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GRP78 Antibody

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP50016

Product Information

Application	WB, IF, IHC
Primary Accession	P11021
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	polyclonal
Calculated MW	72333

Additional Information

Gene ID	3309
Other Names	78 kDa glucose-regulated protein, GRP-78, Endoplasmic reticulum lumenal Ca(2+)-binding protein grp78, Heat shock 70 kDa protein 5, Immunoglobulin heavy chain-binding protein, BiP, HSPA5, GRP78
Dilution	WB~~ 1:1000 IF~~1:100 IHC~~1:50-1:100
Format	Rabbit IgG in phosphate buffered saline (without Mg2+ and Ca2+), pH 7.4, 150mM NaCl, 0.09% (W/V) sodium azide and 50% glycerol.
Storage Conditions	-20℃

Protein Information

Name	HSPA5 (HGNC:5238)
Function	Endoplasmic reticulum chaperone that plays a key role in protein folding and quality control in the endoplasmic reticulum lumen (PubMed:2294010, PubMed:23769672, PubMed:23990668, PubMed:28332555). Involved in the correct folding of proteins and degradation of misfolded proteins via its interaction with DNAJC10/ERdj5, probably to facilitate the release of DNAJC10/ERdj5 from its substrate (By similarity). Acts as a key repressor of the EIF2AK3/PERK and ERN1/IRE1- mediated unfolded protein response (UPR) (PubMed:11907036, PubMed:1550958, PubMed:19538957, PubMed:36739529). In the unstressed endoplasmic reticulum, recruited by DNAJB9/ERdj4 to the luminal region of ERN1/IRE1, leading to disrupt the dimerization of ERN1/IRE1, thereby inactivating ERN1/IRE1 (By similarity). Also binds and inactivates EIF2AK3/PERK in unstressed cells (PubMed:11907036). Accumulation of misfolded protein in the endoplasmic reticulum causes release of HSPA5/BiP from ERN1/IRE1 and EIF2AK3/PERK, allowing their homodimerization and subsequent activation (PubMed:11907036). Plays an auxiliary role in post-translational transport of small presecretory proteins

across endoplasmic reticulum (ER). May function as an allosteric modulator for SEC61 channel-forming translocon complex, likely cooperating with SEC62 to enable the productive insertion of these precursors into SEC61 channel. Appears to specifically regulate translocation of precursors having inhibitory residues in their mature region that weaken channel gating. May also play a role in apoptosis and cell proliferation (PubMed:26045166).

Cellular Location

Endoplasmic reticulum lumen. Melanosome. Cytoplasm {ECO:0000250|UniProtKB:P20029}. Cell surface Note=Identified by mass spectrometry in melanosome fractions from stage I to stage IV (PubMed:12643545). Localizes to the cell surface of epithelial cells in response to high levels of free iron (PubMed:20484814, PubMed:24355926, PubMed:27159390)

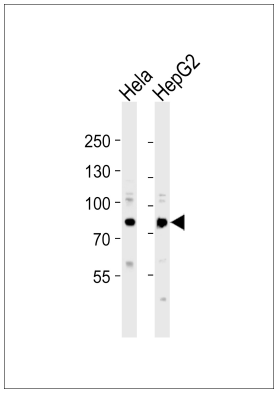
Background

Probably plays a role in facilitating the assembly of multimeric protein complexes inside the endoplasmic reticulum. Involved in the correct folding of proteins and degradation of misfolded proteins via its interaction with DNAJC10, probably to facilitate the release of DNAJC10 from its substrate.

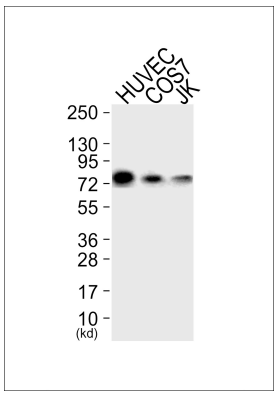
References

Ting J.,et al.DNA 7:275-286(1988).
Chao C.C.K.,et al.Submitted (DEC-1995) to the EMBL/GenBank/DDBJ databases.
Hansen J.J.,et al.Submitted (JAN-2000) to the EMBL/GenBank/DDBJ databases.
Bermudez-Fajardo A.,et al.Submitted (DEC-1999) to the EMBL/GenBank/DDBJ databases.
Humphray S.J.,et al.Nature 429:369-374(2004).

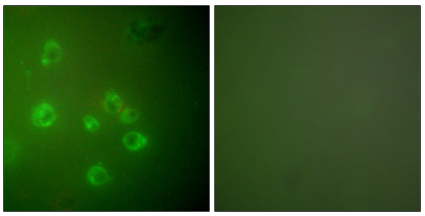
Images



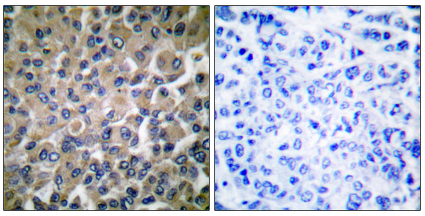
Western blot analysis of lysates from Hela,HepG2 cell line (from left to right),using GRP78 Antibody(C0217). C0217 was diluted at 1:1000 at each lane. A goat anti-rabbit IgG H&L(HRP) at 1:5000 dilution was used as the secondary antibody.Lysates at 35ug per lane.



Western blot analysis of extracts from HUVEC cells (Lane 1), COS7 cells (Lane 2) and JK cells (Lane 3), using GRP78 Antibody. The lane on the left is treated with systhesized peptide.



Immunofluorescence analysis of COS7 cells, using GRP78 antibody .



Immunohistochemical analysis of paraffin-embedded human breast carcinoma tissue using GRP78 antibody .

Citations

- LMAN1 (ERGIC-53) promotes trafficking of neuroreceptors.
- Remodeling the endoplasmic reticulum proteostasis network restores proteostasis of pathogenic GABAA receptors.

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