NMDA receptors are fundamental for both the physiology and the pathology of the nervous system. They control plasticity-related events and adaptive processes in the nervous system, which includes long-term potentiation (LTP), memory formation and the build-up of a neuroprotective shield. However, NMDA receptors can also bring about destruction and cell death. The discovery that the location of the NMDA receptor matters resolved the ‘NMDA receptor paradox’ and provided a unifying concept. NMDA receptors localized to the synapse and activated by synaptic inputs promote neuronal survival, gene expression and plasticity. In contrast, NMDA receptors that are located outside synaptic contacts - the so-called extrasynaptic NMDA receptors - couple to death signaling pathways. The concept ‘death through extrasynaptic NMDA receptor signaling’ has transformed our views on pathogenesis and therapeutic strategies for neurodegenerative and excitotoxic disorders. Increased extrasynaptic NMDA receptor signaling is now considered a key factor in the etiology of human neurodegenerative diseases, including Huntington’s disease, Alzheimer’s disease, and stroke. Extrasynaptic NMDA receptors have become an important target for the development of therapeutic interventions. Meantime, an FDA approved blocker of preferentially extrasynaptic NMDA receptors is the first drug that is being used successfully to treat Alzheimer’s disease patients.
Symposium 33

Saturday, March 23, 2019
8:30 - 10:30, Lecture Hall 103

Chair: Hilmar Bading, Heidelberg

08:30  Opening Remarks

08:35  Hilmar Bading, Heidelberg
THE NMDA RECEPTOR PARADOX: PRO-SURVIVAL VERSUS DEATH SIGNALING (S33-1)

09:00  Lynn A. Raymond, Vancouver, Canada
ROLE FOR EXTRASYNAPTIC NMDA RECEPTORS IN PRODROMAL HUNTINGTON DISEASE: MECHANISMS AND THERAPEUTIC IMPLICATIONS (S33-2)

09:25  Giles E. Hardingham, Edinburgh, UK
PROBING THE ROLES OF GLUN2 C-TERMINAL DOMAIN SIGNALLING IN HEALTH AND DISEASE (S33-3)

09:50  Stuart A. Lipton, La Jolla, USA
THE NOVEL NMDAR ANTAGONIST NITROSYP-NAPSIN AS THERAPY FOR HIPSC- AND MOUSE-MODELS OF HUMAN AUTISM SPECTRUM DISORDER (S33-4)

10:15  Liliana Rojas-Charry, Hamburg
SPECIFIC MUTATIONS IN PRESENILIN 1 HAVE A DIFFERENTIAL ROLE ON MITOCHONDRIAL PHENOTYPE AND FUNCTION (S33-5)

10:25  Concluding Remarks