CXCL5 (MIM *600324 a.k.a. epithelial neutrophil-activating peptide 78 or ENA-78) is a C-X-C chemokine that attracts and activates neutrophils. Furthermore, CXCL5 expression has been shown to be highly inducible in endothelial and vascular smooth muscle cells by IL-1β

CXCL5 and its receptor CXCR2 have been found to be involved in tumorigenesis and cancer progression. Recent studies have shown that CXCR2 is upregulated in glioma tissues, and associated with poor prognosis and recurrence. However, the role of CXCL5/CXCR2 signaling in mediating the malignant phenotypes of glioma cells, as well as the underlying mechanism, still remains unclear. In the present study, we found that CXCL5 was upregulated in glioma tissues compared to that noted in normal brain tissues. High CXCL5 levels were significantly associated with higher tumor grade, advanced clinical stage, and shorter survival time of glioma patients. In vitro studies indicated that the protein expression levels of CXCL5 and CXCR2 were markedly higher in human glioma cell lines (U87, U251, U373 and A172), when compared with those in normal human gliocyte HEB cells. Overexpression of CXLC5 significantly promoted the proliferation and migration of U87 cells, while knockdown of CXCL5 by small interfering RNA markedly inhibited U87 cell proliferation and migration. Moreover, both exogenous CXCL5 treatment and the conditioned medium of CXCL5-overexpressing HEB cells also enhanced the proliferation and migration of U87 cells. Molecular mechanism investigation revealed that CXLC5 activated the ERK, JNK, p38 MAPK signaling pathways, which play key roles in tumor growth and metastases. According to these data, our study suggests that CXCL5 plays a promoting role in glioma in autocrine- and paracrine-dependent manners [1].

1)
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