lncRNAs ZFAS1 and MALAT1 were significantly upregulated ($p < 0.05$), whereas lncRNAs LINC00261 and LINC01619 were significantly downregulated in SAH patients with CVS ($p < 0.05$) compared to SAH patients without CVS. Pan et al. applied this lncRNA signature to retrospectively predict CVS in SAH patients (n = 38 for SAH patients without CVS, and n = 27 for SAH patients with CVS). The 4-lncRNA signature was found to be predictive in >40% of samples and the 2-lncRNA comprising MALAT1 and LINC01619 accurately predicted CVS in ~90% cases. These results are initial steps toward personalized management of SAH patients in clinics and provide novel CSF biomarkers that can substantially improve the clinical management of SAH patients [1].

The function of Long non-coding RNA (lncRNA) ZFAS1 in glioma is still unclear. In a study Gao et al., found that ZFAS1 was upregulated in glioma tissues and cell lines. High ZFAS1 expression in glioma tissues was significantly correlated with advanced tumor stage and poor overall survival. Furthermore, in vitro assays demonstrated that ZFAS1 inhibition significantly suppressed glioma cell proliferation, migration and invasion. Importantly, they further confirmed that epithelial-mesenchymal transition (EMT) and the Notch signaling pathway was inactivated in the glioma cells after ZFAS1 knockdown. Thus, the findings indicated that ZFAS1 could exhibit a tumor oncogenic role in glioma progression by regulating EMT and Notch signaling pathway. LncRNA ZFAS1 might serve as a therapeutic target for the treatment of glioma patients [2].

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2)
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