# MUTYH (m): 293T Lysate: sc-125671



The Power to Question

#### **BACKGROUND**

MUTYH (mutY homolog (E. coli)) is a DNA glycosylase mismatch repair enzyme that in conjunction with mutM (OGG1), cleaves adenine residues paired with either oxidized (8-hydroxyguanines) or non-modified guanines in order to correct A/G and A/C mismatches. Repair of most modified and mispaired bases in the genome is initiated by DNA glycosylases, which bind and cleave N-glycosyl bonds to initiate base excision repair. MUTYH is crucial for the avoidance of mutations resulting from oxidative DNA damage. Multiple N-terminal splice variants of MUTYH exist in mammalian cells. Increasing levels of MUTYH in A549 cells exposed to oxygen and infrared radiation leads to improvements in cell survival. Biallelic MUTYH germ-line mutations predispose humans to colorectal adenomas and carcinomas. MUTYH is abundant in neurons where mitochondrial genomes exposed to reactive oxygen species (ROS) that damage DNA must maintain integrity over the entire mammalian life span.

# **REFERENCES**

- Hayashi, H., et al. 2002. Replication-associated repair of adenine:8-oxoguanine mispairs by MYH. Curr. Biol. 12: 335-339.
- Englander, E.W., et al. 2002. Rat MYH, a glycosylase for repair of oxidatively damaged DNA, has brain-specific isoforms that localize to neuronal mitochondria. J. Neurochem. 83: 1471-1480.
- Halford, S.E., et al. 2003. Germline mutations but not somatic changes at the MYH locus contribute to the pathogenesis of unselected colorectal cancers. Am. J. Pathol. 162: 1545-1548.
- Lee, H.M., et al. 2004. Developmental changes in expression and subcellular localization of the DNA repair glycosylase, MYH, in the rat brain.
   Neurochem. 88: 394-400.
- Tao, H., et al. 2004. A novel splice-site variant of the base excision repair gene MYH is associated with production of an aberrant mRNA transcript encoding a truncated MYH protein not localized in the nucleus. Carcinogenesis 25: 1859-1866.
- 6. Kim, C.J., et al. 2004. Genetic alterations of the MYH gene in gastric cancer. Oncogene 23: 6820-6822.
- Ma, H., et al. 2004. N-terminus of the rat adenine glycosylase MYH
  affects excision rates and processing of MYH-generated abasic sites.
  Nucleic Acids Res. 32: 4332-4339.
- 8. Sieber, O.M., et al. 2004. Myh deficiency enhances intestinal tumorigenesis in multiple intestinal neoplasia (ApcMin/+) mice. Cancer Res. 64: 8876-8881.
- 9. Cardoso, J., et al. 2006. Chromosomal instability in MYH- and APC-mutant adenomatous polyps. Cancer Res. 66: 2514-2519.

# **STORAGE**

Store at -20° C. Repeated freezing and thawing should be minimized. Sample vial should be boiled once prior to use. Non-hazardous. No MSDS required.

## **PROTOCOLS**

See our web site at www.scbt.com for detailed protocols and support products.

#### **CHROMOSOMAL LOCATION**

Genetic locus: Mutyh (mouse) mapping to 4 C7.

### **PRODUCT**

MUTYH (m): 293T Lysate represents a lysate of mouse MUTYH transfected 293T cells and is provided as 100 µg protein in 200 µl SDS-PAGE buffer.

#### **APPLICATIONS**

MUTYH (m): 293T Lysate is suitable as a Western Blotting positive control for mouse reactive MUTYH antibodies. Recommended use:  $10-20~\mu l$  per lane.

Control 293T Lysate: sc-117752 is available as a Western Blotting negative control lysate derived from non-transfected 293T cells.

## **RESEARCH USE**

For research use only, not for use in diagnostic procedures.

Santa Cruz Biotechnology, Inc. 1.800.457.3801 831.457.3800 fax 831.457.3801 Europe +00800 4573 8000 49 6221 4503 0 www.scbt.com