BCL2A1

- RETRACTION: lncRNA PANTR1 Upregulates BCL2A1 Expression to Promote Tumorigenesis and Warburg Effect of Hepatocellular Carcinoma through Restraining miR-587
- Ketone body 3-hydroxybutyrate mitigates apoptosis and enhances osteogenesis in bone organoid construction via the cAMP/PKA/CREB signaling pathway
- BCL2A1- and G0S2-driven neutrophil extracellular traps: A protective mechanism linking preeclampsia to reduced breast cancer risk
- A common ground: an in silico assessment of the sources of intrinsic ex vivo resistance to venetoclax in acute myeloid leukemia
- Early Neutrophil Activation in Psoriatic Skin at Relapse Following Dead Sea Climatotherapy
- Gene expression analysis of Schizophrenia
- Identification of four key genes related to the diagnosis of chronic obstructive pulmonary disease using bioinformatics analysis
- A CEBPB/IL-1β/TNF-α Feedback Loop Drives Drug Resistance to Venetoclax and MDM2 Inhibitors in Monocytic Leukemia

BCL2A1, also known as B-cell lymphoma 2-related protein A1, is a gene that encodes a protein involved in the regulation of apoptosis. It is a member of the BCL-2 family of proteins, which includes various proteins that either promote or inhibit apoptosis.

Key points

Anti-Apoptotic Function: BCL2A1 is classified as an anti-apoptotic protein because it helps protect cells from undergoing apoptosis. Apoptosis is a tightly regulated process of programmed cell death that plays a crucial role in various physiological processes, including tissue development and the removal of damaged or unnecessary cells.

Cell Survival: BCL2A1 helps promote cell survival by inhibiting the activation of pro-apoptotic proteins and preventing the release of cytochrome c from mitochondria, a key event in the apoptotic pathway. This action helps maintain cell viability.

Expression in Different Tissues: BCL2A1 is expressed in a variety of tissues and cell types. Its expression can be induced in response to various cellular stresses, such as exposure to toxins, hypoxia, or growth factor withdrawal, which can trigger the need for increased anti-apoptotic protection.

Role in Disease: Dysregulation of BCL2A1 and other members of the BCL-2 family can have implications in various diseases, including cancer. Some cancers overexpress anti-apoptotic proteins like BCL2A1, which can contribute to cancer cell survival and resistance to chemotherapy.

Research Significance: Understanding the functions of BCL2A1 and related proteins is important in the fields of cell biology, oncology, and drug development. Researchers are studying these proteins to identify potential therapeutic targets for diseases characterized by dysregulated apoptosis.

BCL2A1 is one of several anti-apoptotic proteins within the BCL-2 family that help regulate the balance between cell survival and apoptosis. Balancing the actions of pro-apoptotic and anti-apoptotic proteins is crucial for normal cell homeostasis and for the development of therapeutic strategies in

various diseases, including cancer.

Previous studies have shown that BCL2A1 is closely related to tumorigenesis and resistance to chemotherapy of multiple solid tumors, such as breast cancer.

The expression pattern and potential biological function of BCL2A1 in glioma remain unknown. For the first time, Gao et al. found that the expression of BCL2A1 was higher in human glioma tissues than in normal brain tissues (NBTs) in both public datasets and in-house cohort. High BCL2A1 expression was associated with advanced WHO grade, IDH 1/2 wild type, and the mesenchymal (ME) subtype, and its overexpression in glioma predicted resistance to temozolomide (TMZ) chemotherapy and unfavorable prognosis. In addition, Gene set enrichment analysis (GSEA), Gene Ontology (GO), and Kyoto Encyclopedia of Genes and Genomes (KEGG) analysis indicated that BCL2A1 was significantly correlated with the immune response and immune-related pathways, and BCL2A1 expression was positively correlated with microenvironmental parameters (immune, stromal, and ESTIMATE scores) and macrophage infiltration. Interestingly, bioinformatic prediction and immunohistochemical/immunofluorescence staining analysis revealed that BCL2A1 expression was obviously associated with the tumor-associated macrophages (TAMs) markers CD68 and CCL2. Notably, the knockdown of BCL2A1 significantly inhibited cell proliferation of U87 and U251 in vitro, induced smaller tumor size, and prolonged the survival time of mice in vivo. Co-culture experiments of macrophages and GBM cells showed that BCL2A1 knockdown inhibited macrophage migration. Meanwhile, the knockdown of BCL2A1 was associated with low expression of CD68 and CCL2 in an intracranial xenograft model. This may suggest that BCL2A1 promotes the glioma progression and influences the prognosis of patients by participating in TAM infiltration. In conclusion, these findings suggest that BCL2A1 could serve as a promising prognostic indicator and immunotherapy target in gliomas [1)]

The study by Gao et al. provides valuable insights into the role of BCL2A1 in glioma, particularly its association with clinical parameters, immune responses, and TAM infiltration. These findings suggest that BCL2A1 may have clinical significance and could be a promising prognostic indicator and immunotherapy target. However, further research is needed to fully understand the mechanisms underlying BCL2A1's effects and to translate these findings into clinical practice.

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Gao L, Ye Z, Peng S, Lei P, Song P, Li Z, Zhou L, Hua Q, Cheng L, Wei H, Liu J, Cai Q. BCL2A1 is associated with tumor-associated macrophages and unfavorable prognosis in human gliomas. Aging (Albany NY). 2023 Oct 25;15. doi: 10.18632/aging.205149. Epub ahead of print. PMID: 37889551.