B·DEBATE International Center for Scientific Debate BARCELONA

Sant Joan de Déu HOSPITAL MATERNOINFANTIL - UNIVERSITAT DE BARCELONA

Fundació Sant Joan de Déu

# HENRIK HAGBERG

CV

PARTICIPANT AT:

## EXTREMELY PRETERM BABIES. IMPROVING PERINATAL CARE

**January, 16th-17th, 2013, Barcelona**

**Henrik Hagberg** Professor, Chair in Fetal Medicine, Centre for the Developing Brain, Perinatal Imaging & Health, Kings College London, London

Henrik Hagberg, MD., PhD., is Professor, Chair in Fetal Medicine, Centre for the Developing Brain, Perinatal Imaging & Health, Kings College London, London. Before he was Professor of Obstetrics and Perinatal Medicine, Institute of reproductive & developmental biology, Imperial College, London and Professor in Perinatal Medicine and Obstetrics, Institute for the Health of Women and Children, Sahlgrenska Academy, Göteborg University. Guest Professor at the Neuroscience Lab, Kennedy Krieger Institute, Johns Hopkins School of Medicine, Baltimore, USA. Associate Professor and senior clinical consultant in Perinatal Medicine and Obstetrics (1995-98). Assistant Professor (Docent) in Obstetrics and Gynecology, Göteborg and consultant physician at the Dept of Obstetrics and Gynecology, Sahlgrenska University Hospital (1993-95). Henrik Hagbert has published 235 original articles, 1 manuscript, 51 reviews or book chapters, H-index: 54, average citation/article: 34. His laboratory demonstrated that excitatory amino acids (EAAs) are released also in the immature brain in response to asphyxia in a clinically relevant model and into the CSF of asphyxiated babies. It is the first group showing that post-treatment with EAA receptor antagonists reduced perinatal brain injury and the degree of EAA increase in CSF correlated to outcome in asphyxiated babies. They have published several papers in adult and neonatal animals demonstrating that NMDA receptor dependent ion permeability and metabolic activity is enhanced after hypoxic-ischemic insults, which may be relevant for the neuroprotective efficacy of post-treatment with these agents. They have found that NMDA receptor activation enhances glucose utilization during reperfusion in the immature brain, which is most likely due to an impairment of mitochondrial function. Subsequent studies have also demonstrated a link between the NMDA receptor, mitochondrial function and triggering of the apoptotic cascade encompassing the mitochondrial release of pro-apoptotic molecules. We have also demonstrated that immune cells (microglia, T-cells, neutrophils) as well as cytokines and chemokines is involved in the inflammatory response in the immature CNS (clinical and experimental studies).