

Product datasheet

Recombinant Human KRAS protein ab156314

1 Image

Overview

Product name	Recombinant Human KRAS protein
Protein length	Full length protein

Description

Nature	Recombinant
Source	Escherichia coli
Amino Acid Sequence	
Accession	P01116
Species	Human
Sequence	<p>MGSSHHHHHH SSGLVPRGSH MGSHTMTEYKL VVVGAGGVGK SALTQLIQN HFVDEYDPTI EDSYRKQVVIDGETCLLDIL DTAGQEEYSA MRDQYMRTGE GFLCVFAINN TKSFEDIHHY REQIKRVKDS EDVPMVLVGN KCDLPSRTVD TKQAQDLARS YGIPFIETSA KTRQRVEDAF YTLVREIRQY RLKKISKEEK TPGCVKIKKC</p>
Molecular weight	24 kDa including tags
Amino acids	1 to 186
Tags	His tag N-Terminus

Specifications

Our [Abpromise guarantee](#) covers the use of **ab156314** in the following tested applications.

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

Applications	<p>SDS-PAGE</p> <p>Mass Spectrometry</p>
Mass spectrometry	MALDI-TOF
Purity	>90% by SDS-PAGE.
Form	Liquid

Preparation and Storage

Stability and Storage

Shipped at 4°C. Store at +4°C short term (1-2 weeks). Upon delivery aliquot. Store at -20°C or -80°C. Avoid freeze / thaw cycle.

pH: 8.00

Constituents: 0.02% DTT, 0.32% Tris HCl, 20% Glycerol, 0.58% Sodium chloride

General Info

Function

Ras proteins bind GDP/GTP and possess intrinsic GTPase activity.

Involvement in disease

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.

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.

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.

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.

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 prominent helices. The inheritance of CFC syndrome is autosomal dominant.

Note=KRAS mutations are involved in cancer development.

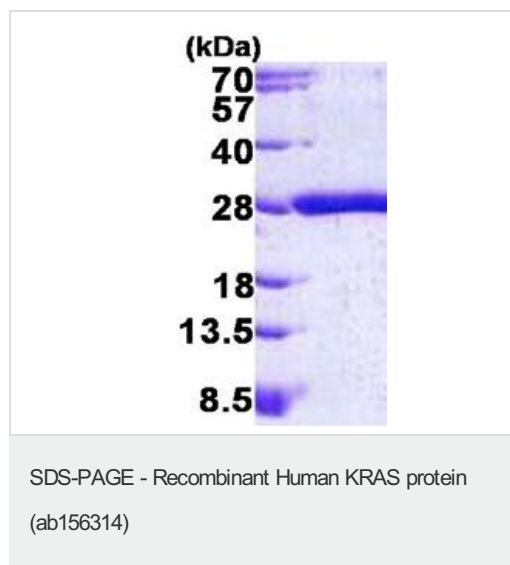
Sequence similarities

Belongs to the small GTPase superfamily. Ras family.

Cellular localization

Cell membrane.

Images



15% SDS-PAGE analysis of ab156314 (3µg).

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