

# Selected Publications

## CIRB - Physiology and physiopathology of the gliovascular unit

10 major publications of the team members during the last 5 years

- 1) Lebas H, Guerit S, Fournier A, **BOULAY AC**, Vivien D, **COHEN-SALMON M**, Docagne F, Bardou I. PAI-1 production by reactive astrocytes drives tissue dysfibrinolysis in multiple sclerosis models. *Cellular and Molecular Life Sciences* 2022, in press.

This study shows the implication of the Plasminogen Activator Inhibitor 1 expressed by astrocytes in Multiple sclerosis and proposes the inhibition of this astrocytic pathway as a new therapeutic strategy against multiple sclerosis.

- 2) **GILBERT A\***, **ELORZA-VIDAL X\***, Rancillac A, Chagnot A, Yetim M, Hingot V, Deffieux T, **BOULAY AC**, **ALVEAR-PEREZ R**, Cisternino S, Martin S, Taïb S, Gelot A, Mignon V, Favier M, Brunet I, Declèves X, Tanter M, Estevez R, Vivien D, Saubaméa B, **COHEN-SALMON M**. Megalencephalic leukoencephalopathy with subcortical cysts is a developmental disorder of the gliovascular unit. *eLife*. 2021 Nov 1;10:e71379. doi: 10.7554/eLife.71379.

This work demonstrates that Megalencephalic leukoencephalopathy with subcortical cysts (**MLC**), a rare type of leukodystrophy, is a developmental disorder of the gliovascular interface. We propose that gliovascular alterations in a MLC mouse model are the primary events that initiate the pathological cascade.

- 3) Escartin C, Galea E, Lakatos A, O'Callaghan JP, Petzold GC, Serrano-Pozo A, Steinhauser C, Volterra A, Carmignoto G, Agarwal A, Allen NJ, Araque A, Barbeito L, Barzilai A, Bergles DE, Bonvento G, Butt AM, Chen WT, **COHEN-SALMON M**, Cunningham C, Deneen B, De Strooper B, Diaz-Castro B, Farina C, Freeman M, Gallo V, Goldman JE, Goldman SA, Gotz M, Gutierrez A, Haydon PG, Heiland DH, Hol EM, Holt MG, Iino M, Kastanenka KV, Kettenmann H, Khakh BS, Koizumi S, Lee CJ, Liddelow SA, MacVicar BA, Magistretti P, Messing A, Mishra A, Molofsky AV, Murai KK, Norris CM, Okada S, Oliet SHR, Oliveira JF, Panatier A, Parpura V, Pekna M, Pekny M, Pellerin L, Perea G, Perez-Nievas BG, Pfrieger FW, Poskanzer KE, Quintana FJ, Ransohoff RM, Riquelme-Perez M, Robel S, Rose CR, Rothstein JD, Rouach N, Rowitch DH, Semyanov A, Sirko S, Sontheimer H, Swanson RA, Vitorica J, Wanner IB, Wood LB, Wu J, Zheng B, Zimmer ER, Zorec R, Sofroniew MV, Verkhratsky A.

Reactive astrocyte nomenclature, definitions, and future directions. **Nat Neurosci.** 2021 24:312-325. Review

This review summarizes research on astrocyte reactivity, redefining this concept and proposing a new nomenclature.

- 4) **MAZARÉ N\*, OUDART M, COHEN-SALMON M.** Local translation in perisynaptic and perivascular astrocytic processes - a means to ensure astrocyte molecular and functional polarity? **J Cell Sci.** 2021 Jan 22;134(2):jcs251629. Review.

This review summarizes our knowledge on local translation in astrocytes, a biological mechanism that we uncovered in astrocytes. We discuss its possible role in the functional astrocytic polarity.

- 5) Deshayes de Cambronne R, Fouet A, Picart A, Bourrel AS, Anjou C, Bouvier G, Candeias C, Bouaboud A, Costa L, **BOULAY AC, COHEN-SALMON M**, Plu I, Rambaud C, Faurobert E, Albigès-Rizo C, Tazi A, Poyart C, Guignot J. CC17 group B *Streptococcus* exploits integrins for neonatal meningitis development. **J Clin Invest.** 2021 Mar 1;131(5):e136737.

This study identifies the brain endothelial integrin receptor of CC17 group B *Streptococcus* allowing its penetration in the newborn brain and leading to meningitis.

- 6) Bello C, Smail Y, Sainte-Rose V, Podglajen I, **GILBERT A\***, **MOREIRA V\***, Chrétien F, **COHEN SALMON M**, Tran Van Nhieu G. Role of astroglial Connexin 43 in pneumolysin cytotoxicity and during pneumococcal meningitis. **PLoS Pathog.** 2020 Dec 28;16(12):e1009152.

This study explores the role of the astroglial gap junction protein Connexin 43, a protein enriched in perivascular astrocytic processes, in bacterial meningitis caused by *Streptococcus pneumoniae*.

- 7) Belmaati Cherkaoui M, Vacca O, Izabelle C, **BOULAY AC**, Boulogne C, Gillet C, Barnier JV, Rendon A, **COHEN-SALMON M**, Vaillend C. Dp71 contribution to the molecular scaffold anchoring aquaporine-4 channels in brain macroglial cells. **Glia.** 2021 Apr;69(4):954-970.

This study demonstrates the role of the dystrophin isoform DP71 in the anchoring Aquaporin 4, the water channel present at the astrocytic membranes facing the brain vessels and suggests that intellectual disability in Duchenne muscular dystrophy is linked to gliovascular water homeostasis defects.

- 8) **COHEN-SALMON M, SLAOUI L, MAZARÉ N, GILBERT A\*, OUDART M, ALVEAR-PEREZ R, ELORZA-VIDAL X\***, CheverO, **BOULAY AC**. Astrocytes in the regulation of cerebrovascular functions. **Glia.** 2021 Apr;69(4):817-841. Review.

This review summarizes current knowledge on the role of astrocytes in the regulation of cerebrovascular functions.

- 9) **MAZARÉ N\*, OUDART M**, Moulard J, Cheung G, **TORTUYAUX R\***, Mailly P, Mazaud D, Bemelmans AP, **BOULAY AC**, Blugeon C, Jourdren L, Le Crom S, Rouach N, **COHEN-SALMON M**. Local Translation in Perisynaptic Astrocytic Processes Is Specific and Changes after Fear Conditioning. **Cell Rep.** 2020 Aug 25;32(8):108076.

This study shows that local translation occurs in perisynaptic astrocytic processes (PAPs). We characterize the repertoire of ribosome-bound mRNAs enriched in hippocampal PAPs and show that RNA distribution and translation change in PAPs after fear conditioning, indicating the role of astrocytic local translation in memory and learning.

- 10) **OUDART M, TORTUYAUX R\***, Mailly P, **MAZARÉ N\*, BOULAY AC, COHEN-SALMON M**. AstroDot - a new method for studying the spatial distribution of mRNA in astrocytes. **J Cell Sci.** 2020 Apr 8;133(7):jcs239756.

This article describes the development of experimental and analytical tools to characterize mRNA distribution in astrocytes and microglia in physiological or pathological settings. It shows that distribution of mRNA in astrocytes is modified in a mouse model of Alzheimer's disease and varies with the presence of amyloid deposits in the brain parenchyma.

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