

**ADNF Symposium VII**  
**Thursday, 29 April 2021**  
**9:00am AEST**

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The symposium will start with A/Prof Jenny Gunnensen, University of Melbourne, who will present “New insights into mechanisms of excitatory synapse development”. Then, Dr Tommas Ellender, University of Oxford UK, will deal with the “Embryonic neural progenitor pools and the generation of fine-scale neural circuits” and Dr Thomas Marissal, INMED France will talk about “Parvalbumin interneurons: the missing link between the micro and macroscopic alterations related to neurodevelopmental disorders?”

**Jenny Gunnensen (Australia)**

A/Prof Jenny Gunnensen leads a research team investigating the development and plasticity of neuronal circuits and the pathological bases of developmental and neurodegenerative disorders. Jenny received her BSc(Hons) in Marine Biochemistry from James Cook University in 1986 and her PhD in molecular endocrinology from the University of Melbourne in 1994. In her first post-doctoral position, she worked with Michael Sendtner in Würzburg, Germany investigating trophic factors for motoneuron survival and regeneration. In 1998, Jenny returned to Melbourne to work with Seong-Seng Tan in the Brain Development Group at the Howard Florey Institute. From 1998-2008, Jenny held an NHMRC Howard Florey Centenary Post-doctoral Research Fellowship (part-time), a Neurosciences Victoria / Centre for Neuroscience Fellowship (part-time) and a Senior Research Fellow position at the Florey Neuroscience Institutes. During this period, she utilized emerging gene expression profiling techniques to obtain the first molecular inventory of the developing cortex and created gene knockout mouse models to determine functional roles for some of the novel genes identified. In 2011, Jenny moved to the Department of Anatomy and Neuroscience at the University

of Melbourne where she holds a Teaching and Research position. Her work, funded by the NHMRC, is focussed on (i) molecular and cellular mechanisms controlling synapse development; (ii) synapse loss in the earliest stages of Alzheimer's disease and how this might be slowed or prevented; (iii) synapse formation/strengthening and how these processes contribute to the pathology of psychostimulant abuse and neuropathic pain.

### **Tommas Eildner (UK)**

Dr Tommas Ellender is a cellular neuroscientist and group leader of the Neuronal Circuit Research laboratory based at the Department of Pharmacology at the University of Oxford. He studied Biology at Leiden University in the Netherlands and travelled to Oxford for his DPhil to work with Prof. Ole Paulsen and subsequently stayed there for his postdocs with Prof. Paul Bolam and Prof. Colin Akerman. During his time as a postdoc he became fascinated by both the workings of the basal ganglia as well as the cellular mechanisms at play in controlling the development of this structure. Over the last few years work in his lab has focussed on the input nucleus of the basal ganglia; the striatum and his lab has been investigating how different types of embryonic progenitor shape the properties of this circuit. His lab is funded by the Wellcome Trust, Medical Research Council and the Royal Society and he currently sits on the Editorial Board of the Journal of Physiology.

### **Thomas Marissal (France)**

Dr Thomas Marissal is an associate researcher at the Mediterranean Institute of Neurobiology (INMED), France. He obtained his PhD at the INMED in the lab of Prof Yehezkel Ben-Ari and Prof Rosa Cossart, where he found that CA3 pyramidal cells, a population of apparently homogenous excitatory neurons, were divided in morpho-functionally distinct subtypes rooted in different temporal embryonic origins (Marissal et al., Nature Communications; 2012). Then he moved to Geneva (Switzerland) in 2012 for a post-doctorate training with Prof. Dominique Müller and Prof. Alan Carleton, where he demonstrated that an impairment in the excitability of parvalbumin-expressing interneurons caused the alteration of neuronal network synchronization in the hippocampus from a mice model related to the most frequent genetic form of schizophrenia (22q11.2 DS). He showed that restoring parvalbumin interneuron excitability using pharmacological or chemogenetic strategies was sufficient to rescue in vitro and in vivo network dynamics and behavior (Marissal et al., Nature Neuroscience; 2018). In 2019, he joined the INMED as associate researcher, in the lab of Prof Valérie Crépel, where he studies the pathophysiological role of peculiar neuronal subpopulations, focusing on hippocampal inhibitory neurons.