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REVIEW ARTICLE
1079  Glial endozepines and energy balance: Old peptides with new tricks
Bruno Lebrun, Manon Barbot, Marie-Christine Tonon, Vincent Prévot, Jérôme Leprince, and Jean-Denis Troadec
MAIN POINTS:
- Glial endozepines expression is modulated by metabolic signals.
- Endozepines were reported to exert anorexigenic effect by acting at the hypothalamus level.
- Recent developments reveal a complex and multifaceted mode of action.

RESEARCH ARTICLES
1094  Beneficial contribution of induced pluripotent stem cell-progeny to Connexin 47 dynamics during demyelination-remyelination
Sabah Mozafari, Cyrille Deboux, Cecilia Laterza, Marc Ehrlich, Tanja Kuhlmann, Gianvito Martino, and Anne Baron-Van Evercooren
MAIN POINTS:
- Loss of oligodendrocyte Cx47 expression caused by lysolecithin (LPC)-induced demyelination is timely rescued during remyelination.
- Panglial repair correlates with remyelination and is accelerated by the grafted iPSC-neural precursor cells.
1110 Roles for H⁺/K⁺-ATPase and zinc transporter 3 in cAMP-mediated lysosomal acidification in bafilomycin A1-treated astrocytes
Huikyong Lee and Jae-Young Koh
MAIN POINTS:
- When astrocytic lysosomal pH is altered by v-ATPase inhibition, cAMP reverses the pH change by recruiting H⁺/K⁺-ATPase in a ZnT3-dependent manner.

1126 A reporter cell system for the triggering receptor expressed on myeloid cells 2 reveals differential effects of disease-associated variants on receptor signaling and activation by antibodies against the stalk region
Melanie Ibach, Mona Mathews, Bettina Linnartz-Gerlach, Sandra Theil, Sathish Kumar, Regina Feederle, Oliver Brüstle, Harald Neumann, and Jochen Walter
MAIN POINTS:
- Disease associated variants impair the signaling activity of TREM2 by distinct mechanisms.
- Targeting the stalk region of TREM2 with bivalent antibodies activates TREM2 signaling.

1140 Regionally diverse astrocyte subtypes and their heterogeneous response to EAE
Malte Borggrewe, Corien Grit, Iliya D. Vainchtein, Nieske Brouwer, Evelyn M. Wesseling, Jan D. Laman, Bart J. L. Eggen, Susanne M. Kooistra, and Erik W. G. M. Boddeke
MAIN POINTS:
- ACSA and GLAST can be used to FACS-sort astrocyte subtypes.
- Astrocytes in forebrain and hindbrain consist of two subtypes based on GLAST expression.
- Spinal cord astrocytes are reactive in acute EAE and are more proliferative in chronic stages.
The complement C3-C3aR pathway mediates microglia–astrocyte interaction following status epilepticus

Yujia Wei, Tingjun Chen, Dale B. Bosco, Manling Xie, Jiaying Zheng, Aastha Dheer, Yanlu Ying, Qian Wu, Vanda A. Lennon, and Long-Jun Wu

MAIN POINTS:
- Microglia are required for astrocytes activation in experimental status epilepticus.
- C3 from astrocytes activates microglia via C3a receptors.
- Microglia-astrocyte interaction promotes gliosis and neuronal injury after seizures.

Endocytosis of the glutamate transporter 1 is regulated by laforin and malin: Implications in Lafora disease

Eva Perez-Jimenez, Rosa Viana, Carmen Muñoz-Ballester, Carlos Vendrell-Tomero, Raquel Moll-Diaz, Maria Adelaida Garcia-Gimeno, and Pascual Sanz

MAIN POINTS:
- Laforin/malin complex ubiquitinates GLT-1 glutamate transporter.
- Laforin/malin complex modulates the endocytosis of GLT-1 negatively.
- Laforin/malin complex opposes the effect of Nedd4.2 on GLT-1, by interacting and ubiquitinating α- and β-arrestins.

