesis and treatment, *BioEssays*. 24(12) (2002), 1178-1187. doi: 10.1002/bies.10196

[28] T.K. Ayele, E.F.D. Goufo, S. Mugisha, Mathematical modeling of HIV/AIDS with optimal control: a case study in Ethiopia, *Results in Physics*. 26 (2021), 104263. doi: 10.1016/j.rinp.2021.104263

[29] S. Wang, Y. Pan, Q. Wang, H. Miao, A.N. Brown, L. Rong, Modeling the viral dynamics of SARS-CoV-2 infection, *Mathematical Biosciences*. 328 (2020), 108438. doi: 10.1016/j.mbs.2020.108438

[30] S.M. Ciupe, N. Tuncer, Identifiability of parameters in mathematical models of SARS-CoV-2 infections in humans, *Scientific Reports*. 12(1) (2022), 14637. doi: 10.1038/s41598-022-18683-x

[31] G. Gonzalez-Parra, D. Martínez-Rodríguez, R.J. Villanueva-Micó, Impact of a new SARS-CoV-2 variant on the population: A mathematical modeling approach, *Mathematical and Computational Applications*, 26(2) (2021). 25. doi: 10.3390/mca26020025

[32] A. Atifa, M.A. Khan, K. Iskakova, F.S. Al-Duais, I. Ahmad, Mathematical modeling and analysis of the SARS-Cov-2 disease with reinfection, *Computational Biology and Chemistry*. 98 (2022), 107678. doi: 10.1016/j.compbiolchem.2022.107678

[33] M. Yavuz, F. Özköse, M. Akman, Z.T. Tastan, A new mathematical model for tuberculosis

epidemic under the consciousness effect, *Mathematical Modelling and Control*. 3(2) (2023), 88-103. doi: 10.3934/mmc.2023009

[34] E.D. Ginting, D. Aldila, I.H. Febriana, A deterministic compartment model for analyzing tuberculosis dynamics considering vaccination and reinfection, *Healthcare Analytics*. 5 (2024), 100341. doi: 10.1016/j.health.2024.100341

[35] J. Wang, G. Lyu, Analysis of an age-space structured tuberculosis model with treatment and relapse, *Studies in Applied Mathematics*. 153(1) (2024), e12700. doi: 10.1111/sapm.12700

[36] E.M. Delgado Moya, J.A. Ordoñez, F. Alves Rubio, M. Niskier Sanchez, R.B. de Oliveira, R. Volmir Anderle, D. Rasella, A Mathematical Model for the Impact of 3HP and Social Programme Implementation on the Incidence and Mortality of Tuberculosis: Study in Brazil, *Bulletin of Mathematical Biology*. 86(6) (2024), 1-25. doi: 10.1007/s11538-024-01285-1

[37] A.A. Gebremeskel, H.E. Krogstad, Mathematical modelling of endemic malaria transmission, *American Journal of Applied Mathematics*. 3(2) (2015), 36-46. doi: 10.11648/j.ajam.20150302.12

[38] S.I. Oke, M.M. Ojo, M.O. Adeniyi, M.B. Matadi, Mathematical modeling of malaria disease with control strategy, *Communications in Mathematical Biology and Neuroscience*. (2020), Article-ID: 43. doi: 10.28919/cmbn/4513

[39] M. Osman, I. Adu, Simple mathematical model for malaria transmission, *Journal of Advances in Mathematics and Computer Science.* 25(6) (2017), 1-24. doi: 10.9734/JAMCS/2017/37843

[40] S. Olaniyi, O.S. Obabiyi, Mathematical model for malaria transmission dynamics in human and mosquito populations with nonlinear forces of infection, *International Journal of Pure and Applied Mathematics*. 88(1) (2013), 125-156. doi: http://dx.doi.org/10.12732/ijpam.v88i1.10

[41] P. A. Naik, Z. Eskandari, M. Yavuz, & Z. Huang, Bifurcation results and chaos in a two-dimensional predator-prey model incorporating Holling-type response function on the predator, *Discrete and Continuous Dynamical Systems-S.* (2024). doi: 10.3934/dcdss.2024045

[42] D. Ghosh, P.K. Santra, G.S. Mahapatra, A three-component prey-predator system with interval number, *Mathematical Modelling and Numerical Simulation with Applications.* 3(1) (2023), 1-16. doi: 10.53391/mmnsa.1273908

