CXCR2

CXCR2 (C-X-C Motif Chemokine Receptor 2) is a Protein Coding gene. Diseases associated with CXCR2 include Whim Syndrome 2 and Autosomal Recessive Severe Congenital Neutropenia Due To Cxcr2 Deficiency. Among its related pathways are the CCR5 Pathway in Macrophages and Chemokine Superfamily: Human/Mouse Ligand-Receptor Interactions. Gene Ontology (GO) annotations related to this gene include G protein-coupled receptor activity and C-X-C chemokine receptor activity. An important paralog of this gene is CXCR1.

CXCR2 is transiently overexpressed in the peripheral monocytes of TBI patients post-surgery, and drives peripheral monocyte chemotaxis toward CSF and monocyte-mediated ICD of nerve cells. Therefore, CXCR2 may be a target for monocyte-based therapies for TBI [1)].

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 a study, Dai et al., 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 metastasis. According to these data, our study suggests that CXCL5 plays a promoting role in glioma in autocrine- and paracrine-dependent manners [2)].

1)
Wang H, Huang Q, Zhang Z, Ji J, Sun T, Wang D. Transient post-operative overexpression of CXCR2 on monocytes of traumatic brain injury patients drives monocyte chemotaxis toward cerebrospinal fluid and enhances monocyte-mediated immunogenic cell death of neurons in vitro. J Neuroinflammation. 2022 Jun 29;19(1):171. doi: 10.1186/s12974-022-02535-6. PMID: 35768823.
2)
Dai Z, Wu J, Chen F, Cheng Q, Zhang M, Wang Y, Guo Y, Song T. CXCL5 promotes the proliferation and migration of glioma cells in autocrine- and paracrine-dependent manners. Oncol Rep. 2016 Oct 10. doi: 10.3892/or.2016.5155. PubMed PMID: 27748886.