S21-01 - **ANIMAL AND HUMAN DATA OF MICROGLIAL ALTERATIONS IN SCHIZOPHRENIA (FOR SYMPOSIUM MÜLLER IMMUNE SYSTEM)**

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Introduction/objectives/aims: Inflammatory and immunological processes interfering with brain development are discussed as one cause of schizophrenia. Various signs of overactivation of the immune system were often found in this disease. Based on post-mortem analysis showing an increased number of activated microglial cells in patients with schizophrenia, it can be hypothesized that these cells contribute to disease pathogenesis and may actively be involved in grey matter loss observed in such patients.

Methods: In the present study, PolyI:C incubation of pregnant dams was used as animal model of schizophrenia, and the number and shape of microglia was assessed in the offspring in the early phase of this disease, using fluorescence immunostaining (Iba1).

Results: Descendants of mice exposed to PolyI:C at embryonic day 9 showed higher number of microglial cells in the hippocampus and striatum, but not in the frontal cortex at postnatal day 30, which is similarly to adolescence in man, as compared to those exposed to saline. Furthermore, offspring microglia from PolyI:C treated mothers were morphologically characterized by a reduced arborisation indicative for a status of higher activation compared to the offspring microglia from vehicle treated mice. Recent data concerning microglial alterations in anterior cingulate cortex of patients with schizophrenia, as assesed in post-mortem tissues, will also be presented.

Conclusions: The data support the hypothesis that maternal infection during embryogenesis contributes to microglial activation in the offspring, which may therefore represent a contributing factor to the pathogenesis of schizophrenia.

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