DATA SHEET

Jena Bioscience

H-Ras ΔC (S17N)

Harvey rat sarcoma viral oncogene homolog, residues 1-166
human, recombinant, *E. coli*

Cat. No.	Amount
PR-202	50 µg

For general laboratory use.

Shipping: shipped on dry ice

Storage Conditions: store at -80 °C

Additional Storage Conditions: avoid freeze/thaw cycles

Shelf Life: 12 months

Molecular Weight: 19.5 kDa (171 amino acids)

Accession number: NP_005334

Purity: > 90 % (SDS-PAGE)

Form: liquid (Supplied in 64 mM Tris-HCl pH 7.2, 10 mM $MgCl_2$ and 5 mM DTE)

Description:
Ras proteins are members of the superfamily of small GTP-binding proteins that function as molecular switches controlling a variety of signaling and transport pathways. H-Ras is one of the classic human Ras proteins (H-, N-, K-Ras4A, and K-Ras4B). H-Ras ΔC (aa 1 - 171) lacks the C-terminus with the CaaX recognition sequence necessary for anchoring Ras into the plasma membrane. The mutation S17N results in a 40-fold increase in the affinity for GTP without affecting its affinity for GDP. Protein preparation is 95% GDP- and 5% GTP-loaded, measured by HPLC.

Selected References:

Nassar *et al.* (2010) Structure of the Dominant Negative S17N Mutant of Ras. *Biochemistry* **49**:1970.

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.

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

Feig *et al.* (1988) Inhibition of NIH 3T3 cell proliferation by a mutant ras protein with preferential affinity for GDP. *Mol. Cell. Biol.* **8**:3235.