HoxB13

HoxB13 (Homeobox B13) is a gene that belongs to the Hox gene family. The Hox genes are a group of highly conserved genes that play a crucial role in the development of body structures along the anterior-posterior (head to tail) axis during embryogenesis. These genes help determine the identity and positioning of different body parts, such as limbs, organs, and segments.

HoxB13 specifically falls within the HoxB cluster, which is located on chromosome 17 in humans. This cluster contains several genes that are responsible for the development of structures in the lower spine, pelvis, and urogenital system.

Mutations or dysregulation of HoxB13 have been associated with various developmental and diseaserelated conditions. In particular, mutations in HoxB13 have been linked to prostate cancer susceptibility in men. Certain variants of the HoxB13 gene are associated with an increased risk of developing prostate cancer. Researchers have been studying the role of HoxB13 in prostate cancer development and progression to better understand its implications.

HoxB13 in Neurosurgery

- Single-cell RNA sequencing analysis of human embryos from the late Carnegie to fetal development
- Immunoreactivity of HOXB13 in Neuroendocrine Neoplasms Is a Sensitive and Specific Marker of Rectal Well-Differentiated Neuroendocrine Tumors
- HOXB13 promotes proliferation, migration, and invasion of glioblastoma through transcriptional upregulation of IncRNA HOXC-AS3
- Long noncoding RNA HOXB13-AS1 regulates HOXB13 gene methylation by interacting with EZH2 in glioma
- The Similarities and Differences between Intracranial and Spinal Ependymomas : A Review from a Genetic Research Perspective
- The Molecular Feature of HOX Gene Family in the Intramedullary Spinal Tumors
- Spinal cord ependymomas and myxopapillary ependymomas in the first 2 decades of life: a clinicopathological and immunohistochemical characterization of 19 cases

Soukup et al. performed HoxB13 immunohistochemistry in tissue microarrays and the whole sections of 232 neuroendocrine neoplasms. These included 34 paragangliomas (PGs), 20 cauda equina neuroendocrine tumors (CENETs), 123 well-differentiated neuroendocrine tumors (WDNETs), and 55 neuroendocrine carcinomas (NECs). WDNETs were additionally analyzed with SATB2 and colorectal WDNETs with CDX2 and serotonin immunohistochemistry. In total, HoxB13 immunoreactivity was observed in 95% (19/20) CENETs, 10.6% (13/123) WDNETs, and 12.9% (7/54) NECs. No PGs were positive. Large intestine WDNETs expressed HoxB13 in 68.4% (13/19); five negative tumors originated in the cecum and one in the rectum. In rectum, 92.9% (13/14) WDNETs expressed HoxB13. HoxB13 was 92.9% sensitive and 100% specific, showing a 100% positive predictive value for the rectal origin of WDNET. In NECs, HoxB13 was positive in 15.4% (2/13) GIT tumors and 80% (4/5) prostatic NECs, but in none of the urinary bladder NECs (0/8). SATB2 was positive in 17.1% (21/123) WDNETs, including 78.9% (15/19) of colorectal WDNETs, 71.4% (5/7) appendiceal WDNETs, and 2.9% (1/34) small intestine WDNETs. All 4 SATB2-negative large bowel tumors originated in the cecum. When both markers combined, HoxB13+/SATB2+ immune profile was seen exclusively in rectal WDNETs (positive of the

appendiceal origin (positive predictive value 71.4%). Therefore, HoxB13 can be useful as an immunohistochemical marker of rectal WDNETs and prostatic NECs ¹⁾.

A study explored the role of HOXB13 in glioblastoma (GBM). Through microarray and immunohistochemistry analyses, HOXB13 was highly expressed in GBM tissues. Furthermore, they showed that high-level expression of HOXB13 in GBM was associated with worse survival, suggesting that HOXB13 could be a prognostic marker for patients with GBM. GBM cells U87 and U251 overexpressing HOXB13 showed enhanced proliferation, migration, and invasion relative to the control cells, while the knockdown of HOXB13 led to decreased cell proliferation, migration, and invasion abilities. In addition, dual-luciferase report assay, chromatin immunoprecipitation assay, and quantitative real-time polymerase chain reaction data showed that HOXB13 is directly bound to the HOXC-AS3 promoter. HOXC-AS3 was involved in HOXB13-induced proliferation, migration, and invasion of GBM cells. In summary, this study revealed the prognostic potential of HOXB13 in GBM. They believed that HOXB13/HOXC-AS3 signaling axis can be served as a therapeutic target for this highly aggressive cancer².

In a review, Lee et al. describe the genetic differences between spinal ependymomas and their intracranial counterparts to better understand their prognosis. From the literature review, many studies have reported that spinal cord ependymoma might be associated with NF2 mutation, NEFL overexpression, Merlin loss, and 9q gain. In myxopapillary ependymoma, NEFL and HOXB13 overexpression were reported to be associated. Prior studies have identified HIC-1 methylation, 4.1B deletion, and 4.1R loss as common features in intracranial ependymoma. Supratentorial ependymoma is usually characterized by NOTCH-1 mutation and p75 expression. TNC mutation, no hypermethylation of RASSF1A, and GFAP/NeuN expression may be diagnostic clues of posterior fossa ependymoma. Although MEN1, TP53, and PTEN mutations are rarely reported in ependymoma has been found to be quite different from intracranial ependymoma in genetic studies, and the favorable prognosis in spinal ependymoma may be the result of the genetic differences. A more detailed understanding of these various genetic aberrations may enable the identification of more specific prognostic markers as well as the development of customized targeted therapies ³¹.

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