



SAINTS-PÈRES
Paris Institute for
the Neurosciences

UMR-S 1124
Toxicology, Pharmacology and Cell Signaling

Saints Pères Neuroscience Seminar Series

Friday, September 24th, 2021 at 11:30

Salle des Conférences (R229)

Centre Universitaire des Saints-Pères

45 rue des Saints-Pères, 75006 Paris

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Modifying macrophages at the periphery has the capacity to modify microglial reactivity and to extend ALS mouse survival

Microglia, the macrophage of the CNS and peripheral macrophages, combined, have been implicated in Amyotrophic Lateral Sclerosis (ALS), the most common adult-onset motor neuron disease, but without discriminating their respective roles. Since we have shown that microglial cells participated to disease progression in mouse models, one focus of my team is to target microglial cells/ macrophages to be able to slow motor neuron degeneration and ALS disease progression. Motor neurons are specific neurons since their cell body is in the CNS and therefore surrounded by microglial cells while their axon, which extends at the periphery is surrounded by peripheral macrophages. Since microglial cells and peripheral macrophages have distinct developmental origins and are in specific environments, we hypothesized that their reaction to the disease could be different. Recently, we showed that macrophages along peripheral motor neuron axons of ALS mice and patients reacted to neurodegeneration and that, in ALS mice, peripheral myeloid cell infiltration into the spinal cord was limited and disease duration dependent. Transcriptomics analyses revealed that sciatic nerve macrophages and microglia reacted very different to neurodegeneration. Replacing macrophages at the periphery reduced both peripheral macrophage and microglial activation, delayed symptoms and increased ALS mouse survival. Thus, modifying macrophages at the periphery has the capacity to influence disease progression and is of therapeutic value for ALS.

Those interested in meeting with the speaker please contact
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