

## Urs Meyer, Ph.D.

MIND Institute Distinguished Lecturer Series – October 17, 2017

### Biographical Information

Dr. Meyer earned his PhD in behavioral neurobiology from the Swiss Federal Institute of Technology (ETH) Zurich, Switzerland, in 2007. He then spent post-doctoral fellowships at the Department of Psychiatry and Psychotherapy, Ludwig-Maximilian University, Munich, Germany, and at the Behavioral Neurobiology Laboratory, ETH Zurich, Switzerland. Between 2011 and 2014, he was a Group Leader and Lecturer at the Physiology and Behavior Laboratory, ETH Zurich, Switzerland. In 2015, he was appointed as Associate Professor of Pharmacology at the Institute of Pharmacology and Toxicology, University of Zurich – Vetsuisse, Switzerland. He is also a faculty member at the Neuroscience Center Zurich, University of Zurich and ETH Zurich, Switzerland. Prof. Meyer's main research interests are centered upon the question of how early-life environmental adversities such as prenatal infection, pubertal stress, and nutritional imbalances can influence brain development and shape the risk of long-term brain abnormalities. His research team combines behavioral and cognitive tests, immunological assays and neuroanatomical techniques in rodent models, including mouse models of gene-environment and environment-environment interactions relevant to multifactorial neurodevelopmental disorders such as schizophrenia and autism. His research also includes molecular investigations to examine the role of epigenetic processes in environmentally induced brain pathologies and uses pharmacological approaches with the aim to establish novel symptomatic and preventive treatments against chronic brain disorders with neurodevelopmental origins.

### Presentation Abstract (4:30pm presentation)

#### *Maternal infection and immune involvement in neurodevelopmental disorder*

Prenatal exposure to infection is increasingly recognized to play an important etiological role in neuropsychiatric and neurological disorders with neurodevelopmental components, including schizophrenia, autism, bipolar disorder, and mental retardation. The adverse effects induced by prenatal infection may reflect an early entry into a deviant neurodevelopmental route, but the specificity of subsequent disease or symptoms is likely to be influenced by the genetic and environmental context in which the prenatal infectious process occurs. The epidemiological link between prenatal infection and increased risk of neurodevelopmental disorders also receives strong support from experimental work in animal models. These models are based on maternal gestational exposure to specific infectious agents such as influenza virus or immune activating agents such as the bacterial endotoxin lipopolysaccharide (LPS) or the viral mimic poly(I:C). Converging evidence from these models suggests that prenatal immune activation can negatively affect early fetal brain development and change the offspring's neurodevelopmental trajectories, which in turn can lead to the emergence of behavioral and cognitive disturbances in later life. Modeling the human epidemiological association between prenatal infection and increased risk of neurodevelopmental disorders in animals has also greatly advanced our understanding of the underlying mechanisms. According to the prevailing view, cytokine-associated inflammatory events, together with downstream pathophysiological effects such as oxidative stress and (temporary) macronutrient and micronutrient deficiency, seem critical in mediating the post-acute effects of maternal infection on the fetal system. Recent findings have further implicated epigenetic processes as possible molecular mechanisms translating the negative effects of prenatal immune activation on the offspring. Not only does prenatal immune activation cause long-lasting epigenetic modifications such as altered DNA methylation and miRNA expression, but it also causes a transgenerational transmission of behavioral and neuronal abnormalities without additional immune exposures. Hence, prenatal infection and associated developmental neuroinflammation may have a pathological role in shaping neurodevelopmental disease risk across generations.