

**bs-18639R****[ Primary Antibody ]****Malectin/MLEC Rabbit pAb**

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**— DATASHEET —**

<b>Host:</b> Rabbit	<b>Isotype:</b> IgG	<b>Applications:</b> <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, Rabbit, Pig, Sheep, Cow, Chicken, Dog, Horse, Turkey)  <b>Predicted MW.:</b> 29 kDa  <b>Subcellular Location:</b> Cell membrane
<b>Clonality:</b> Polyclonal		
<b>GeneID:</b> 9761	<b>SWISS:</b> Q14165	
<b>Target:</b> Malectin/MLEC		
<b>Immunogen:</b> KLH conjugated synthetic peptide derived from human Malectin/MLEC: 201-292/292.		
<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> MLEC is a 292 amino acid single-pass type I membrane protein of the endoplasmic reticulum that belongs to the malectin family and is thought to play a role in N-glycosylation. MLEC may function as a carbohydrate-binding protein that preferentially binds Glc2-N-glycan. The gene encoding MLEC maps to human chromosome 12, which makes up about 4.5% of the human genome. A number of skeletal deformities are linked to chromosome 12, including hypochondrogenesis, achondrogenesis and Kniest dysplasia. Noonan syndrome, which includes heart and facial developmental defects among the primary symptoms, is caused by a mutant form of PTPN11 gene product, SH-PTP2. Chromosome 12 is also home to a homeobox gene cluster, which encodes crucial transcription factors for morphogenesis, and the natural killer complex gene cluster, encoding C-type lectin proteins which mediate the NK cell response to MHC I interaction. Trisomy 12p leads to facial development defects, seizure disorders and a host of other symptoms which vary in severity depending on the extent of mosaicism. It is most severe in cases of complete trisomy.		