[43] P.A. Naik, Z. Eskandari, H.E. Shahkari, K.M. Owolabi, Bifurcation analysis of a discrete-time prey-predator model, *Bulletin of Biomathematics.* 1(2) (2023), 111-123. doi:10.59292/bulletinbiomath.2023006

[44] J. Danane, M. Yavuz, M. Yıldız, Stochastic modeling of three-species Prey–Predator model driven by L´evy Jump with Mixed Holling-II and Bedding-ton–DeAngelis functional responses, *Fractal and Fractional*. 7(10) (2023), 751.doi: 10.3390/fractalfract7100751

[45] O.M. Tessa, Mathematical model for control of measles by vaccination. *In Proceedings of Mali Symposium on Applied Sciences*, 2006: pp. 31-36.

[46] K.A.M. Gaythorpe, C.L. Trotter, B. Lopman, M. Steele, A.J.K. Conlan, Norovirus transmission dynamics: a modelling review, *Epidemiology & Infection*. 146(2) (2018), 147-158.doi:10.1017/S0950268817002692

[47] I.U. Haq., M. Yavuz, N. Ali, A. Akgül, A SARS-CoV-2 fractional-order mathematical model via the modified Euler method, *Mathematical and Computational Applications.* 27(5) (2022), 82. doi: 10.3390/mca27050082

[48] N. Kar, N. Özalp, A fractional mathematical model approach on glioblas-toma growth: tumor visibility timing and patient survival, *Mathematical Modelling and Numerical Simulation with Applications*. 4(1) (2024), 66-85. doi:10.53391/mmnsa.1438916

[49] B. Bolaji, T. Onoja, C. Agbata, B. I. Omede, U. B. Odionyenma, Dynamical analysis of HIV-TB co-infection transmission model in the presence of treatment for TB, *Bulletin of Biomathematics.* 2(1) (2024), 21-56. doi:10.59292/bulletinbiomath.2024002

[50] F. Evirgen, E. Uçar, S. Uçar, N. Özdemir, Modelling influenza A disease dynamics under Caputo-Fabrizio fractional derivative with distinct contact rates, *Mathematical Modelling and Numerical Simulation with Applications*. 3(1) (2023), 58-73. doi:10.53391/mmnsa.1274004

[51] N.E. Binbay, H.B. Gümgüm, Block denklemlerinin nümerik yöntemlerle çözümü, *Fen Bilimleri ve Matematik Alanında Araştırma Makaleleri*. (2019), 155-183.

[52] https://data.tuik.gov.tr/Bulten/Index?p=Olum-ve-Olum-Nedeni-Istatistikleri-2021-45715#:~:text=Bin%20ki%C5%9Fi%20ba%C5%9F%C4%B1na%20d%C3%BC%C5%9Fen%20%C3%B6l%C3%BCm,ba%C5%9F%C4%B1na%206%2C7%20%C3%B6l%C3%BCm%20d%C3%BC%C5%9Ft%C3%BC. Access Date: 11 April 2024

[53] https://www.cdc.gov/hepatitis/statistics/2015surveillance/index.htm#tabs-5-3 Access Date: 11 April 2024

[54] Ö.A. Gümüs, Q. Cui, G.M. Selvam, A. Vianny, Global stability and bifurcation analysis of a discrete time SIR epidemic model, *Miskolc Mathematical Notes*. 23(1) (2022), 193-210. doi: 10.18514/mmn.2022.3417

[55] S.H. Streipert, G.S. Wolkowicz, An augmented phase plane approach for discrete planar maps: Introducing next-iterate operators. *Mathematical Biosciences*, 355 (2023), 108924. doi: 10.1016/j.mbs.2022.108924

[56] L. Boulaasair, H. Bouzahir, M. Yavuz, Global mathematical analysis of a patchy epidemic model, *An International Journal of Optimization and Control: Theories & Applications (IJOCTA)*. 14(4) (2024), 365-377. doi: 10.11121/ijocta.1558

[57] S. Bhatter, S. Kumawat, B. Bhatia, S.D. Purohit, Analysis of COVID-19 epidemic with intervention impacts by a fractional operator, *An International Journal of Optimization and Control: Theories & Applications (IJOCTA)*. 14 (3) (2024), 261-275. doi: 10.11121/ijocta.1515