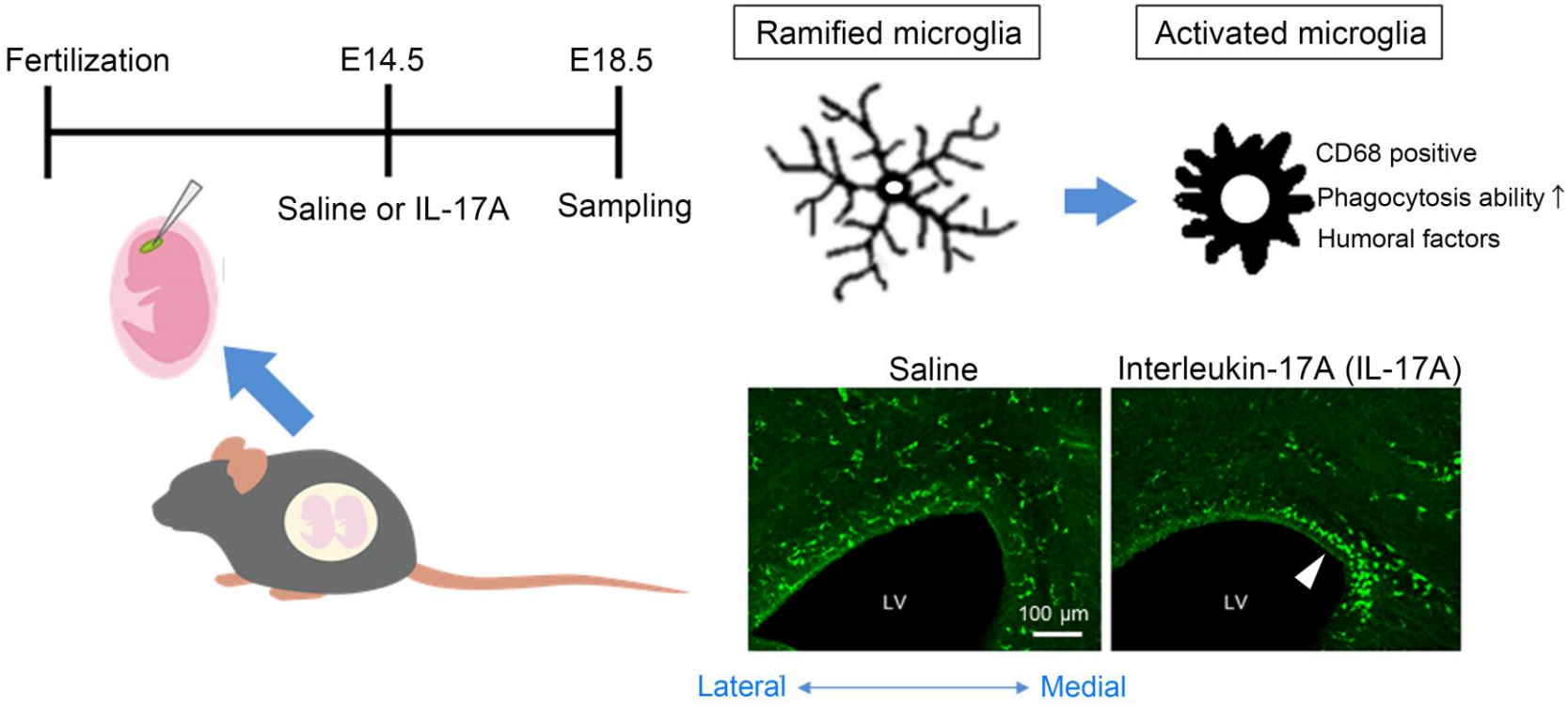
From Department of Anatomy and Neuroscience

Intraventricular IL-17A administration activates microglia and alters their localization in the mouse embryo cerebral cortex



Viral infection during pregnancy has been suggested to increase probability of autism spectrum disorder in offspring. This phenomenon has been modeled in rodents subjected to maternal immune activation (MIA). Previous studies showed that maternal T helper 17 cells and the effector cytokine interleukin-17A (IL-17A) play a central role in MIA-induced behavioral abnormalities and cortical dysgenesis called cortical patch in offspring. However, it is unclear how IL-17A acts on fetal brain cells to cause ASD pathologies. To assess the effect of IL-17A on cortical development, we performed direct administration of IL-17A into lateral ventricles of fetal mouse brain. We analyzed injected brain focusing on microglia, which express IL-17A receptors. We found that IL-17A activated microglia and altered their localization in the cerebral cortex. Our data suggest that IL-17A activates cortical microglia, which could lead to a series of ASD-related brain pathology, including excessive phagocytosis of neural progenitor cells in the ventricular zone.

References: Sasaki, Tome, Takei., Molecular Brain. 13:93. 2020.

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