

Michael Heneka



Transition from acute to chronic neuroinflammation (TracInflam)

Project Coordinator: Michael Heneka, Department of Neurology, University Hospital Bonn, Bonn, Germany

Project Partners: Joseph Bertrand, Dept. of Oncology-Pathology, Karolinska Institutet, Stockholm, Sweden

Séverine Boillée, Institut du Cerveau et de la Moelle épinière (ICM), Paris, France Eran Segal, Dept. of Computer Science and Applied Mathematics, Weizmann Institute of Science, Rehovot, Israel

Marie-ÈveTremblay,Dept.ofMolecularMedicine,UniversitéLaval,Quebec,Canada.

Neuroinflammation is how the brain's immune system fights disease. As a natural defense mechanism, this reaction may harbor beneficial effects, but under circumstances not yet well understood, it may also have detrimental consequences for the brain and even contribute to the progression of the disease that initially stimulated the immune reaction.

TracInflam will study the mechanisms by which neuroinflammation can transition from a beneficial to a detrimental outcome in preclinical models of Alzheimer's disease (the most prevalent form of dementia), amyotrophic lateral sclerosis (the most common motor neuron disease), and septic encephalopathy (the leading cause of mortality in intensive care units), three diseases sharing a prominent inflammatory component. In addition, it aims to identify genetic factors marking the key steps of this transition, both in preclinical models and in samples of patients suffering from the respective diseases, in order to identify points of intervention for early diagnosis and the development of better targeted and more efficient therapeutic strategies.



BrdU incorporation indicative of proliferation in an Iba-1 immunopositive microglial cell at the site of focal activation in an 8-month old APP/PS1 transgenic mouse