

DESCRIPTION

Species Reactivity	Human/Mouse/Rat
Specificity	Detects human, mouse, and rat CDC25B. Four splice variants of CDC25B, with molecular weights of 61, 63, 65, and 67 kDa, are known. The immunogen selected for this antibody is common to all variants. Immunoreactivity consistent with at least 3 variants has
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	<i>E. coli</i> -derived recombinant human CDC25B Glu391-Gln580 Accession # P30305
Conjugate	Alexa Fluor 750 Excitation Wavelength: 749 nm Emission Wavelength: 775 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. [General Protocols](#) are available in the Technical Information section on our website.

Western Blot	Optimal dilution of this antibody should be experimentally determined.
Immunocytochemistry	Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

BACKGROUND

Cell Division Cycle 25B (Cdc25B) phosphatase removes inorganic phosphate groups covalently attached to tyrosine, serine and threonine residues in proteins (1). Breast cancer patients bearing tumors containing high levels of Cdc25B have been found to have a greater incidence of aggressive, high-grade tumors than those with low Cdc25B levels (2). In cells, the levels of Cdc25B activity are highest during the G2/M transition of the cell cycle, where it is suspected to be involved in "checkpoint" control of cell cycle progression (3). Overexpression of Cdc25B reduces the G2/M cell cycle block caused by ionizing radiation (4). Although activated by phosphorylation, Ser323 phosphorylation causes the enzyme to bind the protein 14-3-3, preventing substrate access to the catalytic site (5). One of the major substrates of Cdc25B is Cdc2, a kinase that is activated by dephosphorylation (6). The recombinant protein is truncated to remove the N-terminal regulatory domains and is fully active.

PRODUCT SPECIFIC NOTICES

This product is provided under an agreement between Life Technologies Corporation and R&D Systems, Inc., and the manufacture, use, sale or import of this product is subject to one or more US patents and corresponding non-US equivalents, owned by Life Technologies Corporation and its affiliates. The purchase of this product conveys to the buyer the non-transferable right to use the purchased amount of the product and components of the product only in research conducted by the buyer (whether the buyer is an academic or for-profit entity). The sale of this product is expressly conditioned on the buyer not using the product or its components (1) in manufacturing; (2) to provide a service, information, or data to an unaffiliated third party for payment; (3) for therapeutic, diagnostic or prophylactic purposes; (4) to resell, sell, or otherwise transfer this product or its components to any third party, or for any other commercial purpose. Life Technologies Corporation will not assert a claim against the buyer of the infringement of the above patents based on the manufacture, use or sale of a commercial product developed in research by the buyer in which this product or its components was employed, provided that neither this product nor any of its components was used in the manufacture of such product. For information on purchasing a license to this product for purposes other than research, contact Life Technologies Corporation, Cell Analysis Business Unit, Business Development, 29851 Willow Creek Road, Eugene, OR 97402, Tel: (541) 465-8300. Fax: (541) 335-0354.