Data Sheet (Cat.No.TMAY-02550F)

TargetMol

Anti-FDPS Antibody-FITC (8F434)

Product Details

Ig Type:	Rabbit IgG
Reactivity:	Human
Conjugation:	FITC
Clone:	8F434
Purification:	Protein A

Applications

Verified Activity:	Flow cytometric analysis of Human FDPS expression on HeLa cells. The cells were treated according to manufacturer's manual (BD Pharmingen™ Cat. No. 554714), and then stained with FITC-conjugated anti-Human FDPS. The fluorescence histograms were derived from gated events with the forward and side light-scatter characteristics of intact cells.
Application:	FCM
Recommended	10 μl/Test, 0.1 mg/ml

Properties

Stability & Storage:	Store at 2°C-8°C for 12 months, do not freeze. Keep away from direct sunlight. Sodium azide is toxic to cells and should be disposed of properly. Flush with large volumes of water during disposal.
Shipping:	Shipping with blue ice.

Antigen Details

Immunogen:	Recombinant Protein: Human FDPS protein (TMPY-02654)
Antigen Species:	Human
Synonyms:	FPPS;Farnesyl pyrophosphate synthase;FPS

Research Background

Z-farnesyl diphosphate synthase (FDPS) is an enzyme belonging to the family of transferases, specifically those transferring aryl or alkyl groups other than methyl groups. Z-farnesyl diphosphate synthase (FDPS) functions as key enzyme in isoprenoid biosynthesis which catalyzes the formation of farnesyl diphosphate, a precurcor for several classes of essential metabolites. FDPS catalyzes the production of geranyl pyrophosphate and farnesyl pyrophosphate from isopentenyl pyrophosphate and dimethylallyl pyrophosphate. The resulting product, farnesyl pyrophosphate, is a key intermediate in cholesterol and sterol biosynthesis, a substrate for protein farnesylation and geranylgeranylation, and a ligand or agonist for certain hormone receptors and growth receptors. Drugs that inhibit this enzyme prevent the post-translational modifications of small GTPases and have been used to treat diseases related to bone resorption. Functions of FDPS may be inactivated by interferon-induced RSAD2. This inactivation may result of disruption of lipid rafts at the plasma membrane, and thus have an antiviral effect since many enveloped viruses need lipid rafts to bud efficiently out of the cell.

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