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# GLRX2 Antibody (C-term T135)

**Purified Rabbit Polyclonal Antibody (Pab)**
**Catalog # AP7301B**

## Specification

### GLRX2 Antibody (C-term T135) - Product Information

| | |
|---|---|
| Application | **FC, IHC-P, WB,E** |
| Primary Accession | Q9NS18 |
| Other Accession | Q32L67 |
| Reactivity | **Human, Mouse, Rat** |
| Predicted | **Bovine** |
| Host | **Rabbit** |
| Clonality | **Polyclonal** |
| Isotype | **Rabbit IgG** |
| Antigen Region | **120-146** |

### GLRX2 Antibody (C-term T135) - Additional Information

**Gene ID** 51022

**Other Names**
Glutaredoxin-2, mitochondrial, GLRX2, GRX2

**Target/Specificity**
This GLRX2 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 120-146 amino acids from the C-terminal region of human GLRX2.

**Dilution**
FC~~1:10~50
IHC-P~~1:50~100
WB~~1:1000
E~~Use at an assay dependent concentration.

**Format**
Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

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

**Precautions**
GLRX2 Antibody (C-term T135) is for research use only and not for use in diagnostic or therapeutic procedures.

### GLRX2 Antibody (C-term T135) - Protein Information

**Name** GLRX2

**Synonyms** GRX2

**Function** Glutathione-dependent oxidoreductase that facilitates the maintenance of mitochondrial redox homeostasis upon induction of apoptosis by oxidative stress. Involved in response to hydrogen peroxide and regulation of apoptosis caused by oxidative stress. Acts as a very efficient catalyst of monothiol reactions because of its high affinity for protein glutathione-mixed disulfides. Can receive electrons not only from glutathione (GSH), but also from thioredoxin reductase supporting both monothiol and dithiol reactions. Efficiently catalyzes both glutathionylation and deglutathionylation of mitochondrial complex I, which in turn regulates the superoxide production by the complex. Overexpression decreases the susceptibility to apoptosis and prevents loss of cardiolipin and cytochrome c release.

**Cellular Location**
[Isoform 1]: Mitochondrion.

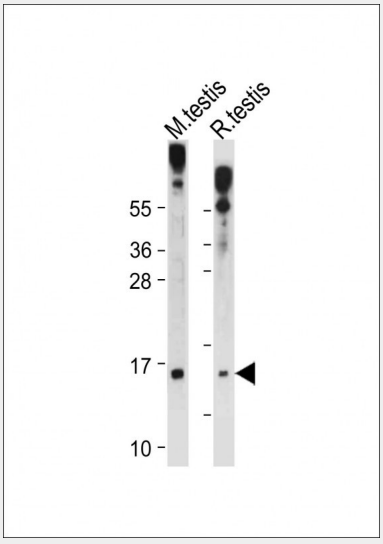
**Tissue Location**
Widely expressed. Expressed in brain, heart, skeletal muscle, colon, thymus, spleen, kidney, liver, small intestine, placenta and lung. Not expressed in peripheral blood leukocytes

## GLRX2 Antibody (C-term T135) - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

## GLRX2 Antibody (C-term T135) - Images




All lanes : Anti-GLRX2 Antibody (C-term T135) at 1:1000 dilution Lane 1: Mouse testis tissue lysate Lane 2: Rat testis tissue lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 18 kDa

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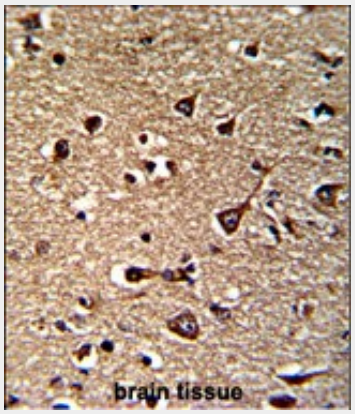
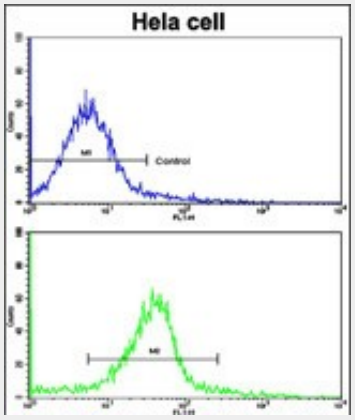
Blocking/Dilution buffer: 5% NFDM/TBST.




Formalin-fixed and paraffin-embedded human brain reacted with GLRX2 Antibody (C-term T135), which was peroxidase-conjugated to the secondary antibody, followed by DAB staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated.




Flow cytometric analysis of hela cells using GLRX2 Antibody (C-term T135)(bottom histogram) compared to a negative control cell (top histogram). FITC-conjugated goat-anti-rabbit secondary antibodies were used for the analysis.

**GLRX2 Antibody (C-term T135) - Background**

GLRX2 are a family of glutathione-dependent hydrogen donors that participate in a variety of cellular redox reactions.

**GLRX2 Antibody (C-term T135) - References**

Lundberg M., Johansson C. J. Biol. Chem. 276:26269-26275(2001)
Gladyshev V.N., Liu A. J. Biol. Chem. 276:30374-30380(2001)
Lillig C.H. Proc. Natl. Acad. Sci. U.S.A. 101:13227-13232(2004)