CD24

Signal transducer CD24 also known as cluster of differentiation 24 or heat-stable antigen CD24 (HSA) is a protein that in humans is encoded by the CD24 gene.

CD24 is a cell adhesion molecule.

CD24 is a sialoglycoprotein expressed at the surface of most B lymphocytes and differentiating neuroblasts. It is also expressed on neutrophils and neutrophil precursors from the myelocyte stage onwards. The encoded protein is anchored via a glycosylphosphatidylinositol (GPI) link to the cell surface. The protein also contributes to a wide range of downstream signaling networks and is crucial for neural development.[5] Cross-linking of CD24 on the surface of neutrophils induces apoptosis,[6] and this appears to be defective in sepsis.[6] CD24 gene is found on chromosome 6 (6q21) An alignment of this gene's sequence finds genomic locations with similarity on chromosomes 1p36, 3p26, 15q21.3, 20q11.2, and Yq11.222. Whether transcription and corresponding translation, occurs at each of these other genomic locations needs to be experimentally determined [1].

CD24 is specifically upregulated and apparently associated with better survival. CD24 and Nestin expression respond differently to alteration of D-2-Hydroxyglutarate levels. CD24 upregulation is associated with histone and DNA demethylation as opposed to hypermethylation in the downregulated genes.

The Neural Stem Cell Marker CD24 has specifically upregulation in IDH-mutant Glioma [2].

On human medulloblastoma, CD24 was found to be highly expressed on Group 3, Group 4 and SHH subgroups compared with the WNT subgroup, which was predominantly positive for CD15, suggesting CD24 is an important marker of non-WNT medulloblastoma initiating cells and a potential therapeutic target in human medulloblastoma.

A study reports the use of CD24 and CD15 to isolate a GCP-like TIC population in Ptch1 deleted medulloblastoma and suggests CD24 expression as a marker to help stratify human WNT tumours from other medulloblastoma subgroups [3].

In the adult rodents' brain, CD24 expression is restricted to immature neurons located in the neurogenesis areas.

Previous studies have confirmed that CD24 expression could be markedly elevated in the cerebral cortex after traumatic brain injury (TBI) both in humans and in mice. Although there is a close relationship between CD24 and neurogenesis, it remains unknown about the specific role of CD24 in neurogenesis areas after TBI. Here, the expression of CD24 was detected in the ipsilateral hippocampus by the Western blotting and real-time quantitative polymerase chain reaction. RNA interference was applied to investigate the effects of CD24 on post-traumatic neurogenesis. Brain sections were labeled with CD24 and doublecortin (DCX) via immunofluorescence. The Morris water maze test was used to assess cognitive functions. The results indicated that both mRNA and protein

levels of CD24 were markedly elevated in the hippocampus after TBI. Meanwhile, TBI could cause a decrease of DCX-positive cells in the dentate gyrus of the hippocampus. Downregulation of CD24 significantly inhibited the phosphorylation of Src homology region 2-containing protein tyrosine phosphatase 2 in the ipsilateral hippocampus. Meanwhile, inhibition of CD24 could reduce the number of DCX-positive cells in the dentate gyrus area and impair cognitive functions of the TBI mice. These data suggested that hippocampal expression of CD24 might positively regulate neurogenesis and improve cognitive functions after TBI [4].

References

1)
https://en.wikipedia.org/wiki/CD24
2)
Tiburcio PDB, Locke MC, Bhaskara S, Chandrasekharan MB, Huang LE. The neural stem-cell marker CD24 is specifically upregulated in IDH-mutant glioma [published online ahead of print, 2020 Jul 1]. Transl Oncol. 2020;13(10):100819. doi:10.1016/j.tranon.2020.100819
3)
Robson JP, Remke M, Kool M, et al. Identification of CD24 as a marker of Patched1 deleted medulloblastoma-initiating neural progenitor cells. PLoS One. 2019;14(1):e0210665. Published 2019 Jan 18. doi:10.1371/journal.pone.0210665
4)
Wang H, Zhou XM, Xu WD, et al. Inhibition of Elevated Hippocampal CD24 Reduces Neurogenesis in Mice With Traumatic Brain Injury. J Surg Res. 2020;245:321-329. doi:10.1016/j.jss.2019.07.082