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Rabbit Anti-G protein alpha S/NESP55/FITC Conjugated antibody

SL3939R-FITC

Product Name:	Anti-G protein alpha S/NESP55/FITC
Chinese Name:	FITC标记的G蛋白αS抗体（鸟嘌呤核苷酸Binding proteinGα s）
Alias:	Adenylate cyclase stimulating G alpha protein; AHO; Alternative gene product encoded by XL exon; Extra large alphas protein; GNAS; GNAS complex locus; GNAS1; GPSA; Gs alpha subunit; GSA; GSP; Guanine nucleotide binding protein (G protein) alpha stimulating activity polypeptide 1; Guanine nucleotide binding protein alpha stimulating activity polypeptide 1; Guanine nucleotide binding protein G(s) subunit alpha isoforms short; Guanine nucleotide binding protein G(s) subunit alpha isoforms XLas; Guanine nucleotide regulatory protein; MGC33735; NESP; NESP55; Neuroendocrine secretory protein; PHP1A; PHP1B; POH; Protein ALEX; SCG6; Secretogranin VI; XLalphas; XLas; C20orf45; dJ309F20.1.1; dJ806M20.3.3; GNAS1_HUMAN.
Organism Species:	Rabbit
Clonality:	Polyclonal
React Species:	Human,Mouse,Rat,Chicken,Dog,Pig,Cow,Horse,Rabbit,Sheep,Danio rerio
Applications:	IF=1:50-200 not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Molecular weight:	111kDa
Cellular localization:	The cell membrane
Form:	Lyophilized or Liquid
Concentration:	1mg/ml
immunogen:	KLH conjugated synthetic peptide derived from human G protein alpha S
Lsotype:	IgG
Purification:	affinity purified by Protein A
Storage Buffer:	0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.
Storage:	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. 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.
Product Detail:	**background:** Guanine nucleotide-binding proteins (G proteins) are involved as modulators or transducers in various transmembrane signaling systems. The Gs 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 (By similarity). **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 (By similarity). **Subcellular Location:** Cell membrane; Peripheral membrane protein. **DISEASE:** Defects in GNAS are the cause of GNAS hyperfunction (GNASHYP) [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. Defects in GNAS are a cause of ACTH-independent macronodular adrenal hyperplasia (AIMAH) [MIM:219080]; also known as adrenal Cushing syndrome due to AIMAH. A rare adrenal defect characterized by multiple, bilateral, non-pigmented, benign, adrenocortical nodules. It results in excessive production of cortisol leading to ACTH-independent Cushing syndrome. Clinical manifestations of Cushing syndrome include facial and trunkal obesity, abdominal striae, muscular weakness, osteoporosis, arterial hypertension, diabetes. 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. Note=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. Defects in GNAS are the cause of pseudohypoparathyroidism type 1C (PHP1C) [MIM:612462]. It is a disorder characterized by end-organ resistance to parathyroid

hormone, hypocalcemia and hyperphosphatemia. It is commonly associated with Albright hereditary osteodystrophy whose features are short stature, obesity, round facies, short metacarpals and ectopic calcification.

Similarity:
Belongs to the G-alpha family. G(s) subfamily. membrane protein.

Database links:

Entrez Gene: 281793 Cow

Entrez Gene: 2778 Human

Entrez Gene: 14683 Mouse

Entrez Gene: 100049657 Pig

Entrez Gene: 24896 Rat

Omim: 139320 Human

SwissProt: P04896 Cow

SwissProt: P63091 Dog

SwissProt: P63092 Human

SwissProt: P84996 Human

SwissProt: Q5JWF2 Human

SwissProt: P63094 Mouse

SwissProt: Q6R0H7 Mouse

SwissProt: P29797 Pig

SwissProt: P63095 Rat

SwissProt: Q63803 Rat

Unigene: 125898 Human

Important Note:
This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.