DNM1L

DNM1L, also known as Dynamin-1-like protein (DNM1L) or Drp1 (Dynamin-related protein 1), is a protein involved in mitochondrial dynamics within cells. It plays a crucial role in regulating the fission or division of mitochondria, which are the energy-producing organelles in eukaryotic cells. Here are some key points about DNM1L:

Mitochondrial Fission: DNM1L is primarily associated with mitochondrial fission, the process by which a single mitochondrion divides into two or more smaller mitochondria. This process is essential for maintaining mitochondrial health, distributing mitochondria within cells, and responding to changes in energy demands or cellular stress.

Structure: DNM1L is a GTPase protein, meaning it can bind and hydrolyze GTP (guanosine triphosphate). It forms a ring-like structure around the mitochondrial membrane, where it pinches and constricts the membrane to initiate fission.

Regulation: The activity of DNM1L is tightly regulated within the cell. Various signaling pathways and post-translational modifications, such as phosphorylation, control its recruitment to mitochondria and its ability to promote fission.

Mitochondrial Health: Proper regulation of mitochondrial fission and fusion (the process by which mitochondria fusr) is essential for maintaining mitochondrial health. DNM1L-mediated fission allows the removal of damaged or dysfunctional parts of mitochondria and the segregation of healthy mitochondria.

Cellular Functions: Mitochondrial dynamics, including fission and fusion, are critical for various cellular processes, including energy production, apoptosis (programmed cell death), and response to cellular stress.

Role in Disease: Dysregulation of DNM1L and mitochondrial dynamics has been linked to various diseases, including neurodegenerative disorders, cardiovascular diseases, and cancer. Abnormal mitochondrial fission or fusion can disrupt cellular homeostasis and contribute to disease pathogenesis.

Researchers are actively studying DNM1L and mitochondrial dynamics to better understand their roles in health and disease. Targeting DNM1L and related proteins is a potential avenue for developing therapies to address mitochondrial dysfunction and associated diseases.

There is an urgent need for novel diagnostic and therapeutic strategies for patients with Glioblastoma. Previous studies have shown that BCL2-like 13 (BCL2L13) is a member of the BCL2 family regulating cell growth and apoptosis in different types of tumors. However, the clinical significance, biological role, and potential mechanism of GBM remain unexplored. In a study, Wang et al. showed that BCL2L13 expression is significantly upregulated in GBM cell lines and clinical GBM tissue samples. Mechanistically, BCL2L13 targeted DNM1L at the Ser616 site, leading to mitochondrial fission and high mitophagy flux. Functionally, these alterations significantly promoted the proliferation and invasion of GBM cells both in vitro and in vivo. Overall, these findings demonstrated that BCL2L13 plays a significant role in promoting mitophagy via DNM1L-mediated mitochondrial fission in GBM. Therefore, the regulation and biological function of BCL2L13 render it a candidate molecular target for

glioblastoma treatment [1].

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Wang J, Chen A, Xue Z, Liu J, He Y, Liu G, Zhao Z, Li W, Zhang Q, Chen A, Wang J, Li X, Wang X, Huang B. BCL2L13 promotes mitophagy through DNM1L-mediated mitochondrial fission in glioblastoma. Cell Death Dis. 2023 Sep 2;14(9):585. doi: 10.1038/s41419-023-06112-4. PMID: 37660127.