Università degli Studi dell'Insubria
**Centro di Ricerca in Farmacologia Medica**
*University of Insubria – Center for Research in Medical Pharmacology*

# Seminario

**Venerdì 23 marzo 2018**
**ore 14:30**

**Aula Magna del Collegio universitario, Via Dunant n. 7 – Varese**

# Of inflammation and depression, of … what?

## *The effects of inflammation on behavior*

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**Moderatore: Marco Cosentino**

***In collaborazione con:***

**Corso di Dottorato di ricerca in**
**MEDICINA CLINICA E SPERIMENTALE E *MEDICAL HUMANITIES***

**Scuola di specializzazione in FARMACOLOGIA MEDICA**

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**Jan Pieter Konsman, PhD**, is a senior scientist in the National Center for Scientific Research (CNRS) Aquitaine Institute for Cognitive and Integrative Neuroscience (INCIA) at the University of Bordeaux.

**Mini-bio -** Jan Pieter Konsman, received a Masters Degree in Neurobiology from the University of Groningen, the Netherlands. He obtained his PhD from the Bordeaux University in France for his work on Immune-to-Brain communication. He then spent three years on post-doc at the University of LinKöping in Sweden working on the distribution of the Interleukin-1 receptor in the rat brain. Dr. Konsman next took up a permanent position at the Centre National de Recherche Scientifique (CNRS) in France working on the neuroimmune substrates of disease-associated anorexia and apathy. His current research focus is on Magnetic Resonance Imaging of septic encephalopathy. He also holds a Masters Degree in History and Philosophy of Science from the Montaigne University of Bordeaux.

## Research interests

- Neuroimmune interactions
- Neuronal substrates of disease-associated anorexia and behavioral depression
- Small animal brain imaging
- History and philosophy of biology of medicine

**More at:** https://www.researchgate.net/profile/Jan_Pieter_Konsman

## Research

The overall focus of my research has been on unraveling the **neuroimmune mechanisms underlying so-called non-specific disease symptoms such as anorexia and apathy** and includes work on animal models of bacterial sepsis and cancer.

Our early work addressed the question of how the immune system signals the nervous system during systemic inflammation to bring about fever as well as a reduction of food intake and social interactions typical of disease. As body temperature, food intake and social behavior are regulated by the brain, changes thereof during infectious and inflammatory disease indicate that mediators released by immune cells can modify central nervous system functioning. However, soluble immune mediators such as cytokines cannot passively cross the blood-brain barrier that separates the systemic circulation from brain parenchyma. The decade 1994-2004 proved a very active research period to elucidate immune-to-brain communication pathways. By following a functional anatomical approach addressing different hypotheses rather than presenting evidence in favor of just one hypothesis, we were able to establish a balanced view of potentially redundant pathways both in a series of original and review articles.

As a follow-up of this work elucidating immune-to-brain signaling pathways, we next addressed the neurobiological mechanisms underlying fever as well as reduced food intake and social interactions during systemic inflammation. To do so, we designed pharmacological intervention studies aimed at blocking the cerebral actions of inflammatory mediators or classic neurotransmitter systems and addressed their effects both on fever and sickness behavior as well as on cellular activation patterns across brain structures. Thus, we have been able to establish that the reduction of social interactions during systemic inflammation depends on the forebrain action of the pro-inflammatory cytokine interleukin-1, while the reduction of food intake involves the brainstem action of glutamate.

In addition, we have been able to show that the cellular activation patterns and the increase of some transcripts that occur in hypothalamic and striatal structures in response to food restriction in healthy animals do not occur in tumor-bearing rats. These findings may explain why an increase in food intake and ensuing restoration of initial body weight is often not observed during cancer. I served as the primary investigator in these studies. In addition, we have seen that even treatment with a chemotherapeutic drug does not allow to restore initial food intake and body weight once the acute anorectic effects of chemotherapy have waned.

Finally, I have also participated with my collaborators in setting up an integrated imaging approach consisting of different modalities of in vivo magnetic resonance imaging (MRI) and post mortem histology in small animal models of sepsis and cancer. The aim of combing MRI and histology is to identify the cells or the cellular changes underlying the observed imaging contrast.