

DESCRIPTION

Species Reactivity	Human/Mouse/Rat
Specificity	Detects human PKM2 in direct ELISAs. Detects human, mouse and rat PKM1/2 in Western blot.
Source	Monoclonal Mouse IgG _{2B} Clone # 945131
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	<i>E. coli</i> -derived recombinant human PKM2 Ser2-Pro531 Accession # P14618-1
Conjugate	Alexa Fluor 532 Excitation Wavelength: 534 nm Emission Wavelength: 553 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.

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

PKM2 (Pyruvate Kinase isoenzyme M2; also p58, OIP3, THBP1 and CTHBP) is a 58-60 kDa member of the PK family of enzymes. It is widely expressed, being found both intracellularly and in blood, and represents the more common splice variant of the PKM gene. PKM2 generates ATP and pyruvate by catalyzing the transfer of a phosphoryl group from PEP to ADP. Thus, when active, PKM2 promotes energy production and glycolysis. PKM2 exists as a marginally active monomer, with full activity achieved through homotetramerization. Notably, in tumor cells, select oncogenes appear to induce PKM2 homodimerization which limits PKM2 activity. PKM2 is known to be regulated by the binding of T3 and Fru-1,6-bisP. Human PKM2 is 531 amino acids (aa) in length. It contains a catalytic region (aa 43-527) plus four utilized Ser/Thr and Tyr phosphorylation sites, respectively. PKM1 is another PKM gene splice variant that shows a 45 aa substitution for aa 389-433 of PKM2. This variant shows limited expression (striated muscle) and hyperbolic Michaelis-Menten kinetics. There are additional isoform variants of PKM2 that show either a deletion of aa 59-132, or a 67 aa substitution for aa 1-82. Over aa 434-531, human PKM2 shares 95% aa sequence identity with mouse PKM2.

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.