Retinol dehydrogenase 10

Retinol dehydrogenase 10 (RDH10) is an enzyme that plays a critical role in the metabolism of vitamin A (retinol) within the body. It is part of the short-chain dehydrogenase/reductase (SDR) family of enzymes. RDH10 is specifically involved in the oxidation of retinol to retinal, a key step in the biosynthesis of retinoic acid. Retinoic acid is an important signaling molecule that regulates gene expression and is essential for various physiological processes, including embryonic development, cell differentiation, and maintenance of epithelial tissues.

Functions of RDH10 Vitamin A Metabolism: RDH10 catalyzes the conversion of all-trans-retinol (vitamin A alcohol) to all-trans-retinal, which is subsequently converted to all-trans-retinoic acid by retinaldehyde dehydrogenases. Retinoic acid acts as a potent transcriptional regulator, influencing the expression of genes involved in growth and differentiation.

Developmental Roles: RDH10 is essential during embryonic development. It is involved in the formation of the heart, central nervous system, and limbs, among other structures. Mutations in the RDH10 gene can lead to severe developmental defects due to impaired retinoic acid signaling.

Regulation of Cellular Processes: Beyond its role in development, retinoic acid produced via RDH10 activity regulates various cellular processes, such as cell growth, differentiation, and apoptosis. These processes are critical for maintaining tissue homeostasis and function.

Clinical Relevance Genetic Mutations: Mutations in the RDH10 gene can cause congenital disorders characterized by developmental abnormalities. For example, defects in RDH10 have been associated with congenital heart defects, limb malformations, and other developmental anomalies due to disrupted retinoic acid signaling.

Disease Associations: Aberrant RDH10 function or expression has been implicated in diseases related to vitamin A deficiency or imbalance. Given its role in retinoid metabolism, RDH10 is also being studied in the context of diseases like cancer, where retinoic acid signaling is often dysregulated.

Summary RDH10 is a vital enzyme in the vitamin A metabolic pathway, specifically involved in the conversion of retinol to retinal, a precursor of retinoic acid. This enzyme's activity is crucial for normal embryonic development and the regulation of various cellular processes throughout life. Its significance extends to clinical contexts, where mutations or dysfunctions in RDH10 can result in developmental disorders and contribute to disease pathogenesis.

There has been research showing that Retinol Dehydrogenase 10 (RDH10) may be a tumor promoting factor in brain glioma, but the biological effects of RDH10 remain undefined in Spinal cord glioma.

Zhao et al. performed gene set enrichment analysis (GSEA) and unsupervised clustering analysis to investigate the roles of EMT (epithelial-mesenchymal transition) in glioma. DEG (differently expressed gene) screening and correlation analysis were conducted to filter the candidate genes which were closely associated with EMT process in SCG. Enrichment analysis and GSVA (Gene Set Variation Analysis) were conducted to investigate the potential mechanism of RDH10 for SCG. Trans-well and healing assay were performed to explore the role of RDH10 in the invasion of SCG. Western blotting was performed to evaluate the levels of markers in PI3K-AKT and EMT pathway. In vivo tests were conducted to verify the role of RDH10 in EMT process. Results: Bioinformatic analysis demonstrated

the EMT pathway was associated with dismal prognosis of glioma. Further analysis demonstrated that RDH10 showed the strongest correlation with the EMT process. Retinol Dehydrogenase 10 expression was significantly increased in SCG tissues, correlating with advanced tumor grade and unfavorable prognosis. Functional analysis indicated that decreasing RDH10 levels impeded the invasive and migratory abilities of SCG cells, whereas increasing RDH10 levels augmented them. Enrichment analysis and western blot revealed that RDH10 regulated EMT process of SCG by PI3K-AKT pathway. They observed that the enhanced invasion ability and increased EMT-related protein induced by RDH10 overexpression can be suppressed by PI3K-AKT pathway inhibitor (LY294002). The research found that RDH10 was an effective biomarker associated with tumor grade and prognosis of SCG. RDH10 could regulate EMT process of SCG through PI3K-AKT pathway [1)].

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Zhao Z, Song Z, Wang Z, Zhang F, Ding Z, Zhao Z, Liu L, Fan T. Retinol dehydrogenase 10 promotes epithelial-mesenchymal transition in spinal cord gliomas via PI3K/AKT pathway. Int J Immunopathol Pharmacol. 2024 Jan-Dec;38:3946320241276336. doi: 10.1177/03946320241276336. PMID: 39180753.