

Poster presentation

Expression and role of type I interferons in primary mouse neurons after infection with Theiler's virus

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Type I interferons (IFN-I), also called IFN-alpha/beta, are cytokines that are induced upon viral infection. These cytokines play a crucial antiviral role. Studies in mice with IFN-I receptor gene deficiency have highlighted the importance of IFN-I system against central nervous system viral infections.

Due to their essential role and their limited capacity to renew, neurons are thought to possess their own strategies to limit viral infections by favouring non-cytolytic clearance.

Upon infection of mice with neurotropic viruses (Theiler's virus and La Crosse virus), we have previously shown that neurons are able to take part in immune antiviral response by expressing IFN-I. Nevertheless, only 3% of La Crosse virus-infected neurons were detected as IFN-I producers, suggesting that IFN-I expression could be more restricted in neurons than in other cell types [1].

We decided to compare IFN-I expression but also IFN-mediated response in primary mouse neurons and in fibroblasts infected with Theiler's virus to determine if IFN-I system is differently regulated in these two cell types.

We observed that a large majority of infected-neurons express IFN-I *in vitro*, from 12 h post-infection. As in fibroblasts, IFN-I expression in neurons is amplified by a positive feedback loop. At present, we are comparing the

importance of MDA-5 sensor for Theiler's virus detection in neurons and in fibroblasts.

Although IFN-I expression seems to be similarly regulated in neurons and fibroblasts, preliminary results let us to think that response to IFN could vary between neurons and fibroblasts.

References

1. Delhaye S, Paul S, Blakqori G, Minet M, Weber F, Staeheli P, Michiels T: **Neurons produce type I interferon during viral encephalitis.** *Proc Natl Acad Sci USA* 2006, **103**:7835-784.