

**bs-11940R****[ Primary Antibody ]****RAI1 Rabbit pAb**

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**— DATASHEET —**

<b>Host:</b> Rabbit	<b>Isotype:</b> IgG	<b>Applications:</b> <b>WB</b> (1:500-2000) <b>IHC-P</b> (1:100-500) <b>IHC-F</b> (1:100-500) <b>IF</b> (1:100-500) <b>ICC/IF</b> (1:100-500) <b>ELISA</b> (1:5000-10000)  <b>Reactivity:</b> (predicted: Human, Mouse, Rat)  <b>Predicted MW.:</b> 203 kDa  <b>Subcellular Location:</b> Cytoplasm ,Nucleus
<b>Clonality:</b> Polyclonal		
<b>GeneID:</b> 10743	<b>SWISS:</b> Q7Z5J4	
<b>Target:</b> RAI1		
<b>Immunogen:</b> KLH conjugated synthetic peptide derived from human RAI1: 421-520/1906.		
<b>Purification:</b> affinity purified by Protein A		
<b>Concentration:</b> 1mg/ml		
<b>Storage:</b> 0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol. Shipped at 4°C. Store at -20°C for one year. Avoid repeated freeze/thaw cycles.		
<b>Background:</b> Retinoic acid induced 1 (RAI1) is a 1,906 amino acid protein containing an N-terminal polyglutamine stretch that is expressed in most tissues, with highest expression in neuronal tissues. RAI1 functions as a transcriptional regulator and is important for embryonic and postnatal developments. Heterozygous deletions of the RAI1 gene are associated with Smith-Magenis syndrome (SMS), a mental retardation syndrome with behavioral, neurological and skeletal anomalies. Individuals affected with SMS usually display self-injurious behaviors, sleep disturbance, developmental delay and reduced motor and cognitive skills. RAI1 haploinsufficiency is specifically responsible for the obesity and craniofacial symptoms of SMS. RAI1 mutations have also been implicated in schizophrenia and spinocerebellar ataxia type 2.		