

富士フイルム和光純薬株式会社

第2回日本がん神経科学研究会 ランチョンセミナー

日時

2026年10月10日(土) 12:15-13:15

会場

第2会場 (国立がん研究センター研究所 セミナールームA・B)

演題1

The synapse as a starting point: from brain development to cancer neuroscience

上阪 直史 教授 (東京科学大学 医歯学総合研究科 認知神経生物学分野)

The developing brain forms far more synapses than it keeps and circuits mature by removing the excess. We have identified the retrograde signals that instruct this choice. Sema3A and progranulin maintain synapses, Sema7A and BDNF removes them, and their balance determines which single input survives. In 2015 and 2017, neuronal activity was shown to promote glioma growth through secreted NLGN3, so the tumor was not autonomous but dependent on circuit activity. Glutamatergic synapses onto tumor cells were described soon after, making it a postsynaptic partner rather than a recipient of a factor. Tumor biology had arrived at the synapse. NLGN3, BDNF, progranulin, and semaphorins made the overlap literal. Each is a signal the developing brain uses to keep or discard a connection, and each drives glioma growth. But development teaches that refinement is never decided by one force, only by the balance between signals that maintain and signals that remove. Only the promoting side, the accelerator, had been examined. We asked whether the brain also has a brake for brain tumor. We show that it does. Inhibitory neurons form functional synapses onto glioma cells, and this input acts as a brake on tumor progression (Collaboration work of Uesaka lab, Maruyama lab, Kawauchi lab, Neuron, 2026). Here I will show the detail in this lunch session.

演題2

Marker Antibodies for Neuroscience from FUJIFILM Wako: Developed to Meet Researchers' Needs

平塚 大士 (富士フイルム和光純薬株式会社)