

**H-Ras (G12V)<sup>GST</sup>**

Harvey rat sarcoma viral oncogene homolog  
human, recombinant, *E. coli*

Cat. No.	Amount
PR-358	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

**Accession number:** NP\_005334

**Purity:** > 90 % (SDS-PAGE)

**Form:** liquid (Supplied in 25 mM Tris-HCl pH 7.5, 150 mM NaCl, 5 mM MgCl<sub>2</sub> and 5 mM beta-mercaptoethanol)

**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). The mutation G12V leads to elimination of the intrinsic GTPase activity. H-Ras (G12V) is effective in activation of PI3K and PKB, whereas N-Ras and K-Ras are more potent towards MAP kinase. The GST-Tag facilitates the protein's application in typical GST pull-down assays.

**Selected References:**

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.

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  $\gamma$ . *Cell* **103**:931.

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