

# Malectin/MLEC Rabbit pAb

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Catalog # AP57193

## Product Information

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<b>Application</b>	IHC-P, IHC-F, IF, E
<b>Primary Accession</b>	<a href="#">Q14165</a>
<b>Predicted</b>	
<b>Host</b>	Human, Mouse, Rat, Chicken, Dog, Pig, Horse, Rabbit, Sheep, Turkey
<b>Clonality</b>	Rabbit
<b>Calculated MW</b>	Polyclonal
<b>Physical State</b>	32234
<b>Immunogen</b>	Liquid
<b>Epitope Specificity</b>	KLH conjugated synthetic peptide derived from human Malectin/MLEC
<b>Isotype</b>	201-292/292
<b>Purity</b>	IgG
	affinity purified by Protein A
<b>Buffer</b>	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
<b>SUBCELLULAR LOCATION</b>	Endoplasmic reticulum membrane.
<b>SIMILARITY</b>	Belongs to the malectin family.
<b>Important Note</b>	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
<b>Background Descriptions</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.

## Additional Information

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<b>Gene ID</b>	9761
<b>Other Names</b>	Malectin, MLEC ( <a href="#">HGNC:28973</a> ), KIAA0152
<b>Dilution</b>	IHC-P=1:100-500,IHC-F=1:100-500,ICC/IF=1:100-500,IF=1:100-500,ELISA=1:500 0-10000

<b>Storage</b>	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
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## Protein Information

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<b>Name</b>	MLEC ( <a href="#">HGNC:28973</a> )
<b>Synonyms</b>	KIAA0152
<b>Function</b>	Carbohydrate-binding protein with a strong ligand preference for Glc2-N-glycan. May play a role in the early steps of protein N- glycosylation (By similarity).
<b>Cellular Location</b>	Endoplasmic reticulum membrane; Single-pass type I membrane protein

## Background

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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.

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.