

# TRβ1 (Phospho Ser142) Rabbit pAb

CatalogNo: YP0479 Orthogonal Validated 

## Key Features

### Host Species

- Rabbit

### Reactivity

- Human, Mouse, Rat

### Applications

- WB, ELISA

### MW

- 45kD (Observed)

### Isotype

- IgG

## 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.

## Recommended Dilution Ratios

**WB 1:500-1:2000**

**ELISA 1:5000**

**Not yet tested in other applications.**

## Basic Information

**Clonality** Polyclonal

## Immunogen Information

**Immunogen** The antiserum was produced against synthesized peptide derived from human TR-beta1 around the phosphorylation site of Ser142. AA range:116-165

**Specificity** Phospho-TRβ1 (S142) Polyclonal Antibody detects endogenous levels of TRβ1 protein only when phosphorylated at S142. The name of modified sites may be influenced by many factors, such as species (the modified site was not originally found in human samples) and the change of protein sequence (the previous protein sequence is incomplete, and the protein sequence may be prolonged with the development of protein sequencing technology). When naming, we will use the "numbers" in historical reference to keep the sites consistent with the reports. The antibody binds to the following modification sequence (lowercase letters are modification sites):HPsYS

## Target Information

**Gene name** THRB

**Protein Name** Thyroid hormone receptor beta

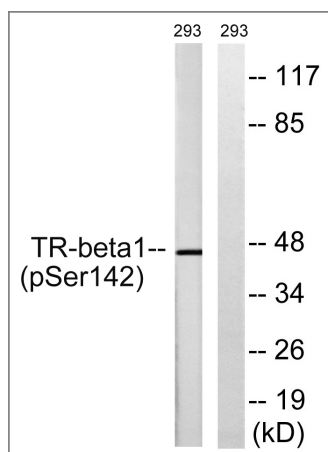
Organism	Gene ID	UniProt ID
Human	<a href="#">7068</a> ;	<a href="#">P10828</a> ;
Mouse	<a href="#">21834</a> ;	<a href="#">P37242</a> ;
Rat		<a href="#">P18113</a> ;

**Cellular Localization** Nucleus.

**Tissue specificity** Brain,Kidney,Pituitary,Placenta,Testis,

**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.,

## Validation Data



Western blot analysis of lysates from 293 cells treated with PMA 125ng/ml 30', using TR-beta1 (Phospho-Ser142) Antibody. The lane on the right is blocked with the phospho peptide.

## | Contact information

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Please scan the QR code to access additional product information:  
**TRβ1 (Phospho Ser142) Rabbit pAb**

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[Antibody](#) | [ELISA Kits](#) | [Protein](#) | [Reagents](#)