

Anti-Kidins220 [p220 1F8/3]

Catalogue number: 151252

Sub-type: Primary antibody

Images:

Contributor

Inventor: Giampietro Schiavo

Institute: Cancer Research UK, London Research Institute: Lincoln's Inn Fields

Images:

Tool details

***FOR RESEARCH USE ONLY**

Name: Anti-Kidins220 [p220 1F8/3]

Alternate name:

Class: Monoclonal

Conjugate: Unconjugated

Description: Kidins220/ARMS (Kinase D-Interacting Substrate of 220 kDa, ARMS; Ankyrin Repeat-rich Membrane Spanning) is an integral membrane protein that is selectively expressed in brain and neuroendocrine cells and phosphorylated by PKD (protein Kinase D). Kidins220 has also been shown to function downstream of the Trk and Eph receptor tyrosine kinases.

Purpose:

Parental cell:

Organism:

Tissue:

Model:

Gender:

Isotype: IgG1

Reactivity: Rat

Selectivity:

Host: Mouse

Immunogen: Recombinant GST-Kidins220/ARMS (last 347 amino acids at the carboxy terminus) fusion protein.

Immunogen UNIPROT ID:

Sequence:

Growth properties:

Production details:

Formulation:

Recommended controls:

Bacterial resistance:

Selectable markers:

Additional notes:

Target details

Target: Kidins220/ARMS

Target alternate names:

Target background: Kidins220/ARMS (Kinase D-Interacting Substrate of 220 kDa, ARMS; Ankyrin Repeat-rich Membrane Spanning) is an integral membrane protein that is selectively expressed in brain and neuroendocrine cells and phosphorylated by PKD (protein Kinase D). Kidins220 has also been shown to function downstream of the Trk and Eph receptor tyrosine kinases.

Molecular weight: 220 kDa

Ic50:

Applications

Application: ELISA ; IHC ; IP ; WB

Application notes:

Handling

Format: Liquid

Concentration: 1 mg/ml

Passage number:

Growth medium:

Temperature:

Atmosphere:

Volume:

Storage medium:

Storage buffer: PBS with 0.02% azide

Storage conditions: -15° C to -25° C

Shipping conditions: Shipping at 4° C

Related tools

Related tools:

References

References: Holt et al. 2010. Development. 137(8):1297-304. PMID: 20223764. ; Spatial regulation of APC^{Cdh1}-induced cyclin B1 degradation maintains G2 arrest in mouse oocytes.

CancerTools.org