Discovery of LAMP-2A as potential biomarkers for glioblastoma

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Video Byte

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Abstract

Glioblastoma is a devastatingly aggressive and prevalent primary brain tumor. Despite the discovery of many potential biomarkers and treatment targets, there has been little improvement in survival. One unexplored pathway in glioblastoma is chaperone-mediated autophagy (CMA), which has been implicated in a variety of human malignancies. A new paper examined CMA and its key component, lysosome-associated membrane protein type 2A (LAMP-2A), using clinical samples, in vitro experiments, and a mouse xenograft model. In clinical samples, glioblastoma showed elevated expression of LAMP-2A compared to peritumoral regions and low-grade glioma and an associated decrease in nuclear receptor co-repressor (N-CoR). Glioblastoma with high LAMP-2A expression also had inhibited unfolded protein response and apoptosis. In vitro, silencing LAMP-2A up-regulated N-CoR and activated the unfolded protein response pathway, which led to apoptosis. Mouse experiments confirmed that LAMP-2A inhibition arrested tumor development by promoting apoptosis. These results suggest that CMA plays a central role in protecting glioblastoma cells from apoptosis and that further research into selective modulators of LAMP-2A may provide novel therapeutic strategies.