

Product datasheet

Recombinant Human KRAS (mutated Q61H) protein ab96817

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Description

Product name	Recombinant Human KRAS (mutated Q61H) protein
Purity	> 90 % SDS-PAGE. ab96817 is purified using conventional chromatography techniques.
Expression system	Escherichia coli
Accession	<u>P01116-2</u>
Protein length	Full length protein
Animal free	No
Nature	Recombinant
Species	Human
Sequence	MGSSHHHHHH SSGLVPRGSH MTEYKLVVVG AGGVGKSALT IQLIQNHFVD EYDPTIEDSY RKQVVIDGET CLLDILD TAG HEEYSAMRDQ YMRTGEGFLC VFAINNTKSF EDIHHYREQI KRVKDS EDVP MVLVGNKCDL PSRTVDTKQA QDLARSYGIP FIETSAKTRQ GVDDAFYTLV REIRKHKEKM SKDGKKKKKK SKTKC
Predicted molecular weight	23 kDa including tags
Amino acids	1 to 185
Modifications	mutated Q61H
Tags	His tag N-Terminus
Additional sequence information	Corresponds to K-Ras4B (Isoform 2B)

Specifications

Our **Abpromise guarantee** covers the use of **ab96817** in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Applications	Mass Spectrometry
	SDS-PAGE
	Western blot

Mass spectrometry	MALDI-TOF-TOF
Form	Liquid
Additional notes	Isoform 2B

Preparation and Storage

Stability and Storage	<p>Shipped at 4°C. Upon delivery aliquot and store at -20°C or -80°C. Avoid repeated freeze / thaw cycles.</p> <p>pH: 8.00</p> <p>Constituents: 0.0154% DTT, 0.316% Tris HCl, 10% Glycerol, 0.58% Sodium chloride</p>
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General Info

Function	Ras proteins bind GDP/GTP and possess intrinsic GTPase activity.
Involvement in disease	<p>Defects in KRAS are a cause of acute myelogenous leukemia (AML) [MIM:601626]. AML is a malignant disease in which hematopoietic precursors are arrested in an early stage of development.</p> <p>Defects in KRAS are a cause of juvenile myelomonocytic leukemia (JMML) [MIM:607785]. JMML is a pediatric myelodysplastic syndrome that constitutes approximately 30% of childhood cases of myelodysplastic syndrome (MDS) and 2% of leukemia. It is characterized by leukocytosis with tissue infiltration and in vitro hypersensitivity of myeloid progenitors to granulocyte-macrophage colony stimulating factor.</p> <p>Defects in KRAS are the cause of Noonan syndrome type 3 (NS3) [MIM:609942]. Noonan syndrome (NS) [MIM:163950] is a disorder characterized by dysmorphic facial features, short stature, hypertelorism, cardiac anomalies, deafness, motor delay, and a bleeding diathesis. It is a genetically heterogeneous and relatively common syndrome, with an estimated incidence of 1 in 1000-2500 live births. Rarely, NS is associated with juvenile myelomonocytic leukemia (JMML). NS3 inheritance is autosomal dominant.</p> <p>Defects in KRAS are a cause of gastric cancer (GASC) [MIM:613659]; also called gastric cancer intestinal or stomach cancer. Gastric cancer is a malignant disease which starts in the stomach, can spread to the esophagus or the small intestine, and can extend through the stomach wall to nearby lymph nodes and organs. It also can metastasize to other parts of the body. The term gastric cancer or gastric carcinoma refers to adenocarcinoma of the stomach that accounts for most of all gastric malignant tumors. Two main histologic types are recognized, diffuse type and intestinal type carcinomas. Diffuse tumors are poorly differentiated infiltrating lesions, resulting in thickening of the stomach. In contrast, intestinal tumors are usually exophytic, often ulcerating, and associated with intestinal metaplasia of the stomach, most often observed in sporadic disease. Note=Defects in KRAS are a cause of pilocytic astrocytoma (PA). Pilocytic astrocytomas are neoplasms of the brain and spinal cord derived from glial cells which vary from histologically benign forms to highly anaplastic and malignant tumors.</p> <p>Defects in KRAS are a cause of cardiofaciocutaneous syndrome (CFC syndrome) [MIM:115150]; also known as cardio-facio-cutaneous syndrome. CFC syndrome is characterized by a distinctive facial appearance, heart defects and mental retardation. Heart defects include pulmonic stenosis, atrial septal defects and hypertrophic cardiomyopathy. Some affected individuals present with ectodermal abnormalities such as sparse, friable hair, hyperkeratotic skin lesions and a generalized ichthyosis-like condition. Typical facial features are similar to Noonan syndrome. They include high forehead with bitemporal constriction, hypoplastic supraorbital ridges, downslanting palpebral fissures, a depressed nasal bridge, and posteriorly angulated ears with</p>

prominent helices. The inheritance of CFC syndrome is autosomal dominant.
Note=KRAS mutations are involved in cancer development.

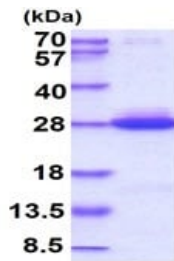
Sequence similarities

Belongs to the small GTPase superfamily. Ras family.

Cellular localization

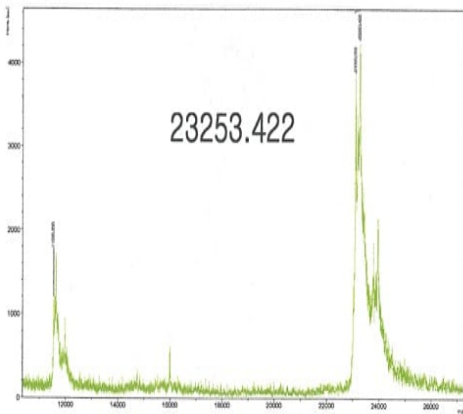
Cell membrane.

Images



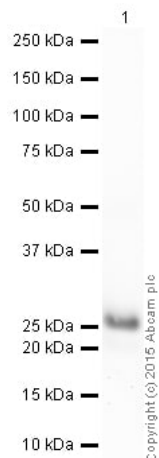
15% SDS-PAGE showing ab96817 at approximately 23.2 kDa (3 μ g).

SDS-PAGE - Recombinant Human KRAS (mutated Q61H) protein (ab96817)



MALTI-TOF result: of 23253.422 Da for Human KRAS (mutated Q61H) protein.

Mass Spectrometry - Recombinant Human KRAS (mutated Q61H) protein (ab96817)



Western blot - Recombinant Human KRAS (mutated Q61H) protein (ab96817)

This blot was produced using a 4-12% Bis-tris gel under the MES buffer system. The gel was run at 200V for 35 minutes before being transferred onto a Nitrocellulose membrane at 30V for 70 minutes. The membrane was then blocked for an hour using 5% Bovine Serum Albumin before being incubated with [ab84573](#) overnight at 4°C. Antibody binding was detected using an anti-rabbit antibody conjugated to HRP, and visualised using ECL development solution.

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