




## Product datasheet

### Anti-Ret (phospho Y1015) antibody ab74154

[3 References](#) [1 Image](#)

#### Overview

<b>Product name</b>	Anti-Ret (phospho Y1015) antibody
<b>Description</b>	Rabbit polyclonal to Ret (phospho Y1015)
<b>Host species</b>	Rabbit
<b>Tested applications</b>	<b>Suitable for:</b> WB
<b>Species reactivity</b>	<b>Reacts with:</b> African green monkey <b>Predicted to work with:</b> Mouse, Rat 
<b>Immunogen</b>	Synthetic peptide corresponding to Human Ret (phospho Y1015). <a href="#">Run BLAST with</a>  <a href="#">Run BLAST with</a> 
<b>Positive control</b>	Extracts from COS7 cells treated with EGF (200ng/ml, 30mins).
<b>General notes</b>	<p>The Life Science industry has been in the grips of a reproducibility crisis for a number of years. Abcam is leading the way in addressing this with our range of recombinant monoclonal antibodies and knockout edited cell lines for gold-standard validation. Please check that this product meets your needs before purchasing.</p> <p>If you have any questions, special requirements or concerns, please send us an inquiry and/or contact our Support team ahead of purchase. Recommended alternatives for this product can be found below, along with publications, customer reviews and Q&amp;As</p>

#### Properties

<b>Form</b>	Liquid
<b>Storage instructions</b>	Shipped at 4°C. Store at -20°C. Stable for 12 months at -20°C.
<b>Storage buffer</b>	pH: 7.40 Preservative: 0.02% Sodium azide Constituents: 50% Glycerol (glycerin, glycerine), 0.87% Sodium chloride, PBS  Without Mg <sup>2+</sup> and Ca <sup>2+</sup>
<b>Purity</b>	Immunogen affinity purified
<b>Purification notes</b>	ab74154 was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific phosphopeptide. The antibody against non-phosphopeptide was removed by chromatography using non-phosphopeptide corresponding to the phosphorylation site.
<b>Clonality</b>	Polyclonal

Isotype

IgG

## Applications

### The Abpromise guarantee

Our **Abpromise guarantee** covers the use of ab74154 in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Application	Abreviews	Notes
WB		1/500 - 1/1000. Detects a band of approximately 124 kDa (predicted molecular weight: 124 kDa).

## Target

### Function

Probable receptor with tyrosine-protein kinase activity; important for development.

### Involvement in disease

Defects in RET may be a cause of colorectal cancer (CRC) [MIM:114500].

Defects in RET are a cause of Hirschsprung disease (HSCR) [MIM:142623]. HSCR is a genetic disorder of neural crest development characterized by the absence of intramural ganglion cells in the hindgut, often resulting in intestinal obstruction. Occasionally, MEN2A or FMTC occur in association with HSCR.

Defects in RET are the cause of medullary thyroid carcinoma (MTC) [MIM:155240]. MTC is a rare tumor derived from the C cells of the thyroid. Three hereditary forms are known, that are transmitted in an autosomal dominant fashion: (a) multiple neoplasia type 2A (MEN2A), (b) multiple neoplasia type IIB (MEN2B) and (c) familial MTC (FMTC), which occurs in 25-30% of MTC cases and where MTC is the only clinical manifestation.

Defects in RET are the cause of multiple neoplasia type 2B (MEN2B) [MIM:162300]. MEN2B is an uncommon inherited cancer syndrome characterized by predisposition to MTC and pheochromocytoma which is associated with marfanoid habitus, mucosal neuromas, skeletal and ophtalmic abnormalities, and ganglioneuromas of the intestine tract. Then the disease progresses rapidly with the development of metastatic MTC and a pheochromocytoma in 50% of cases.

Defects in RET are a cause of susceptibility to pheochromocytoma (PCC) [MIM:171300]. A catecholamine-producing tumor of chromaffin tissue of the adrenal medulla or sympathetic paraganglia. The cardinal symptom, reflecting the increased secretion of epinephrine and norepinephrine, is hypertension, which may be persistent or intermittent.

Defects in RET are the cause of multiple neoplasia type 2A (MEN2A) [MIM:171400]; also known as multiple neoplasia type 2 (MEN2). MEN2A is the most frequent form of medullary thyroid cancer (MTC). It is an inherited cancer syndrome characterized by MTC, pheochromocytoma and/or hyperparathyroidism.

