DATA SHEET

Jena Bioscience

KRas^{His} - G-domain

Kirsten rat sarcoma viral oncogene homolog, G-domain
human, recombinant, *E. coli*

Cat. No.	Amount
PR-239	50 µg

For general laboratory use.

Shipping: shipped on gel packs

Storage Conditions: store at -20 °C

Additional Storage Conditions: avoid freeze/thaw cycles

Shelf Life: 12 months

Molecular Weight: 25 kDa

Accession number: NM_004985.3

Purity: > 90 % (SDS-PAGE)

Form: liquid (Supplied in PBS pH 7.5, 5 mM $MgCl_2$ and 50 % glycerol)

Description:
Ras proteins are members of the superfamily of small GTP-binding proteins that function as molecular switches controlling a variety of signalling and transport pathways.
KRAS gene performs an essential function in normal tissue signaling, and the mutation of a KRAS gene is an essential step in the development of many cancers.
KRAS is usually tethered to cell membranes because of the presence of an isoprenyl group on its C-terminus.
Protein preparation is 100 % GDP-loaded, measured by HPLC.
Human K-Ras is a truncated protein containing amino acids 1-163. The 6His-tag is located at the N-terminus.

Selected References:

Zimmermann *et al.* (2013) Small molecule inhibition of the KRAS-PDE? interaction impairs oncogenic KRAS signalling. Nature 497:638.

Sasazuki *et al.* (2005) Transformation by Oncogenic RAS Sensitizes

Human Colon Cells to TRAIL-induced Apoptosis by Up-regulating Death Receptor 4 and Death Receptor 5 through a MEK-dependent Pathway. J. Biol. Chem. 280:22856.

931Li *et al.* (2004) Transformation Potential of Ras Isoforms Correlates with Activation of Phosphatidylinositol 3-Kinase but Not ERK. J. Biol. Chem. 279:37398.

Wittinghofer *et al.* (2000) Ras - a molecular switch involved in tumor formation. Angew. Chem. Int. Ed. 39:4192.

Li *et al.* (1997) Uncoupling of membrane ruffling and pinocytosis

during Ras signal transduction. J. Biol. Chem. 272:10337.

Pacold *et al.* (2000) Crystal structure and functional analysis of Ras

binding to its effector Phosphoinositide 3-kinase. Cell 103:931.