

Genetic markers that predict sensitivity of tumor cells to infection by Sendai virus



Pirogov Russian National Research Medical University (RNRMU)

Anastasia O. Sosnovtseva^{1,2}, Dmitry V. Kochetkov², Anastasia V. Lipatova
Darya A. Berzhitskaya, Andrey O. Zheltukhin²

¹Pirogov Russian National Research Medical University, Moscow, 117997 Russia
²Engelhardt Institute of molecular biology RAS, Moscow, Russia



Introduction

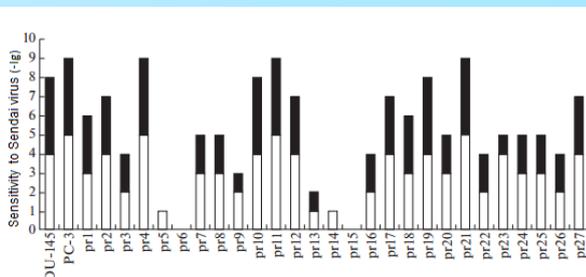
Oncolytic virotherapy is a new promising approach to treatment of various oncological diseases. At present Sendai Virus (SeV) is being actively investigated as a promising oncolytic drug. A number of studies have shown that recombinant and UV inactivated SeV is capable of effective elimination of tumor cells while sparing normal cells. SeV eradicates tumor cells mostly via induction of apoptosis, autophagy and necroptosis and, also, by induction of antitumor immunity

Materials & Methods

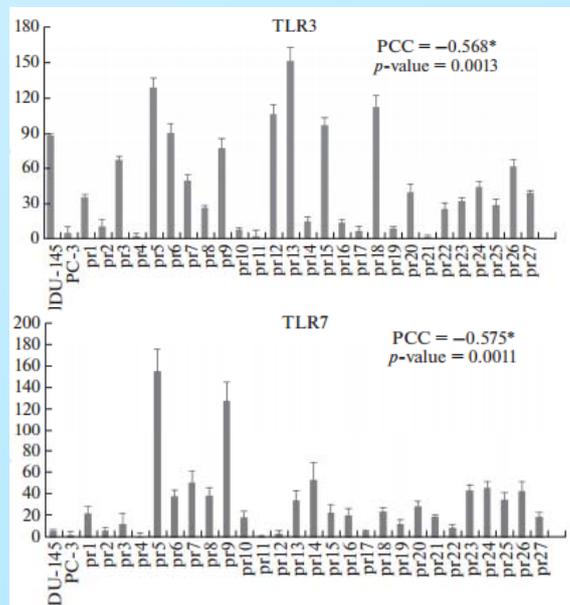
Sensitivity primary cell line of prostate cancer to Sendai virus was measured in Reed–Muench method. . Quantitative PCR was used to estimate levels of expression of genes encoding RIG-I like and Toll like receptors (TLR).

Results

In this study we have demonstrated that primary lines derived from adenocarcinoma of prostate exhibit a differential sensitivity to SeV. It's necessary to uncover genetic markers of interaction between malignant cells and viruses to enable a personalized approach to treatment and increase effectiveness of virotherapy.



Quantitative PCR demonstrated that levels of expression of TLR3 and TLR7 correlated with the degree of sensitivity of tumor cells from prostate gland to Sendai virus in a statistically significant way. Cell lines with the least expression of TLR3 and TLR7 exhibit the highest degree of sensitivity to that virus. A more detailed investigation of the mechanism of action of that viral strain will allow to uncover additional markers that would enable prediction of sensitivity of tumors from a particular patient to SeV and effectiveness of the oncolytic drug in the therapeutic regimen used with an utmost accuracy.



Acknowledgements

The work was supported by Russian Ministry of Education grant RFMEFI60714X0067 supported by the Russian Ministry of Education, unique project code - RFMEFI60714X0067.

Contact: Anastasiia O. Sosnovtseva
aososnovtseva@gmail.com