Recombinant Human HRAS/GTPase Hras Protein (His Tag)

Catalog Number: PKSH030919

Note: *Centrifuge before opening to ensure complete recovery of vial contents.*

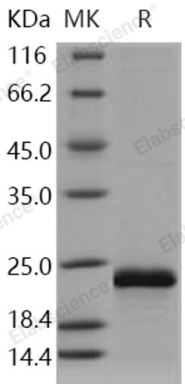
Description

Species	Human
Source	Baculovirus-Insect Cells-derived Human HRAS/GTPase Hras protein Met 1-Cys 186, with an C-terminal His
Calculated MW	22.4 kDa
Observed MW	23 kDa
Accession	P01112
Bio-activity	Not validated for activity

Properties

Purity	> 94 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per μg of the protein as determined by the LAL method.
Storage	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80 ℃. Reconstituted protein solution can be stored at 4-8℃ for 2-7 days. Aliquots of reconstituted samples are stable at < -20℃ for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from sterile 50mM Tris, 100mM NaCl, pH 8.0, 10% glycerol Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.
Reconstitution	Please refer to the printed manual for detailed information.

Data



> 94 % as determined by reducing SDS-PAGE.

Background

HRas, also known as HRAS, belongs to the small GTPase superfamily, Ras family and is widely expressed. It functions in signal transduction pathways. HRas can bind GTP and GDP, and they have intrinsic GTPase activity. It undergoes a continuous cycle of de- and re-palmitoylation, which regulates its rapid exchange between the plasma membrane and the Golgi apparatus. Defects in HRAS are the cause of faciocutaneoskeletal syndrome (FCSS). FCSS is arare condition characterized by prenatally increased growth, postnatal growth deficiency, mental retardation, distinctive facial appearance, cardiovascular abnormalities, tumor predisposition, skin and musculoskeletal abnormalities. Defects in HRAS also can cause congenital myopathy with excess of muscle spindles. HRAS deficiency may be a cause of susceptibility to Hurthle cell thyroid carcinoma. It has been shown that defects in HRAS can cause susceptibility to bladder cancer which is a malignancy originating in tissues of the urinary bladder. It often presents with multiple tumors appearing at different times and at different sites in the bladder. Most bladder cancers are transitional cell carcinomas. They begin in cells that normally make up the inner lining of the bladder. Other types of bladder cancer include squamous cell carcinoma (cancer that begins in thin, flat cells) and adenocarcinoma (cancer that begins in cells that make and release mucus and other fluids). Bladder cancer is a complex disorder with both genetic and environmental influences. Defects in HRAS are the cause of oral squamous cell carcinoma.

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