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BCL10 Human

Catalog #:PRPS-1065

For research use only.

Description:BCL10 Human Recombinant produced in E. coli is a single polypeptide chain containing 257 amino acids (1-233) and having a molecular mass of 28.8kDa (molecular weight on SDS-PAGE will appear higher).BCL10 is fused to a 24 amino acid His-tag at N-terminus & purified by proprietary chromatographic techniques.

Synonyms:B-cell CLL/lymphoma 10, B-cell lymphoma/leukemia 10, Mammalian CARD-containing adapter molecule E10, CARD-containing molecule enhancing NF-kappa-B, CED-3/ICH-1 prodomain homologous E10-like regulator, CARD-containing apoptotic signaling protein, caspase-r

Source:E.coli.

Physical Appearance: Sterile Filtered colorless solution.

Amino Acid Sequence:MGSSHHHHHH SSGLVPRGSH MGSHMEPTAP SLTEEDLTEV KKDALENLRV YLCEKIIAER HFDHLRAKKI LSREDTEEIS CRTSSRKRAG KLLDYLQENP KGLDTLVESI RREKTQNFLI QKITDEVLKL RNIKLEHLKG LKCSSCEPFP DGATNNLSRS NSDESNFSEK LRASTVMYHP EGESSTTPFF STNSSLNLPV LEVGRTENTI FSSTTLPRPG DPGAPPLPPD LQ

Purity: Greater than 85% as determined by SDS-PAGE.

Formulation:
The BCL10 solution (1mg/ml) contains 20mM Tris-HCl buffer (pH 8.0), 50mM Nacl, 1mM DTT and 10% glycerol.

Stability:
Store at 4°C if entire vial will be used within 2-4 weeks. Store, frozen at -20°C for longer periods of time. For long term storage it is recommended to add a carrier protein (0.1% HSA or BSA).Avoid multiple freeze-thaw cycles.

Usage:
NeoBiolab's products are furnished for LABORATORY RESEARCH USE ONLY. The product may not be used as drμgs, agricultural or pesticidal products, food additives or household chemicals.

Introduction:
BCL10 holds a caspase recruitment domain (CARD), and is known as an inducer of apoptosis and as NF-kappaB activator. BCL10 cooperates with other CARD domain containing proteins such as CARD9, 10, 11 and 14, which operates as upstream regulators in NF-kappaB signaling. BCL10 is known to create a complex with MALT1, a protein encoded by another gene and is translocated in MALT lymphoma. MALT1 and BCL10 protein synergize the activation of NF-kappaB, and the deregulation of either of them facilitates the same pathogenetic process which leads to the malignancy.

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