

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human SLC31A1 in direct ELISA.
Source	Recombinant Monoclonal Rabbit IgG Clone # 2938A
Purification	Protein A or G purified from cell culture supernatant
Immunogen	Synthetic peptide Accession # O15431
Conjugate	Alexa Fluor 405 Excitation Wavelength: 405 nm Emission Wavelength: 421 nm
Formulation	Supplied 0.2 mg/mL in a saline solution containing BSA and 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.

Flow Cytometry	Titration recommended for optimal concentration with starting range of 0.1-1 µg/1 million cells. Sample used for this experiment was K562 human chronic myelogenous leukemia cell line.
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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

SLC31A1/CTR1 (solute carrier family 31 member 1/copper transporter 1) is a homotrimeric high-affinity, saturable copper importer implicated in dietary copper uptake. Homeostasis of copper is tightly controlled by inter-regulatory circuitry containing copper, Sp1, and CTR1, wherein Sp1 transcription factor acts as a copper sensor in modulating CTR1 expression which in turn controls cellular copper and Sp1 levels in a 3-way mutual regulatory loop. CTR1 mediated copper uptake is time, temperature, and pH dependent and specific for reduced form of Cu (I) and copper reduction has been proposed to be caused by Steap protein family or Dcytb protein (Cybrd1) both of which are reported to possess cupric reductase activity. The intracellular localization of CTR1 is variable in different cell lines: CTR1 is localized at the plasma membrane in HEK293, CaCo-2, and A2780 cells, whereas, in other cells such as HeLa, it is predominantly localized to vesicular compartments, and variability in trafficking rates between vesicular and plasma membrane compartments has been suggested as the cause of this differential localization. Post-translational O-linked glycosylation of CTR1 protects it against proteolytic cleavage of N terminal 17-kD fragment and the biological function of this cleaved variant or protease responsible is largely unknown. Selective knockout of Ctr1 in murine intestinal epithelial cells results in severe systemic copper deficiency, ataxia, and death prior to weaning.

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