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Product datasheet

Ras GTPase ELISA Kit (Chemiluminescent) ab134640

6 References 1 Image

Overview

Product name	Ras GTPase ELISA Kit (Chemiluminescent)
Detection method	Luminescent
Sample type	Cell culture extracts, Tissue Extracts
Assay type	Sandwich (quantitative)
Sensitivity	> 3000 ng/well
Range	3000 ng/well - 25000 ng/well
Assay time	4h 30m
Assay duration	Multiple steps standard assay
Species reactivity	**Reacts with:** Mouse, Human
Product overview	ab134640 Ras GTPase ELISA Kit is designed specifically for the study of Ras activation and can be used to study novel signaling pathways for activating Ras. The kit can also be used as a diagnostic test to detect oncogenic Ras related to malignancy. Ras GTPase ELISA Kits contain a Raf-RBD protein fused to GST that will be coated onto the provided 96-well (12 x 8 well plate), glutathione-coated plate. The activated Ras contained in cellular extract specifically binds to Raf-RBD, while inactive Ras does not bind. Bound Ras is detected by incubating with a primary antibody that detects H-Ras in mouse and H- & K-Ras in human samples. Addition of a secondary antibody conjugated to horseradish peroxidase (HRP) and developing solution provides a sensitive chemiluminescent readout that is easily quantified by luminescence. The 96-well plate with individual plate of 12 strips is suitable for manual use or high-throughput screening applications.
	Ras GTPase ELISA Kit specifically detects activated H- and K-Ras in Human and H-Ras in rodent samples.
Notes	Abcam has not and does not intend to apply for the REACH Authorisation of customers' uses of products that contain European Authorisation list (Annex XIV) substances. It is the responsibility of our customers to check the necessity of application of REACH Authorisation, and any other relevant authorisations, for their intended uses.
Platform	Microplate

Properties

Storage instructions Please refer to protocols.

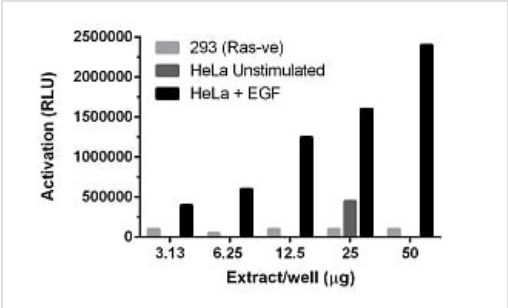
Components	1 x 96 tests	1 x 96 tests
10X Antibody Binding Buffer	1 x 2.2ml	1 x 2.2ml
10X Wash Buffer	2 x 22ml	2 x 22ml
96-well assay plate	1 unit	1 unit
Anti-rat HRP-conjugated IgG	1 x 11µl	1 x 11µl
Chemiluminescent Reagent	1 x 2ml	1 x 2ml
GST-Raf-RBD	4 x 25µl	4 x 25µl
HeLa whole-cell extract (EGF treated)	2 x 40µl	2 x 40µl
H-Ras antibody	1 x 11µl	1 x 11µl
Lysis/Binding Buffer	1 x 50ml	1 x 50ml
Plate sealer	1 unit	1 unit
Protease Inhibitor Cocktail	1 x 500µl	1 x 500µl
Reaction Buffer	1 x 4ml	1 x 4ml

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

Involvement in disease Defects in HRAS are the cause of faciocutaneoskeletal syndrome (FCSS) [MIM:218040]. A rare condition characterized by prenatally increased growth, postnatal growth deficiency, mental retardation, distinctive facial appearance, cardiovascular abnormalities (typically pulmonic stenosis, hypertrophic cardiomyopathy and/or atrial tachycardia), tumor predisposition, skin and musculoskeletal abnormalities.
Defects in HRAS are the cause of congenital myopathy with excess of muscle spindles (CMEMS) [MIM:218040]. CMEMS is a variant of Costello syndrome.
Defects in HRAS may be a cause of susceptibility to Hurthle cell thyroid carcinoma (HCTC) [MIM:607464]. Hurthle cell thyroid carcinoma accounts for approximately 3% of all thyroid cancers. Although they are classified as variants of follicular neoplasms, they are more often multifocal and somewhat more aggressive and are less likely to take up iodine than are other follicular neoplasms.
Note=Mutations which change positions 12, 13 or 61 activate the potential of HRAS to transform cultured cells and are implicated in a variety of human tumors.
Defects in HRAS are a cause of susceptibility to bladder cancer (BLC) [MIM:109800]. 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.

	Note=Defects in HRAS are the cause of oral squamous cell carcinoma (OSCC).
Sequence similarities	Belongs to the small GTPase superfamily. Ras family.
Post-translational modifications	Palmitoylated by the ZDHHC9-GOLGA7 complex. A continuous cycle of de- and re-palmitoylation regulates rapid exchange between plasma membrane and Golgi. S-nitrosylated; critical for redox regulation. Important for stimulating guanine nucleotide exchange. No structural perturbation on nitrosylation.
Cellular localization	Cell membrane. Golgi apparatus membrane. The active GTP-bound form is localized most strongly to membranes than the inactive GDP-bound form (By similarity). Shuttles between the plasma membrane and the Golgi apparatus.

Images



ELISA - Ras GTPase ELISA Kit (ab134640)

Quantification of activated Ras: Increasing amounts of whole-cell extracts from unstimulated 293T/17 and EGF stimulated HeLa cells were assayed for Ras activity using ab134640 Ras GTPase ELISA Kit. To illustrate the Kit's specificity for activated Ras, 293T/17 cells which do not contain basal levels of activated Ras were used as a negative control. Data was also shown for unstimulated HeLa cells, which do contain basal levels of activated Ras. This data is provided for demonstration only.

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