

Interferon system response to virus infection

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Motivation and Aim: The interferon system is a key factor in the reactions of innate immunity, which implements the mechanisms for protecting the body from virus infection. Interferon is activated in response to the entry of viruses into the body and prevents them from replicating in the host cell and further spreading to the neighboring ones. The aim of the work is to study a mathematical model that describes the interaction of two processes: the process of HIV-1 replication in the cell [1, 2] and the response of the interferon system to the penetration of the virus into the cell.

Methods and Results: The mathematical model describing the stages of the interferon systems response to the HIV-1 infection in terms of ordinary differential equations has been considered. The system consists of 39 equations representing the following stages of the HIV-1 life cycle: entry, reverse transcription, integration, transcription, translation, assembly, budding and maturation new virions and the process of the interferon response to the HIV-1 penetration into the CD4⁺ T-cell. Numerical experiments have been done to verify and calibrate the model as well as to analyze the sensitivity of the model.

Conclusion: As a result of the numerical analysis of the model sensitivity, the stages of the interferon response and the model parameters that have greatest impact on the efficiency of producing the viral particles having the infected cell have been determined.

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References

1. Shcherbatova O., Grebennikov D., Sazonov I., Meyerhans A., Bocharov G. Modeling of the HIV-1 life cycle in productively infected cells to predict novel therapeutic targets. *Pathogens*. 2020;9(4):255. DOI 10.3390/pathogens9040255.
2. Likhoshvai V.A., Khlebodarova T.M., Bazhan S.I., Gainova I.A., Chereshev V.A., Bocharov G.A. Mathematical model of the Tat-rev regulation of HIV-1 replication in an activated cell predicts the existence of oscillatory dynamics in the synthesis of viral components. *BMC Genomics*. 2014;15(Suppl 12):S1.