Defects in RET are a cause of thyroid papillary carcinoma (TPC) [MIM:188550]. TPC is a common tumor of the thyroid that typically arises as an irregular, solid or cystic mass from otherwise normal thyroid tissue. Papillary carcinomas are malignant neoplasm characterized by the formation of numerous, irregular, finger-like projections of fibrous stroma that is covered with a surface layer of neoplastic epithelial cells. Note=Chromosomal aberrations involving RET are found in thyroid papillary carcinomas. Inversion inv(10)(q11.2;q21) generates the RET/CCDC6 (PTC1) oncogene; inversion inv(10)(q11.2;q11.2) generates the RET/NCOA4 (PTC3) oncogene; translocation t(10;14)(q11;q32) with GOLGA5 generates the RET/GOLGA5 (PTC5) oncogene; translocation t(8;10)(p21.3;q11.2) with PCM1 generates the PCM1/RET fusion; translocation t(6;10)(p21.3;q11.2) with RFP generates the Delta RFP/RET oncogene; translocation t(1;10)

(p13;q11) with TRIM33 generates the TRIM33/RET (PTC7) oncogene; translocation t(7;10) (q32;q11) with TRIM24/TIF1 generates the TRIM24/RET (PTC6) oncogene. The PTC5 oncogene has been found in 2 cases of PACT in children exposed to radioactive fallout after Chernobyl. A chromosomal aberration involving TRIM27/RFP is found in thyroid papillary carcinomas. Translocation t(6;10)(p21.3;q11.2) with RET. The translocation generates TRIM27/RET and delta TRIM27/RET oncogenes.

Defects in RET are a cause of renal adysplasia (RADYS) [MIM:191830]; also known as renal agenesis or renal aplasia. Renal agenesis refers to the absence of one (unilateral) or both (bilateral) kidneys at birth. Bilateral renal agenesis belongs to a group of perinatally lethal renal diseases, including severe bilateral renal dysplasia, unilateral renal agenesis with contralateral dysplasia and severe obstructive uropathy.

Defects in RET are a cause of congenital central hypoventilation syndrome (CCHS) [MIM:209880]; also known as congenital failure of autonomic control or Ondine curse. CCHS is a rare disorder characterized by abnormal control of respiration in the absence of neuromuscular or lung disease, or an identifiable brain stem lesion. A deficiency in autonomic control of respiration results in inadequate or negligible ventilatory and arousal responses to hypercapnia and hypoxemia.

### Sequence similarities

Belongs to the protein kinase superfamily. Tyr protein kinase family.  
Contains 1 cadherin domain.  
Contains 1 protein kinase domain.

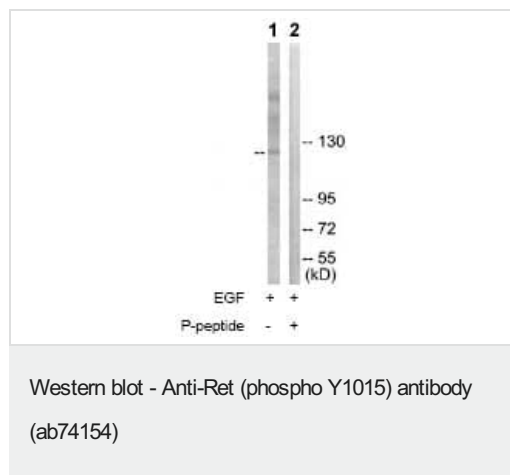
### Post-translational modifications

Autophosphorylated on C-terminal tyrosine residues upon ligand stimulation. Dephosphorylated by PTPRJ on Tyr-905, Tyr-1015 and Tyr-1062.

### Cellular localization

Membrane.

## Images



**All lanes :** Anti-Ret (phospho Y1015) antibody (ab74154) at 1/500 dilution

**Lane 1 :** Extracts from COS7 cells treated with EGF (200ng/ml, 30mins)

**Lane 2 :** Extracts from COS7 cells treated with EGF (200ng/ml, 30mins) with immunising peptide at 10 µg

Lysates/proteins at 30 µg per lane.

**Predicted band size:** 124 kDa

**Observed band size:** 124 kDa

**Please note:** All products are "FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC PROCEDURES"

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