

Recombinant Mouse NKG2DL/ULBP-1 Protein (His Tag)

Catalog Number: PKSM041116



Note: Centrifuge before opening to ensure complete recovery of vial contents.

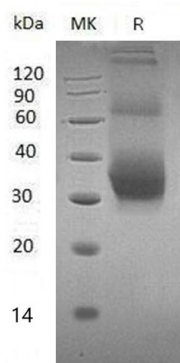
Description

Species	Mouse
Mol_Mass	22.3 kDa
Accession	Q8HWA3
Bio-activity	Not validated for activity

Properties

Purity	> 95 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per µg of the protein as determined by the LAL method.
Storage	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80 °C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of reconstituted samples are stable at < -20°C for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from a 0.2 µm filtered solution of 20mM PB, 150mM NaCl, pH 7.4. Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.
Reconstitution	Please refer to the printed manual for detailed information.

Data



> 95 % as determined by reducing SDS-PAGE.

Background

Mouse ULBP1, also known as RAET1I and NKG2DL1, is a member of the ULBP/RAET1 gene family. ULBP1 plays an important role in immune responses, especially in cancer and infectious diseases, and is well-known to bind to NKG2D together with at least ULBP 2 and 3. These proteins are distantly related to major histocompatibility class I (MHC I) molecules, possessing the alpha 1 and alpha 2 Ig-like domains, but lacking the alpha 3 domain. Unlike MHC Class I, they have no capacity to bind peptide or interact with beta2-microglobulin. It can activate multiple signaling pathways in primary NK cells, gamma delta T cells, and CD8+ alpha beta T cells, resulting in the production of cytokines and chemokines. ULBP1 is expressed in wide range of tissues including heart, brain, lung, liver, bone marrow and some tumor cells, T-cells, B-cells, As an unconventional member of the MHC class I family, ULBP1 is able to interact with soluble CMV glycoprotein UL16 in CMV infected cells. The interaction with UL16 blocked the interaction with the NKG2D receptor, and thus might escape the immune surveillance. Furthermore, UL16 also causes ULBP1 to be retained in the ER and cis-Golgi apparatus so that it does not reach the cell surface. The ULBP1 regulation may have implications for development of new therapeutic strategies against cancer cells.

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A Reliable Research Partner in Life Science and Medicine
Tel:400-999-2100

Email:techsupport@elabscience.cn

Web:www.elabscience.cn

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