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A coupling between a neurotransmitter's metabolism and epigenetic regulations promotes intra-tumor heterogeneity in glioma

Cell populations with differing proliferative, stem-like and tumorigenic states co-exist in most tumors and especially malignant gliomas. Whether metabolic variations can drive this heterogeneity by controlling dynamic changes in cell states is unknown. We addressed this question by combining cell biology and neuropathological approaches. Metabolite profiling of human adult glioblastoma stem-like cells upon loss of their tumorigenicity, followed by genetic and pharmaceutical manipulations highlighted a novel signaling module that couples the catabolism of the GABA neurotransmitter and the formation of DNA epigenetic marks. This signaling module was efficient in adult glioblastoma cells with varying molecular profiles, along with cells from pediatric pontine gliomas. Importantly, we verified the relevance of all our findings in the context of the human pathology, using bioinformatics analyzes of patient-derived data at the tissue and single cell level, and immunohistochemical and metabolite analyzes of patients' tumor samples. These results highlight unexpected levels of heterogeneity among the tumor cells, and support an active participation of metabolic variations in the genesis of tumor heterogeneity.

Biography

Marie-Pierre Junier's interest in brain neoplasms developed as a natural extension of fundamental research studies aiming at deciphering the role of neuron-glia interactions during development and neurodegenerative diseases. Following studies of the role of troubles of glial cell differentiation in cancerous transformation, she created with Dr. H Chneiweiss the team "Glial plasticity and neuro-oncology" located in the Neuroscience laboratory of the Institute of Biology Paris Seine Institute (IBPS). The team project questions the dynamics of tumor cell functional heterogeneity, and its impact on malignant glioma growth.

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