

Cetuximab

Cetuximab is an [epidermal growth factor receptor](#) (EGFR) inhibitor used for the treatment of metastatic colorectal cancer, metastatic non-small cell lung cancer and head and neck cancer. Cetuximab is a chimeric (mouse/human) monoclonal antibody given by intravenous infusion that is distributed under the trade name Erbitux in the U.S. and Canada by the drug company Bristol-Myers Squibb and outside the U.S. and Canada by the drug company Merck KGaA. In Japan, Merck KGaA, Bristol-Myers Squibb and Eli Lilly have a co-distribution.

In July 2009, the FDA approved cetuximab (Erbitux) for treatment of colon cancer with wild-type KRAS, since it had little or no effect in colorectal tumors harboring a KRAS mutation (this also applied to the EGFR antibody panitumumab). This was the first genetic test to guide treatment of cancer.

In July 2012, the FDA approved a real-time PCR companion diagnostic test for KRAS, the therascreen KRAS test.

Eller et al. demonstrated that cetuximab alone was effective against EGFR-amplified glioblastoma multiforme (GBM) cells in vivo and in vitro ¹⁾.

Systemic administration of recombinant monoclonals, including bevacizumab directed against angiogenesis and cetuximab directed against proliferation have demonstrated reduced GBM growth and increased survival in animal models, but the systemic administration of these monoclonals have not increased overall life expectancy in clinical trials ^{2) 3) 4) 5)}.

Cetuximab significantly increased [neurogenesis](#) in the lesion site. Meanwhile, implanting cetuximab modified linear ordered collagen scaffolds (LOCS) into SCI lesion sites in dogs resulted in neuronal regeneration, including neuronal differentiation, maturation, myelination, and synapse formation. The neuronal regeneration eventually led to a significant locomotion recovery. Furthermore, LOCS implantation could also greatly decrease chondroitin sulfate proteoglycan (CSPG) deposition at the lesion site. These findings suggest that endogenous neurogenesis following acute complete SCI is achievable in species ranging from rodents to large animals via functional scaffold implantation. LOCS-based Cetuximab delivery system has a promising therapeutic effect on activating endogenous neurogenesis, reducing CSPGs deposition and improving motor function recovery ⁶⁾.

¹⁾

Eller JL, Longo SL, Kyle MM, Bassano D, Hicklin DJ, Canute GW. Anti-epidermal growth factor receptor monoclonal antibody cetuximab augments radiation effects in glioblastoma multiforme in vitro and in vivo. *Neurosurgery*. 2005;56(1):155-62; discussion 162. PubMed PMID: 15617598.

²⁾

Fu P, He YS, Huang Q, Ding T, Cen YC, Zhao HY, et al. Bevacizumab treatment for newly diagnosed glioblastoma: Systematic review and meta-analysis of clinical trials. *Mol Clin Oncol*. 2016;4(5):833-8. doi: 10.3892/mco.2016.816

³⁾

Khasraw M, Ameratunga M, Grommes C. Bevacizumab for the treatment of high-grade glioma: an update after phase III trials. *Expert Opin Biol Ther*. 2014;14(5):729-40. doi: 10.1517/14712598.2014.898060

⁴⁾

Niyazi M, Harter PN, Hattingen E, Rottler M, von Baumgarten L, Proescholdt M, et al. Bevacizumab and radiotherapy for the treatment of glioblastoma: brothers in arms or unholy alliance? *Oncotarget*. 2016;7(3):2313–28. doi: 10.18632/oncotarget.6320

⁵⁾

Staedtke V, Bai RY, Lattera J. Investigational new drugs for brain cancer. *Expert Opin Investig Drugs*. 2016:1–20.

⁶⁾

Li X, Zhao Y, Cheng S, Han S, Shu M, Chen B, Chen X, Tang F, Wang N, Tu Y, Wang B, Xiao Z, Zhang S, Dai J. Cetuximab modified collagen scaffold directs neurogenesis of injury-activated endogenous neural stem cells for acute spinal cord injury repair. *Biomaterials*. 2017 May 18;137:73-86. doi: 10.1016/j.biomaterials.2017.05.027. [Epub ahead of print] PubMed PMID: 28544974.

From:

<https://neurosurgerywiki.com/wiki/> - **Neurosurgery Wiki**

Permanent link:

<https://neurosurgerywiki.com/wiki/doku.php?id=cetuximab>

Last update: **2024/06/07 02:56**

