Introductory Remarks to Symposium 15

The brain oxytocin system - its complex impact on autism, social behavior, and stress

Benjamin Jurek and Adam Steven Smith, Regensburg and Lawrence, USA

In recent years, the neuropeptide oxytocin (OXT) attracted considerable attention of scientists due to its ability to modulate aspects of socio-emotional behavior and stress, which we will present and discuss in this symposium on a psychological, behavioral, and molecular level in humans, rodents, and cells.

The use of OXT as pharmacological treatment option for core symptoms of autism spectrum disorder has been anticipated by studies showing that intranasal application of OXT temporarily enhances social cognition, empathy, and reciprocity in autistic patients. During behavioral interventions, individuals with autism spectrum disorder showed OXT-induced enhanced learning when the learning target is social. Such behavioral interventions rely on a core mechanism, i.e. social reinforcement learning (Schulte-Rüther).

In order to complement psychological studies in humans with the molecular processes that regulate behavior, animal research is of paramount importance. For instance, OXT dampens anxiety-related behavior in stressed voles via the activation of GABAergic OXTR neurons in the hypothalamic paraventricular nucleus (PVN), which in turn diminish the activity of stress-responsive hypothalamic corticotropin-releasing factor neurons (Smith).

We will also consider the molecular events involved in OXTR binding, the main signaling pathways activated by the OXTR and on intracellular and plasma membrane OXTR trafficking, all of which contribute to the quantitative and qualitative features of OXT responses in the brain (Busnelli).

Finally, the effects of chronically applied OXT, in contrast to an acute infusion, on anxiety-related behavior will be addressed. Chronic OXT activates specific transcription factors, such as MEF-2. Dysregulated MEF-2 is associated with autism spectrum disorder and activated by the OXTR, thereby providing the molecular mechanism that links social aspects, anxiety/stress and intracellular signaling (Jurek).

By addressing such diverse aspects of OXT research, we hope to create a comprehensive picture of the differential effects and their involvement in autism, social behavior, and stress.

Symposium 15

Thursday, March 21, 2019
14:30 - 16:30, Lecture Hall 104

Chairs: Benjamin Jurek and Adam Steven Smith, Regensburg and Lawrence, USA

14:30 **Opening Remarks**

14:35 Martin Schulte-Rüther, Aachen
SOCIAL REINFORCEMENT LEARNING AND ITS NEURAL MODULATION BY OXYTOCIN IN AUTISM SPECTRUM DISORDER (S15-1)

14:55 Adam Steven Smith, Lawrence, USA
OXYTOCIN AND SOCIAL CONTACT REDUCE ANXIETY (S15-2)

15:15 Marta Busnelli, Milan, Italy
OXYTOCIN: ITS SIGNALING OF ACTION AND RECEPTOR SIGNALLING IN THE BRAIN (S15-3)

15:35 Benjamim Jurek, Regensburg
THE BRAIN OXYTOCIN SYSTEM AND ITS COMPLEX IMPACT ON STRESS AND ANXIETY (S15-4)

16:00 Magdalena Meyer, Regensburg
OXYTOCIN ALTERS THE MORPHOLOGY OF HYPOTHALAMIC NEURONS VIA THE TRANSCRIPTION FACTOR MYOCYTE ENHANCER FACTOR 2A (MEF-2A) (S15-5)

16:10 Dominik Fiedler, Münster
BRAIN-DERIVED NEUROTROPHIC FACTOR MODULATES SYNAPTIC PROPERTIES OF OVBNST NEURONS VIA TRKB RECEPTORS (S15-6)

16:20 **Concluding Remarks**