Session PSTR332 - Neuroinflammation: Animal Models

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PSTR332.24 / S10 - Persistent cognitive deficit despite prompt recovery of well-being behavior following LPS-related neuroinflammation in mice - sequential and temporal cytokine correlates

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Presenter at Poster
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S. WAGNER, C. DUCHEMIN-NEVEU, L. PETER, C. ALBAC, F. LAUGA, E. POIRAUD, B. HUYARD, C. PINAULT, *E. ANDRIAMBELOSON; NEUROFIT, ILLKIRCH, France. Persistent cognitive deficit despite prompt recovery of well-being behavior following LPS-related neuroinflammation in mice - sequential and temporal cytokine correlates. Program No. PSTR332.24. 2023 Neuroscience Meeting Planner. Washington, D.C.: Society for Neuroscience, 2023. Online.

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Disclosures
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Abstract
Neuroinflammation and the associated exaggerated or prolonged cytokine release is believed to be an underlying mechanism of sickness behaviors and cause neuropsychiatric or neurological conditions. In the present work, neuroinflammation was induced in mice by a single intraperitoneal injection of non-sepsis dose of Lipopolysaccharide (0.25 mg/kg LPS). The time course of IL-1β, TNF α and IL-17 expression was assessed in the brain hippocampus. In addition, sickness behaviors were investigated at different timepoints and includes changes in their 1) burrowing performance (surrogate measure of animal's well-being), 2) motor activity in the open-field and 3) spontaneous alternation in the T-maze (to evaluate the cognitive function). In LPS mice the results showed a hippocampal IL-1β release that sharply peaked at 4h and a returned to baseline level by 24h. By 48h (day 2) a raise in TNF α followed and lasted for only about a week. Increase in IL-17 occurred at later timepoints (day 5 post-LPS) but was sustained up to day 21 (3 weeks). Behavioral measures showed a dramatic reduction in burrowing performance of LPS-mice at 2hr post-LPS, followed by a fully recovery by 48h post-LPS. At this timepoint and up to 3 weeks post-LPS, no significant impairment of motor performance of LPS-mice was observed in the open-field. In contrast, cognitive function as assessed by the spontaneous alternation in the T-maze was significantly impaired in LPS-mice as early as day 1 and the deficit remained up to 3 weeks. The above-mentioned burrowing and cognitive deficits were sensitive to anti-inflammatory treatments. The above results highly suggest a sequentially and temporally orchestrated release of brain hippocampal cytokines following LPS administration in mice. These cytokines appear to play a critical role in the genesis and the perpetuation of sickness behavior in mice.