Molecular mechanisms behind *TRIM28* expression

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Popular science summary

Autoimmune diseases are a group of diseases where the immune system turns against its hosts and can result in discomfort, pain or even damage of tissue and/or organs that can threaten one's life or sometimes lead to terminal illness. There is a lot of research around these diseases but still many mechanisms remain unknown and there are no treatments repairing the malfunctioning immune system. Some of this research is focused on the interferon (IFN) system that is involved in the pathogenesis of a number of autoimmune diseases, each characterised by an interferon signature. The system is complex and not fully understood and researchers are working on revealing mechanisms of various elements of the system.

TRIM28 encodes a protein able to modulate gene expression of other genes and therefore can be an important element in the IFN system. Since our unpublished data proved strongly that IFN regulates *TRIM28* expression we decided to focus on mechanisms behind its expression. In our study we focused on two elements of its transcriptional and post-transcriptional regulation: the first in order to find out if there are any transcription factors affecting the expression and second to see if the *TRIM28* transcript is affected by any miRNAs, post-transcriptionally. We found that the transcription factor E2F1 and miR-582 are strong candidates for the *TRIM28* expression regulation but they are not controlled by type I IFN *in vivo*.

E2F1 and miR-582 are possible mediators in IFN-β regulation of *TRIM28* expression and the discovery is a step forward in understanding the role of TRIM28 in the IFN system.

Degree project 30 hp

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