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# Glioblastoma(GBM) affects approximately 10,000 new patients per year in the US, and is themostaggressiveprimarybraintumor. GBM has a dismal prognosis and improved therapeutic approaches are badly needed

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Evidencehasbeenaccumulatingoverrecentyearslinkingcytomegalovirus (CMV)tohumanGBM and other cancers. CMV DNA, RNA and protein have been detected in tumor samples by multiple independent research groups in the majority of patient GBM cases. In vitro, CMV infection of GBM stem-like cells enhances their self-renewal properties, and several CMV gene products have been shown to activate known oncogenicmechanisms in GBM includingthePDGFR/PI3K pathwayand also shown to inactivate GBM tumor suppressors such as RB.Through its interactions with the host immune system, CMV mayalso altertheGBMmicroenvironment to favor tumor growth. However, this has not been studied in detail and the role of CMV in GBM is still very poorly defined. Therefore, inordertoimproveourunderstanding ofthemechanisms underlyingthe link betweenCMVand GBMwehave developed a murinemodelofCMV-mediated glioma progression.Inthis model, perinatal murineCMV(MCMV)infection promotes tumor progression and significantly shortenssurvivalin the mut3 mouse(GFAP-cre; Nf1loxP/+; Trp53-/+)geneticmodelof malignant glioma. In the model MCMV is detectable in mouse brains in the first weeks after infection and also in tumors, which form after 18-20 weeks, where CMV expression is mainly detected in CD45+ lymphocytes. In the presence of MCMV, tumors show increased activation of STAT3, a known transcriptional driver of GBM growth and viral tumor-promoting effects can be reversed by a small molecule STAT3 inhibitor. These findings thus associate CMV infection to a STAT3-dependent modulatory role in gliomaformation/progression in the context of tumor suppressor mutations in mice and possibly in humans.

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