NFYB

NFYB, also known as Nuclear Transcription Factor Y Subunit Beta, is a gene that encodes a protein subunit of the Nuclear Factor Y (NF-Y) transcription factor complex. The NF-Y complex is a key transcription factor that plays a critical role in the regulation of gene expression in eukaryotic cells.

The NF-Y complex is composed of three subunits: NFYA (NF-Y subunit A), NFYB (NF-Y subunit B), and NFYC (NF-Y subunit C). Together, these subunits form a trimeric complex that binds to specific DNA sequences in the promoter regions of target genes. This binding serves to activate or repress the transcription of those genes, depending on the context and the presence of other regulatory factors.

NFYB, as part of the NF-Y complex, contributes to the stability and DNA-binding capacity of the complex. The NF-Y complex is involved in the transcriptional regulation of a wide range of genes that are crucial for various cellular processes, including cell cycle control, DNA replication, differentiation, and responses to stress and DNA damage.

Research has shown that NFYB and the NF-Y complex can be associated with various physiological and pathological processes, including cancer. Alterations in NFYB expression or mutations can impact gene regulation and contribute to disease development.

It's worth noting that NFYB is just one component of the NF-Y complex, and the overall function of NF-Y is dependent on the coordinated activity of all three subunits. The NF-Y complex is an important transcriptional regulator with diverse roles in cellular biology, and ongoing research continues to uncover its functions and regulatory mechanisms in different contexts.

Zhang et al.investigated potential mechanisms underlying temozolomide resistance and glycolysis in GBM cells through regulation by nuclear transcription factor Y subunit β (NFYB) of the oncogene serine hydroxymethyltransferase 2 (SHMT2). GBM U251 cells were transfected with NFYB-, SHMT2-, and the potential NFYB target histone deacetylase 5 (HDAC5)-related vectors. Glucose uptake and lactate production were measured with detection kits. CCK-8/colony formation, scratch, Transwell, and flow cytometry assays were performed to detect cell proliferation, migration, invasion, and apoptosis, respectively. The binding of NFYB to the HDAC5 promoter and the regulation of NFYB on HDAC5 promoter activity were detected with chromatin immunoprecipitation and dual-luciferase reporter assays, respectively. NFYB and HDAC5 were poorly expressed and SHMT2 was expressed at high levels in GBM U251 cells. NFYB overexpression or SHMT2 knockdown decreased glucose uptake, lactate production, proliferation, migration, and invasion and increased apoptosis and TMZ sensitivity of the cells. NFYB activated HDAC5 to inhibit SHMT2 expression. SHMT2 overexpression nullified the inhibitory effects of NFYB overexpression on glycolysis and TMZ resistance. Thus, NFYB may reduce tumorigenicity and TMZ resistance of GBM through effects on the HDAC5/SHMT2 axis [1)]

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Zhang Y, Huang H, Liu P, Xie Y. NFYB increases chemosensitivity in glioblastoma by promoting HDAC5-mediated transcriptional inhibition of SHMT2. J Neuropathol Exp Neurol. 2023 Sep 23:nlad073. doi: 10.1093/jnen/nlad073. Epub ahead of print. PMID: 37742129.