

DESCRIPTION

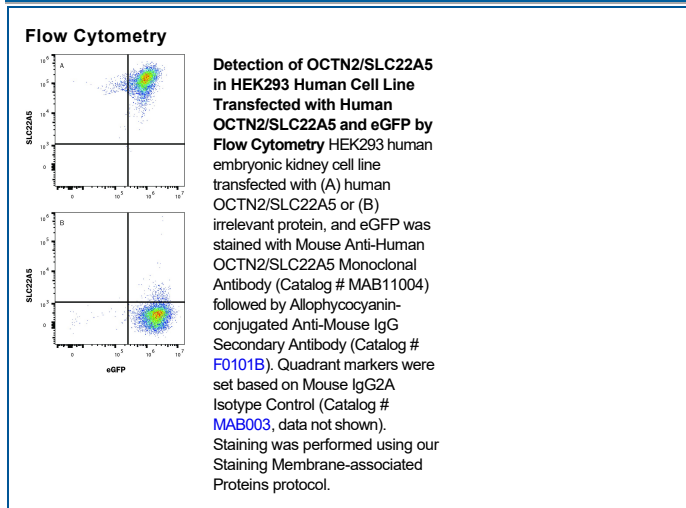
Species Reactivity	Human
Specificity	Detects human OCTN2/SLC22A5 in ELISA.
Source	Monoclonal Mouse IgG _{2A} Clone # 893753
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	NS0 transfected with human SLC22A5 Accession # O76082
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied either lyophilized or as a 0.2 µm filtered solution in PBS.

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	0.25 µg/10 ⁶ cells	HEK293 Human Cell Line Transfected with Human OCTN2/SLC22A5 and eGFP

DATA



PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.5 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> • 12 months from date of receipt, -20 to -70 °C as supplied. • 1 month, 2 to 8 °C under sterile conditions after reconstitution. • 6 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

Polyspecific organic cation transporters in the liver, kidney, intestine, and other organs are critical for the elimination of many endogenous small organic cations as well as a wide array of drugs and environmental toxins. SLC22A5 is an integral plasma membrane protein which functions as an organic cation transporter and as a sodium-dependent high affinity carnitine transporter. Mutations in SLC22A5 are the cause of systemic primary carnitine deficiency (CDSP), an autosomal recessive disorder manifested early in life by hypoketotic hypoglycemia and acute metabolic decompensation, and later in life by skeletal myopathy or cardiomyopathy.