

Modelling of tumour-immune system interactions in the presence of HIV virus

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Human immunodeficiency virus (HIV) is a retrovirus often leading to a disease called acquired immunodeficiency syndrome (AIDS) which is estimated to be responsible for killing more than 25 million people since its first recognition in 1981 up to 2005.

One of the very serious problems that encounter HIV-infected patients is the high rate of cancer occurrence (for instance Kaposi's sarcoma or non-Hodgkin's lymphoma). As no viral sequence has been found in the DNA of cancer cells common hypothesis proclaim the immune surveillance deficiency. It is therefore of great importance to better understand links between HIV — related immunosuppression and cancer prognosis.

We propose the model of AIDS-related cancers on the basis of simple tumour-immune system interactions model. This model stands for our "control" case. It is described by a system of delayed differential equations reflecting interactions between cancer cells and effectors of the immune system (NK lymphocytes). In the model we take into account inactivation of NK cells after killing cancer cell and subsequent activation of most of them after some time (which we assume to be constant).

In the presence of HIV virus we divide the population of immunocompetent cells into two subpopulations: healthy cells and cells infected by virus.

Both models are described in terms of Lotka-Volterra systems. The behaviour of them depends on the model parameters, namely on the magnitude of the cancer reproduction rate. Of course, in the presence of HIV virus the prognosis for patient surveillance is always worst than in the absence of this virus.