Kif3a deletion prevents primary cilia assembly on oligodendrocyte progenitor cells, reduces oligodendrogenesis and impairs fine motor function

Carlie L. Cullen, Megan O’Rourke, Shannon J. Beasley, Loic Auderset, Yilan Zhen, Renee E. Pepper, Robert Gasperini, and Kaylene M. Young

MAIN POINTS:
- OPCs disassemble primary cilia upon cell cycle re-entry.
- Deletion of Kif3a from OPCs prevents primary cilia assembly and reduces OPC proliferation.
- Deletion of Kif3a from adult OPCs in vivo reduces oligodendrogenesis and impairs motor function.
Mesenchymal stem cells instruct a beneficial phenotype in reactive astrocytes
Tiziana Vigo, Androniki Voulgari-Kokota, Mariella Errede, Francesco Girolamo, Jasmin Ortolan, Maria C. Mariani, Giovanni Ferrara, Daniela Virgintino, Annalisa Buffo, Nicole Kerlero de Rosbo, and Antonio Uccelli
MAIN POINTS:
- Exogenous MSC prevent astrogliosis and reduce expression of astrocyte-derived factors that sustain immune cell infiltration and inhibit remyelination in EAE.
- MSC promote acquisition of neural stem cell-like phenotype in reactive astrocytes.

Microglial recruitment and mechanisms involved in the disruption of afferent synaptic terminals on spinal cord motor neurons after acute peripheral nerve injury
Sara Salvany, Anna Casanovas, Lídia Piedrafita, Olga Tarabal, Sara Hernández, Jordi Calderó, and Josep E. Esquerda
MAIN POINTS:
- Early after axotomy, microglia recruited near injured motor neurons, emit processes that tend to contact their afferent synaptic terminals.
- Extracellular vesicles resulting from necroptotic synaptic disruption are removed by microglia.

Reducing L-lactate release from hippocampal astrocytes by intracellular oxidation increases novelty induced activity in mice
Barbara Vaccari Cardoso, Alexey V. Shevelkin, Chantelle Terrillion, Olga Mychko, Valentina Mosienko, Sergey Kasparov, Mikhail V. Pletnikov, and Anja G. Teschemacher
MAIN POINTS:
- Lactate oxidase (LOx) expression limits the intra-astrocytic pool of lactate and decreases its release.
- LOx in astrocytes of the dorsal mouse hippocampus causes increased activity in novel environment.
A loss of mature microglial markers without immune activation in schizophrenia

Gijsje J. L. J. Snijders, Welmoed van Zuiden, Marjolein A. M. Sneeboer, Amber Berdenis van Berlekom, Astrid T. van der Geest, Tatiana Schnieder, Donald J. MacIntyre, Elly M. Hol, René S. Kahn, and Lot D. de Witte

MAIN POINTS:
- Microglia density is unaltered in postmortem brain tissue of schizophrenia patients, but several mature microglial markers are downregulated in schizophrenia. This expression pattern is largely opposite from microglial changes in Alzheimer’s disease.

Microglial inflammasome activation drives developmental white matter injury

Rebecca K. Holloway, Graeme Ireland, Gemma Sullivan, Julie-Clare Becher, Colin Smith, James P. Boardman, Pierre Gressens, and Veronique E. Miron

MAIN POINTS:
- Microglia inflammasome activation correlates with white matter injury.
- Inflammasome inhibition promotes myelination.
- IL1β increases the activinA inhibitor follistatin, which impedes myelination.
- ActivinA enhances myelination following injury.

Single-cell transcriptomic profiling of satellite glial cells in stellate ganglia reveals developmental and functional axial dynamics

Valerie Y. H. van Weperen, Russell J. Littman, Douglas V. Arneson, Jaime Contreras, Xia Yang, and Olujimi A. Ajijola

MAIN POINTS:
- Satellite glial cells in murine stellate ganglia comprise five transcriptomic subtypes.
- Subtypes transition along two nonlinear, developmental and functional axes.
- Biochemical pathway analyses suggest dynamic transition between functional states.
Transcriptional profiling of microglia in the injured brain reveals distinct molecular features underlying neurodegeneration
Cong Liu, Shang-Kun Dai, Ruo-Xi Shi, Xuan-Cheng He, Ying-Ying Wang, Bao-Dong He, Xiao-Wen Sun, Hong-Zhen Du, Chang-Mei Liu, and Zhao-Qian Teng

MAIN POINTS:
- Microglia display distinct temporal and sexual molecular signatures of transcriptome after cortical injury.
- Hypotheses and gene candidates are presented to explore the roles of microglia in neurotrauma and in sex-biased neurodegenerative diseases.

Cover Illustration: Electron micrograph showing an area near the interaction sites between an Iba1-immunostained microglial cell (shaded in violet) and the surface of a motor neuron at the spinal cord ventral horn (shaded in red) following sciatic nerve transection. The extracellular space is delimited in yellow. Extracellular multilamellar bodies and vesicles derived from degenerating afferent synaptic boutons are seen interposed between the microglial cell and motor neuron cell body; some vesicles undergo a process of enwrapping and phagocytosis by microglia. (See Esquerda, JE, et al., https://doi.org/10.1002/glia.23959.)

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