

DESCRIPTION

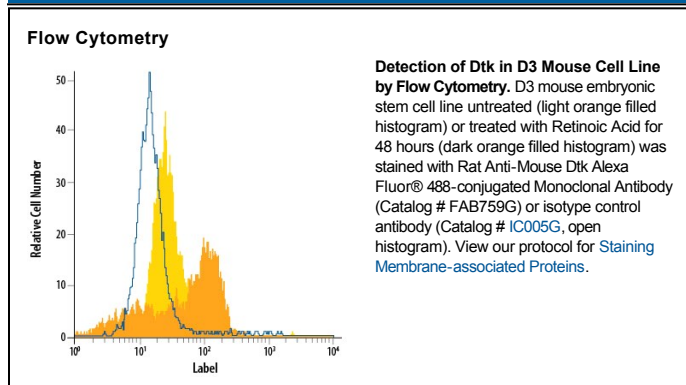
Species Reactivity	Mouse
Specificity	Detects mouse Dtk extracellular domain in direct ELISAs and Western blots. In direct ELISAs and Western blots, no cross-reactivity with recombinant human Dtk, recombinant mouse (rm) Mer, or rmAxl is observed.
Source	Monoclonal Rat IgG ₁ Clone # 109646
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant mouse Dtk Ala31-Ser418 Accession # P55144
Conjugate	Alexa Fluor 488 Excitation Wavelength: 488 nm Emission Wavelength: 515-545 nm
Formulation	Supplied in a saline solution containing BSA and Sodium Azide. See Certificate of Analysis for details. *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.

	Recommended Concentration	Sample
Flow Cytometry	5 µL/10 ⁶ cells	See Below

DATA



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

Axl (Ufo, Ark), Dtk (Sky, Tyro3, Rse, Brt) and Mer (human and mouse homologues of chicken c-Eyk) constitute a new receptor tyrosine kinase subfamily. The extracellular domain of these proteins contain two Ig-like motifs and two fibronectin type III motifs. This characteristic topology is also found in neural cell adhesion molecules and in receptor tyrosine phosphatases. All three receptors bind the vitamin K-dependent protein growth-arrest specific gene 6 (Gas6) which is structurally related to the anticoagulation factor protein S. The binding affinities for Gas6 is in the order of Axl > Dtk > Mer. Gas6 binding induces tyrosine phosphorylation and downstream signaling pathways that can lead to cell proliferation, migration, or the prevention of apoptosis. Dtk is widely expressed during embryonic development. In adults, Dtk is predominantly expressed in neurons in restricted regions of the brain.

References:

1. Nagata, K. *et al.* (1996) *J. Biol. Chem.* **22**:30022.
2. Crosier, K.E. and P.S Crosier (1997) *Pathology* **29**:131.

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.