Data Sheet (Cat.No.TMAH-01001)

Anti-PRKAG1 Antibody (6O628)

Product Details

Ig Type:	Rabbit IgG
Reactivity:	Human
Conjugation:	Unconjugated
Clone:	6O628
Purification:	Affinity-chromatography

Applications

Verified Activity:	IHC image of TMAH-01001 diluted at 1:100 and staining in paraffin-embedded human Prostate cancer performed on a Leica BondTM system. After dewaxing and hydration, antigen retrieval was mediated by high pressure in a citrate buffer (pH 6.0). Section was blocked with 10% normal goat serum 30min at RT. Then primary antibody (1% BSA) was incubated at 4°C overnight. The primary is detected by a Goat anti-rabbit polymer IgG labeled by HRP and visualized using 0.05% DAB.
Application:	ELISA, IHC
Recommended	IHC:1:50-1:200.

Properties

Stability & Storage:	Store at -20°C or -80°C for 12 months. Avoid repeated freeze-thaw cycles.
Shipping:	Shipping with blue ice.

Antigen Details

Immunogen:	A synthetic peptide: Human PRKAG1
Antigen Species:	Human
Gene ID:	5571
Uniprot ID:	P54619
Synonyms:	AMPKg;5'-AMP-activated protein kinase subunit gamma-1;AMPK subunit gamma-1;AMPK gamma1
Biology Area:	Cancer, Cardiovascular, Metabolism, Signal transduction

Research Background

AMP/ATP-binding subunit of AMP-activated protein kinase (AMPK), an energy sensor protein kinase that plays a key role in regulating cellular energy metabolism. In response to reduction of intracellular ATP levels, AMPK activates energy-producing pathways and inhibits energy-consuming processes: inhibits protein, carbohydrate and lipid biosynthesis, as well as cell growth and proliferation. AMPK acts via direct phosphorylation of metabolic enzymes, and by longer-term effects via phosphorylation of transcription regulators. Also acts as a regulator of cellular polarity by remodeling the actin cytoskeleton; probably by indirectly activating myosin. Gamma non-catalytic subunit mediates binding to AMP, ADP and ATP, leading to activate or inhibit AMPK: AMP-binding results in allosteric activation of alpha catalytic subunit (PRKAA1 or PRKAA2) both by inducing phosphorylation and preventing dephosphorylation of catalytic subunits. ADP also stimulates phosphorylation, without stimulating already

phosphorylated catalytic subunit. ATP promotes dephosphorylation of catalytic subunit, rendering the AMPK enzyme inactive.

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