

RSAD2 Polyclonal Antibody

Cat No: HR1AP5375

For research use only

Overview

Product Name	RSAD2 Polyclonal Antibody
Source	Rabbit
Applications	WB,IHC-p,ELISA
Species Reactivity	Human
Recommended Dilutions	
Immunogen	
Species	Rabbit
Storage	-20°C/1 year
Isotype	
Clonality	
Concentration	1 mg/ml
Observed band	42kDa
GeneID?Human?	RSAD2
Human Swiss-Prot No.	
Cellular localization	
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-inducibl
Background	<p>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 (HCMV), 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.,</p>