

**HPRT1 Antibody**  
**Purified Mouse Monoclonal Antibody**  
**Catalog # AO1947a**

**Specification**

**HPRT1 Antibody - Product Information**

Application	<b>E, WB, FC, IHC</b>
Primary Accession	<a href="#">P00492</a>
Reactivity	<b>Human</b>
Host	<b>Mouse</b>
Clonality	<b>Monoclonal</b>
Isotype	<b>IgG1</b>
Calculated MW	<b>24.6kDa KDa</b>

**Description**

The protein encoded by this gene is a transferase, which catalyzes conversion of hypoxanthine to inosine monophosphate and guanine to guanosine monophosphate via transfer of the 5-phosphoribosyl group from 5-phosphoribosyl 1-pyrophosphate. This enzyme plays a central role in the generation of purine nucleotides through the purine salvage pathway. Mutations in this gene result in Lesch-Nyhan syndrome or gout.

**Immunogen**

Purified recombinant fragment of human HPRT1 (AA: FULL(1-218)) expressed in E. Coli.

**Formulation**

Purified antibody in PBS with 0.05% sodium azide.

**HPRT1 Antibody - Additional Information**

**Gene ID** 3251

**Other Names**

Hypoxanthine-guanine phosphoribosyltransferase, HGPRT, HGPRTase, 2.4.2.8, HPRT1, HPRT

**Dilution**

E~~1/10000  
WB~~1/500 - 1/2000  
FC~~1/200 - 1/400  
IHC~~1/200 - 1/1000

**Storage**

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

**Precautions**

HPRT1 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

**HPRT1 Antibody - Protein Information**

**Name** HPRT1

**Synonyms** HPRT

**Function**

Converts guanine to guanosine monophosphate, and hypoxanthine to inosine monophosphate. Transfers the 5-phosphoribosyl group from 5- phosphoribosylpyrophosphate onto the purine. Plays a central role in the generation of purine nucleotides through the purine salvage pathway.

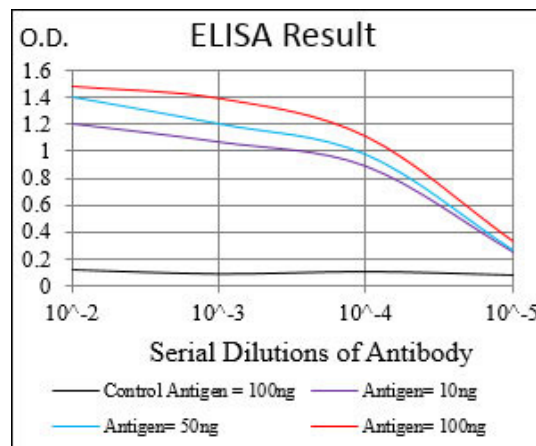
**Cellular Location**

Cytoplasm.

**HPRT1 Antibody - Protocols**

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)



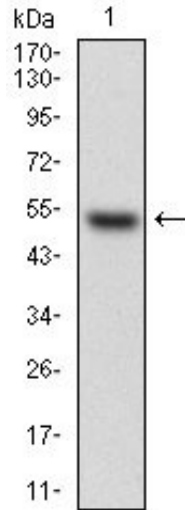


Figure 1: Western blot analysis using HPRT1 mAb against human HPRT1 (AA: FULL(1-218)) recombinant protein. (Expected MW is 50.5 kDa)

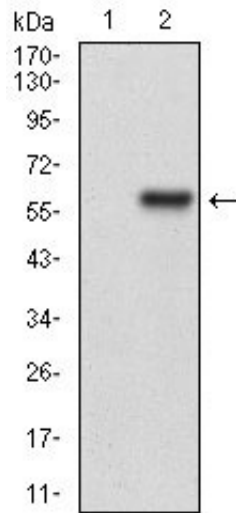


Figure 2: Western blot analysis using HPRT1 mAb against HEK293 (1) and HPRT1 (AA: FULL(1-218))-hlgGfC transfected HEK293 (2) cell lysate.

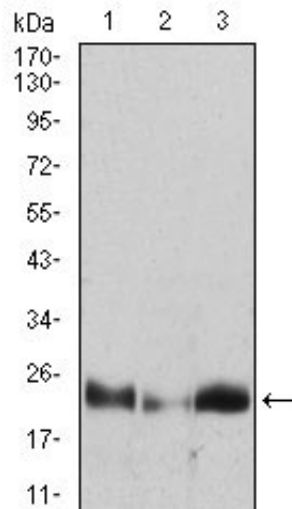


Figure 3: Western blot analysis using HPRT1 mouse mAb against HeLa (1), A431 (2), A549 (3) cell lysate.

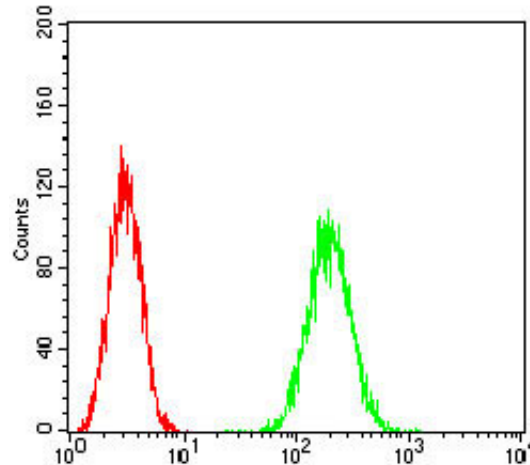


Figure 4: Flow cytometric analysis of HeLa cells using HPRT1 mouse mAb (green) and negative control (red).

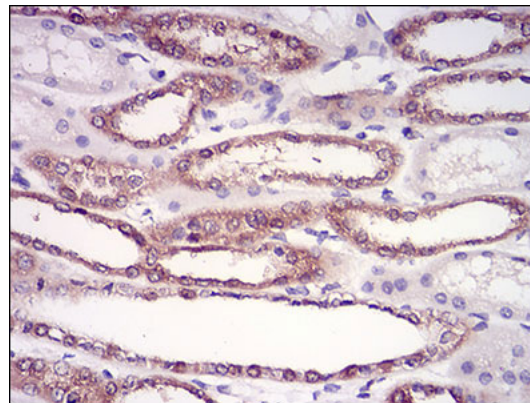


Figure 5: Immunohistochemical analysis of paraffin-embedded kidney tissues using HPRT1 mouse mAb with DAB staining.

### HPRT1 Antibody - Background

C17orf53 (chromosome 17 open reading frame 53) is a 647 amino acid protein that is encoded by a gene mapping to human chromosome 17. Chromosome 17 makes up over 2.5% of the human genome with about 81 million bases encoding over 1,200 genes. Two key tumor suppressor genes are associated with chromosome 17, namely, p53 and BRCA1. Tumor suppressor p53 is necessary for maintenance of cellular genetic integrity by moderating cell fate through DNA repair versus cell death. Malfunction or loss of p53 expression is associated with malignant cell growth and Li-Fraumeni syndrome. Like p53, BRCA1 is directly involved in DNA repair, specifically it is recognized as a genetic determinant of early onset breast cancer and predisposition to cancers of the ovary, colon, prostate gland and fallopian tubes. Chromosome 17 is also linked to neurofibromatosis, a condition characterized by neural and epidermal lesions, and dysregulated Schwann cell growth. Alexander disease, Birt-Hogg-Dube syndrome and Canavan disease are also associated with chromosome 17. ; ;

### HPRT1 Antibody - References

1. Nucleosides Nucleotides Nucleic Acids. 2011 Dec;30(12):1248-55.2. Mol Ther. 2010 Jan;18(1):54-62.