

Inmed Conference Room

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**Invited by: Jérôme EPSZTEIN**

It is now widely recognized that oxytocin (OT) has potent analgesic properties (1). We recently contributed to better understand how analgesia can be achieved when OT is released in the spinal cord (central action) and in the blood (peripheral action) (2,3). In fact, a small population of oxytocinergic parvocellular neurons in the paraventricular nuclei seems to coordinate these analgesic effects with different time scale (4). OT is also known to be a key player during early life development. Therefore, we examined the impact of neonatal maternal separation (NMS) on the nociception ontogeny and on the efficacy of oxytocinergic analgesia. We already described that NMS is associated with pain hypersensitivity of newborn rodent pups and of adult rats (5). We also found that several pain inhibitory controls, including those using OT, are non-functional in NMS-exposed young and adult rats. A pharmacological rescue, aimed at restoring OT levels, its downstream signaling or preventing epigenetic changes has been attempted. Together, these results suggest that early life stress durably impairs the adaptative processes of rats submitted to pain and non-painful stress. This might be of importance to better understand the role of OT in other types of neurodevelopmental pathologies.

Bibliography

1. Poisbeau, Grinevich, Charlet (2017) Curr Top Behav Neurosci.

2. Juif & Poisbeau (2013) Pain

3. Juif et al (2013) J Neurosci

4. Eliava et al (2016) Neuron

5. Juif et al (2016) Eur J Neurosci

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**Oxytocin analgesia and its impairment after**

**early life adverse events**

**SEMINAR**

**EXTERNAL**