



# GNAS2 rabbit pAb

<b>Catalog No</b>	YP-Ab-11305
<b>Isotype</b>	IgG
<b>Reactivity</b>	Human; Mouse;Rat
<b>Applications</b>	WB
<b>Gene Name</b>	GNAS GNAS1 GSP
<b>Protein Name</b>	GNAS2
<b>Immunogen</b>	Synthesized peptide derived from human GNAS2 AA range: 137-187
<b>Specificity</b>	This antibody detects endogenous levels of GNAS2 at Human/Mouse/Rat
<b>Formulation</b>	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
<b>Source</b>	Polyclonal, Rabbit,IgG
<b>Purification</b>	The antibody was affinity-purified from rabbit serum by affinity-chromatography using specific immunogen.
<b>Dilution</b>	WB 1: 500-2000
<b>Concentration</b>	1 mg/ml
<b>Purity</b>	≥90%
<b>Storage Stability</b>	-20°C/1 year
<b>Synonyms</b>	
<b>Observed Band</b>	
<b>Cell Pathway</b>	Cell membrane ; Lipid-anchor .
<b>Tissue Specificity</b>	
<b>Function</b>	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 pseudohypoparathyroidism, hypocalcemia, and elevated PTH levels.
<b>Background</b>	This locus has a highly complex imprinted expression pattern. It gives rise to maternally, paternally, and biallelically expressed transcripts that are derived from four alternative promoters and 5' exons. Some transcripts contain a



differentially methylated region (DMR) at their 5' exons, and this DMR is commonly found in imprinted genes and correlates with transcript expression. An antisense transcript is produced from an overlapping locus on the opposite strand. One of the transcripts produced from this locus, and the antisense transcript, are paternally expressed noncoding RNAs, and may regulate imprinting in this region. In addition, one of the transcripts contains a second overlapping ORF, which encodes a structurally unrelated protein - Alex. Alternative splicing of downstream exons is also observed, which results in different forms of the stimulatory G-protein alpha subunit, a key element of the classical signal transduction pathway linking receptor-ligand interactions with the activation of adenylyl cyclase and a variety of cellular responses. Multiple transcript variants encoding different isoforms have been found for this gene. Mutations in this gene result in pseudohypoparathyroidism type 1a, pseudohypoparathyroidism type 1b, Albright hereditary osteodystrophy, pseudopseudohypoparathyroidism, McCune-Albright syndrome, progressive osseus heteroplasia, polyostotic fibrous dysplasia of bone, and some pituitary tumors. [provided by RefSeq, Aug 2012],

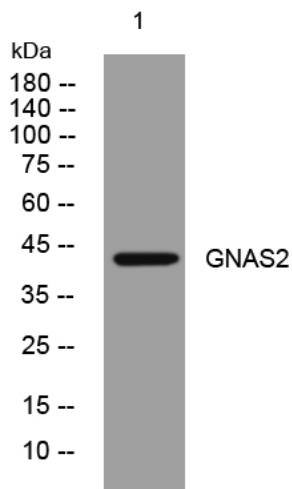
**matters needing attention**

Avoid repeated freezing and thawing!

**Usage suggestions**

This product can be used in immunological reaction related experiments. For more information, please consult technical personnel.

## Products Images



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