The protein DNM1L, also known as Dynamin-1-like protein (DNM1L) or Drp1 (Dynamin-related protein 1), is involved in mitochondrial dynamics and plays a crucial role in mitochondrial fission, the process by which a single mitochondrion splits into two or more smaller mitochondria. The function and regulation of DNM1L are critical for maintaining the health and proper functioning of mitochondria within cells.

The “Ser616 site” likely refers to a specific phosphorylation site on the DNM1L protein, which is the serine residue at position 616. Phosphorylation is a common post-translational modification of proteins, where phosphate groups are added to specific amino acid residues, such as serine, threonine, or tyrosine. This modification can regulate the activity, localization, and function of the protein.

Phosphorylation of DNM1L at Ser616 can have various effects on its function, including its ability to promote mitochondrial fission. Depending on the context and the specific cellular signals involved, phosphorylation at this site can either activate or inhibit DNM1L activity. It's part of the intricate regulatory mechanisms that control mitochondrial dynamics.

The precise effects of phosphorylation at Ser616 and its role in cellular processes may vary depending on the specific research or context you are interested in. If you have a more specific question or need information on a particular aspect of DNM1L and Ser616 phosphorylation, please provide additional details, and I'll do my best to assist you further.

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)].

1)
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.