GNAS2 Rabbit pAb

CatalogNo: YT6670

Key Features

Host Species
- Rabbit

Reactivity
- Human,Mouse,Rat

Applications
- WB

MW
- 43kD (Calculated)

Isotype
- IgG

Recommended Dilution Ratios

WB 1:500-2000

Storage

Storage*	-15°C to -25°C/1 year(Do not lower than -25°C)
Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.

Basic Information

Clonality	Polyclonal

Immunogen Information

Immunogen	Synthesized peptide derived from human GNAS2 AA range: 137-187
Specificity	This antibody detects endogenous levels of GNAS2 at Human/Mouse/Rat

Target Information

Gene name	GNAS GNAS1 GSP

Protein Name GNAS2

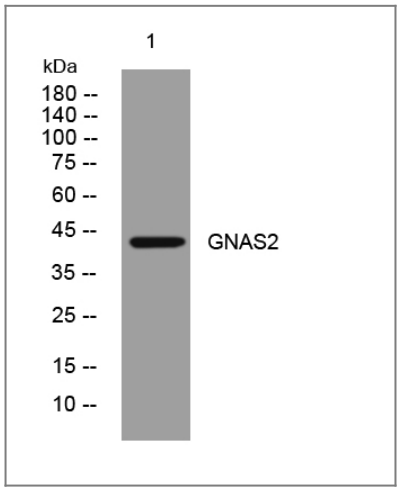
Organism	Gene ID	UniProt ID
Human	2778;	P63092;
Mouse	14683;	P63094;
Rat	24896;	P63095;

Cellular Localization Cell membrane ; Lipid-anchor .

Function

Caution:The sequence shown here is derived from an Ensembl automatic analysis pipeline and should be considered as preliminary data.,Disease:Defects in GNAS are a cause of ACTH-independent macronodular adrenal hyperplasia (AIMAH) [MIM:219080]; also known as adrenal Cushing syndrome due to AIMAH. AIMAH is an endogenous form of adrenal Cushing syndrome characterized by multiple bilateral adrenocortical nodules that cause a striking enlargement of the adrenal glands.,Disease:Defects in GNAS are the cause of a subset of growth hormone secreting pituitary tumors (somatotrophinoma) [MIM:102200].,Disease:Defects in GNAS are the cause of Albright hereditary osteodystrophy (AHO) [MIM:103580]. AHO is an autosomal dominant disorder characterized by a short stature, brachydactyly, subcutaneous ossifications. AHO is often associated with pseudohypoparathyoidism, hypocalcemia, and elevated PTH levels. The expression or the activity of GNAS is reduced in AHO.,Disease:Defects in GNAS are the cause of GNAS hyperfunction [MIM:139320]. This condition is characterized by increased trauma-related bleeding tendency, prolonged bleeding time, brachydactyly and mental retardation. Both the XLas isoforms and the ALEX protein are mutated which strongly reduces the interaction between them and this may allow unimpeded activation of the XLas isoforms.,Disease:Defects in GNAS are the cause of McCune-Albright syndrome (MAS) [MIM:174800]. MAS is characterized by polyostotic fibrous dysplasia, cafe-au-lait lesions, and a variety of endocrine disorders, including precocious puberty, hyperthyroidism, hypercortisolism, growth hormone excess, and hyperprolactinemia. The mutations producing MAS lead to constitutive activation of GS alpha.,Disease:Defects in GNAS are the cause of progressive osseous heteroplasia (POH) [MIM:166350]. POH is a rare autosomal dominant disorder characterized by extensive dermal ossification during childhood, followed by disabling and widespread heterotopic ossification of skeletal muscle and deep connective tissue.,Disease:Defects in GNAS are the cause of pseudohypoparathyroidism type 1A (PHP1A) [MIM:103580]. Pseudohypoparathyroidism is a term applied to a heterogeneous group of disorders whose common feature is resistance to parathyroid hormone.,Disease:Defects in GNAS may be a cause of colorectal cancer (CRC) [MIM:114500].,Disease:Genetic variations in GNAS are the cause of pseudohypoparathyroidism type 1B (PHP1B) [MIM:603233]. PHP1B is characterized by parathyroid hormone (PTH)-resistant hypocalcemia and hyperphosphatemia. Patients affected with PHP1B have normal activity of the product of GNAS, lack developmental defects characteristic of AHO, and typically show no other endocrine abnormalities besides resistance to PTH. Most affected individuals have defects in methylation of the gene. In some cases microdeletions involving the STX16 appear to cause loss of methylation at exon A/B of GNAS, resulting in PHP1B. Paternal uniparental isodisomy have also been observed.,Function:Guanine nucleotide-binding proteins (G proteins) are involved as modulators or transducers in various transmembrane signaling systems. The G(s) protein is involved in hormonal regulation of adenylate cyclase: it activates the cyclase in response to beta-adrenergic stimuli.,Function:Guanine nucleotide-binding proteins (G proteins) are involved as modulators or transducers in various transmembrane signaling systems. The G(s) protein is involved in hormonal regulation of adenylate cyclase: it activates the cyclase in response to beta-adrenergic stimuli. XLas isoforms interact with the same set of receptors as Gnas isoforms.,Function:May inhibit the adenylyl cyclase-stimulating activity of guanine nucleotide-binding protein G(s) subunit alpha which is produced from the same locus in a different open reading frame.,miscellaneous:The GNAS locus is imprinted in a complex manner, giving rise to distinct paternally, maternally and biallelically expressed proteins. The XLas isoforms are paternally derived, the Gnas isoforms are biallelically derived and the Nesp55 isoforms are maternally derived.,miscellaneous:This protein is produced by a bicistronic gene which also produces guanine nucleotide-binding protein G(s) subunit alpha from an overlapping reading frame.,miscellaneous:This protein is produced by a bicistronic gene which also produces the ALEX protein from an overlapping reading frame.,PTM:Binds keratan sulfate chains.,PTM:May be proteolytically processed to give rise to a number of active peptides.,similarity:Belongs to the ALEX family.,similarity:Belongs to the G-alpha family. G(s) subfamily.,similarity:Belongs to the NESP55 family.,subcellular location:Neuroendocrine secretory granules.,subcellular location:Predominantly associated with cell membrane ruffles.,subunit:G proteins are composed of 3 units; alpha, beta and gamma. The alpha chain contains the guanine nucleotide binding site.,subunit:G proteins are composed of 3 units; alpha, beta and gamma. The alpha chain contains the guanine nucleotide binding site. Interacts through its N-terminal region with ALEX which is produced from the same locus in a different open reading frame. This interaction may inhibit its adenylyl cyclase-stimulating activity.,subunit:Interacts with the N-terminal region of the XLas isoforms of guanine nucleotide-binding protein G(s) subunit alpha.,

Validation Data



Western blot analysis of lysates from Jurkat cells, primary antibody was diluted at 1:1000, 4°over night

Contact information

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