**Product Name: RSAD2 Rabbit Polyclonal Antibody**
**Catalog #: APRab17401**

## Summary

| | |
|---|---|
| **Production Name** | RSAD2 Rabbit Polyclonal Antibody |
| **Description** | Rabbit Polyclonal Antibody |
| **Host** | Rabbit |
| **Application** | WB,IHC, |
| **Reactivity** | Human,Rat,Mouse |

## Performance

| | |
|---|---|
| **Conjugation** | Unconjugated |
| **Modification** | Unmodified |
| **Isotype** | IgG |
| **Clonality** | Polyclonal |
| **Form** | Liquid |
| **Storage** | Store at 4°C short term. Aliquot and store at -20°C long term. Avoid freeze/thaw cycles. |
| **Buffer** | Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N. |
| **Purification** | Affinity purification |

## Immunogen

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|---|---|
| **Gene Name** | RSAD2 |
| **Alternative Names** | RSAD2; CIG5; Radical S-adenosyl methionine domain-containing protein 2; Cytomegalovirus-induced gene 5 protein; Viperin; Virus inhibitory protein, endoplasmic reticulum-associated, interferon-inducible |
| **Gene ID** | 91543.0 |
| **SwissProt ID** | Q8WXG1.The antiserum was produced against synthesized peptide derived from the N-terminal region of human RSAD2. AA range:21-70 |

## Application

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|---|---|
| **Dilution Ratio** | WB 1:500 - 1:2000. IHC-p: 1:100-1:300. ELISA: 1:20000.. |
| **Molecular Weight** | 42kD |

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## Background

cofactor:Binds 1 4Fe-4S cluster. The cluster is coordinated with 3 cysteines and an exchangeable S-adenosyl-L-methionine.,function:Involved in antiviral defense. May impair virus budding by disrupting lipid rafts at the plasma membrane, a feature which is essential for the budding process of many viruses. Acts through binding with and inactivating FPPS, an enzyme involved in synthesis of cholesterol, farnesylated and geranylated proteins, ubiquinones dolichol and heme. Plays a major role in the cell antiviral state induced by type I and type II interferon. Displays antiviral effect against HIV-1 virus, hepatitis C virus, human cytomegalovirus, and aphaviruses, but not vesiculovirus.,induction:By interferon type I, type II and LPS. Little or no induction by interferon gamma is observed in monocytic cell lines. Induced by infection with human cytomegalovirus (HMCV), hepatitis C virus, yellow fever virus and Sendai virus, presumably through type I interferon pathway.,miscellaneous:Up-regulated in atherosclerosis. Latent viruses like HCMV may be involved in atherogenesis by initiating local inflammation. This may induce up-regulation of antiviral gene RSAD2, which modulates lipids synthesis, and thus could play a role in abnormal lipid accumulation leading to atherosclerosis.,similarity:Belongs to the RSAD2 family.,subcellular location:Probably associates with the cytosolic side of the endoplasmic reticulum. Infection with human cytomegalovirus (HCMV) causes relocation to the Golgi apparatus and to cytoplasmic vacuoles which also contain HCMV proteins glycoprotein B and pp28.,subunit:Interacts with FPPS.,cofactor:Binds 1 4Fe-4S cluster. The cluster is coordinated with 3 cysteines and an exchangeable S-adenosyl-L-methionine.,function:Involved in antiviral defense. May impair virus budding by disrupting lipid rafts at the plasma membrane, a feature which is essential for the budding process of many viruses. Acts through binding with and inactivating FPPS, an enzyme involved in synthesis of cholesterol, farnesylated and geranylated proteins, ubiquinones dolichol and heme. Plays a major role in the cell antiviral state induced by type I and type II interferon. Displays antiviral effect against HIV-1 virus, hepatitis C virus, human cytomegalovirus, and aphaviruses, but not vesiculovirus.,induction:By interferon type I, type II and LPS. Little or no induction by interferon gamma is observed in monocytic cell lines. Induced by infection with human cytomegalovirus (HMCV), hepatitis C virus, yellow fever virus and Sendai virus, presumably through type I interferon pathway.,miscellaneous:Up-regulated in atherosclerosis. Latent viruses like HCMV may be involved in atherogenesis by initiating local inflammation. This may induce up-regulation of antiviral gene RSAD2, which modulates lipids synthesis, and thus could play a role in abnormal lipid accumulation leading to atherosclerosis.,similarity:Belongs to the RSAD2 family.,subcellular location:Probably associates with the cytosolic side of the endoplasmic reticulum. Infection with human cytomegalovirus (HCMV) causes relocation to the Golgi apparatus and to cytoplasmic vacuoles which also contain HCMV proteins glycoprotein B and pp28.,subunit:Interacts with FPPS.,

## Research Area

## Image Data

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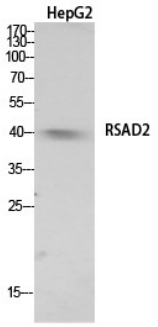
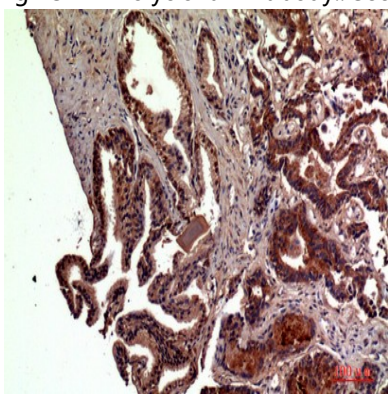
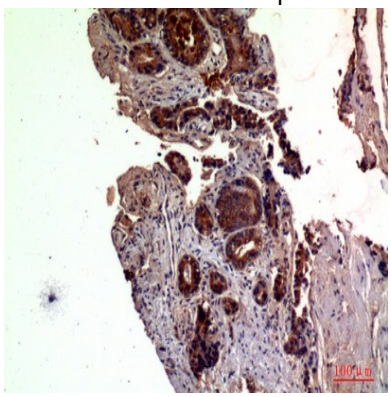


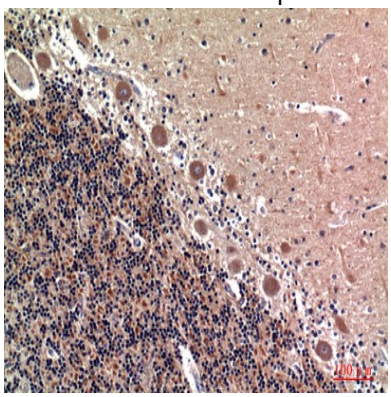

Western Blot analysis of HepG2 cells using RSAD2 Polyclonal Antibody.. Secondary antibody was diluted at 1:20000



Immunohistochemical analysis of paraffin-embedded human-prostate-cancer, antibody was diluted at 1:100



Immunohistochemical analysis of paraffin-embedded human-prostate-cancer, antibody was diluted at 1:100



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Immunohistochemical analysis of paraffin-embedded human-brain, antibody was diluted at 1:100

## Note

For research use only.