Hyaluronic acid (HA), is an important component of the extracellular matrix in the tumor microenvironment, which abnormally accumulates in a variety of tumors. However, the role of abnormal HA accumulation in glioma remains unclear. A study indicated that HA, hyaluronic acid synthase 3 (HAS3), and a receptor of HA named CD44 were expressed at high levels in human glioma tissues and negatively correlated with the prognosis of patients with glioma. Silencing HAS3 expression or blocking CD44 inhibited glioma cell proliferation in vitro and in vivo. The underlying mechanism was attributed to the inhibition of autophagy flux and maintaining glioma cell cycle arrest in the G1 phase. More importantly, 4-methylumbelliferone (4-MU), a small competitive inhibitor of Uridine diphosphate (UDP) with the ability to penetrate the blood-brain barrier (BBB), also inhibited glioma cell proliferation in vitro and in vivo. Thus, approaches that interfere with HA metabolism by altering the expression of HAS3 and CD44 and the administration of 4-MU potentially represent effective strategies for glioma treatment [1].

1)
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