CECAD Excellence Cluster. University of Cologne

PRESS RELEASE

Astrid Bergmeister. CECAD PR & Marketing

New Perspectives for Patients suffering from Brain Injury – Cologne CECAD researchers investigate metabolic processes in inflamed nerve tissue

Cologne, December 03, 2013. The new findings from research on metabolic activities in inflamed nerve tissue carried out by Dr Matteo Bergami from the CECAD Excellence Cluster at the University of Cologne in collaboration with an international team of scientists generate new perspectives for many patients suffering from traumatic brain injury. Their results open the path to a deeper understanding of the processes that take place in damaged nerve cells, particularly in astrocytes. These cells play fundamental roles in supporting neuronal functioning and in regulating brain energy balance. This study now shows that inflammation can lead to changes in astrocyte mitochondria, causing them to fragment. The researchers identified key regulators that allow astrocytes to keep functional their mitochondrial network during neuroinflammation, a condition commonly associated to brain injury as well as to a variety of other brain diseases.

Joining their efforts, a team of researchers from the Excellence Clusters of Cologne (CECAD) and Munich (LMU), together with the University of Bologna (Italy), can now offer new insights into the metabolic processes taking place in damaged brain tissue. Their research has focused on astrocytes, the cells in the central nervous system that regulate energy metabolism and synapse functioning. Given their important physiological role in the healthy brain, researchers have addressed how astrocytes may change their metabolic activity in response to nerve tissue inflammation, a condition caused by acute injury, stroke, or neurodegenerative diseases. This may reveal to be an essential aspect of most brain diseases as a failure of astrocyte reactivity during an inflammatory process may worsen the pathology and eventually accelerate neurodegeneration.

To date it has been assumed that nervous system cells react uniformly to acute brain damage such as that caused by traumatic injury. However, Dr Bergami and his team of researchers discovered that astrocytes within different zones of the lesion show different forms of reactivity in response to inflammatory insults. This reactivity especially affects mitochondria, the powerhouse of the cells.
The function of mitochondria is strictly dependent upon two types of dynamics: fusion and fission. These two reactions are key for maintaining mitochondrial architecture and function. Faulty regulation of these mitochondrial dynamics results in defective mitochondria, which can lead to cellular aging and trigger many neurodegenerative diseases.
The researchers were able to show that astrocytic mitochondria within the core of the damaged, highly proinflammatory brain area, demonstrate an accelerated tendency towards fission, leading to their fragmentation. In the surrounding zones, mitochondria show an increase in fusion.
The researchers also succeeded in discovering a key metabolic process regulating astrocyte mitochondrial function: they were able to show that autophagy, a process involving the self-digestion of components in the cell, is critical to maintain mitochondrial structure. In contrast to neurons, astrocytes survive surprisingly well to acute inflammation. The new study reveals that autophagy is the major mechanism conferring this resistance. When autophagy is ablated astrocytes loose their capability to regenerate their network which ultimately leads to astrocyte cell death.

Although the reorganisation of metabolic pathways triggered by inflammation goes beyond the influence of mitochondria, these research findings clearly demonstrate that mitochondrial function is absolutely essential to astrocyte survival. Additionally they provide new insights for our understanding of how brain cells react to inflammation. Further characterization of these metabolic pathways may hopefully enable the researchers to protect neurons from dying during acute or chronic neuroinflammation. This will potentially allow for the development of new approaches aimed at helping patients exposed to brain injury or stroke, in order to preserve brain function and improving the patient´s quality of life.

Contact:
Dr Matteo Bergami
CECAD Cluster of Excellence at the University of Cologne
Tel. +49 (0) 221 478 841 71
matteo.bergami@uk-koeln.de

Astrid Bergmeister
Head of CECAD PR & Marketing
Tel. + 49 (0) 221-478-84043
astrid.bergmeister@uk-koeln.de