

Immunotag™ TRβ1 (phospho Ser142) Polyclonal Antibody

Antibody Specification	
Catalog No.	ITP0479
Product Description	Immunotag™ TRβ1 (phospho Ser142) Polyclonal Antibody
Size	50 µg, 100 µg
Conjugation	HRP, Biotin, FITC, Alexa Fluor® 350, Alexa Fluor® 405, Alexa Fluor® 488, Alexa Fluor® 555, Alexa Fluor® 594, Alexa Fluor® 647
IMPORTANT NOTE	This product is custom manufactured with a lead time of 3-4 weeks. Once in production, this item cannot be cancelled from an order and is not eligible for return.
Target Protein	TRβ1 (Ser142)
Clonality	Polyclonal
Storage/Stability	-20°C/1 year
Application	WB,ELISA
Recommended Dilution	Western Blot: 1/500 - 1/2000. ELISA: 1/5000. Not yet tested in other applications.
Concentration	1 mg/ml
Reactive Species	Human,Mouse,Rat
Host Species	Rabbit
Immunogen	Synthesized phospho-peptide around the phosphorylation site of human TRβ1 (phospho Ser142)
Specificity	Phospho-TRβ1 (S142) Polyclonal Antibody detects endogenous levels of TRβ1 protein only when phosphorylated at S142.
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen
Form	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Gene Name	THRB
Accession No.	P10828 P37242 P18113
Alternate Names	THRB; ERBA2; NR1A2; THR1; Thyroid hormone receptor beta; Nuclear receptor subfamily 1 group A member 2; c-erbA-2; c-erbA-beta

Antibody Specification

Description	thyroid hormone receptor beta(THRB) Homo sapiens The protein encoded by this gene is a nuclear hormone receptor for triiodothyronine. It is one of the several receptors for thyroid hormone, and has been shown to mediate the biological activities of thyroid hormone. Knockout studies in mice suggest that the different receptors, while having certain extent of redundancy, may mediate different functions of thyroid hormone. Mutations in this gene are known to be a cause of generalized thyroid hormone resistance (GTHR), a syndrome characterized by goiter and high levels of circulating thyroid hormone (T3-T4), with normal or slightly elevated thyroid stimulating hormone (TSH). Several alternatively spliced transcript variants encoding the same protein have been observed for this gene. [provided by RefSeq, Jul 2008],
Cell Pathway/ Category	Neuroactive ligand-receptor interaction,
Protein Expression	Brain,Kidney,Pituitary,Placenta,Testis,
Subcellular Localization	nuclear chromatin,nucleus,nucleoplasm,extracellular exosome,
Protein Function	disease:Defects in THRB are the cause of generalized thyroid hormone resistance (GTHR) [MIM:188570, 274300]. GTHR is transmitted as an autosomal dominant trait, but an autosomal recessive form also exists. The disease is characterized by goiter, abnormal mental functions, increased susceptibility to infections, abnormal growth and bone maturation, tachycardia and deafness. Affected individuals may also have attention deficit-hyperactivity disorders (ADHD) and language difficulties. GTHR patients also have high levels of circulating thyroid hormones (T3-T4), with normal or slightly elevated thyroid stimulating hormone (TSH).,disease:Defects in THRB are the cause of selective pituitary thyroid hormone resistance (PRTH) [MIM:145650]; also called familial hyperthyroidism due to inappropriate thyrotropin secretion. PRTH is a variant form of thyroid hormone resistance and is characterized by clinical hyperthyroidism, with elevated free thyroid hormones, but inappropriately normal serum TSH. Unlike GRTH, where the syndrome usually segregates with a dominant allele, the mode of inheritance in PRTH has not been established.,domain:Composed of three domains: a modulating N-terminal domain, a DNA-binding domain and a C-terminal steroid-binding domain.,function:High affinity receptor for triiodothyronine.,similarity:Belongs to the nuclear hormone receptor family. NR1 subfamily.,similarity:Contains 1 nuclear receptor DNA-binding domain.,subunit:Interacts with NOCA7 in a ligand-inducible manner. Interacts with C1D.,
Usage	For Research Use Only! Not for diagnostic or therapeutic procedures.