

Product name:	RSAD2 Rabbit Polyclonal Antibody
Cat number:	ABN17401
Conjugate:	Unconjugated
Size:	100µL
Clone:	Polyclonal
Concentration:	1mg/ml
Host:	Rabbit
Isotype:	IgG
Immunogen:	The antiserum was produced against synthesized peptide derived from the N-terminal region of human RSAD2. AA range:21-70
Reactivity:	Human,Rat,Mouse
Applications:	WB 1:500-1:2000,IHC 1:100-1:300,ICC/IF 1:50-1:200,ELISA 1:10000-1:20000
Molecular Weight:	42kDa
Purification:	Affinity purification
Form:	Liquid
Buffer:	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N.
Storage:	Store at 4°C short term. Aliquot and store at -20°C for 12 months. Avoid freeze/thaw cycles.

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 (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.,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.,