2-O-D2-1 Oral Sessions**CDK8 maintains stemness and tumorigenicity of glioma stem cells by controlling the c-MYC pathway**

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Glioblastoma (GBM), the most malignant type of primary brain tumor, has a very poor prognosis. Glioma stem cells (GSCs) play a key role in tumor initiation and progression. Cyclin-dependent kinase 8 (CDK8), which belongs to the transcription-related CDK family, is considered both an oncogene and a tumor suppressor. However, the functional role and underlying mechanisms of CDK8 expressed in GSCs on gliomagenesis is still poorly understood both *in vitro* and *in vivo*. Disruption of *CDK8* by shRNA resulted in an attenuation of the self-renewal potential and tumorigenicity of patient-derived GSCs, which can be significantly rescued by the overexpression of *MYC*, a stem cell transcription factor. Moreover, the pharmacological inhibition by CDK8 inhibitor significantly repressed the self-renewal potential and tumorigenicity of GSCs. Bioinformatics analyses have revealed that *CDK8* expression was significantly higher in human GBM tissues compared with normal brain tissues, and its expression was positively correlated with stem cell markers including *MYC* and *SOX2* in human GBM specimens. Additionally, *CDK8* expression is associated with poor survival in GBM patients. These findings highlight the importance of the CDK8-c-MYC axis in maintaining stemness and tumorigenicity in GSCs, indicating that targeting GSCs through CDK8 inhibition could be a promising strategy against GBM.