Anti-Growth hormone receptor antibody ab65304

Overview

Product name: Anti-Growth hormone receptor antibody
Description: Rabbit polyclonal to Growth hormone receptor
Host species: Rabbit
Tested applications: Suitable for: ICC/IF, WB
Species reactivity: Reacts with: Human
Predicted to work with: Non human primates

Immunogen: Synthetic peptide conjugated to KLH derived from within residues 1 - 100 of Human Growth hormone receptor. Read Abcam’s proprietary immunogen policy (Peptide available as ab73395.)

Positive control: This antibody gave a positive result in the following whole cell lysates: A549, HeLa, DU 145

Properties

Form: Liquid
Storage instructions: Shipped at 4°C. Store at +4°C short term (1-2 weeks). Upon delivery aliquot. Store at -20°C or -80°C. Avoid freeze / thaw cycle.
Storage buffer: pH: 7.40
Preservative: 0.02% Sodium azide
Constituent: PBS

Batches of this product that have a concentration < 1mg/ml may have BSA added as a stabilising agent. If you would like information about the formulation of a specific lot, please contact our scientific support team who will be happy to help.

Purity: Immunogen affinity purified
Clonality: Polyclonal
Isotype: IgG

Applications

Our Abpromise guarantee covers the use of ab65304 in the following tested applications.
The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.
<table>
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<tr>
<th>Application</th>
<th>Abreviews</th>
<th>Notes</th>
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</thead>
<tbody>
<tr>
<td>ICC/IF</td>
<td>⭐⭐⭐⭐⭐</td>
<td>Use a concentration of 5 µg/ml.</td>
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<tr>
<td>WB</td>
<td>⭐⭐⭐⭐⭐</td>
<td>Use a concentration of 1 µg/ml. Detects a band of approximately 76 kDa (predicted molecular weight: 71 kDa).</td>
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**Target**

**Function**
Receptor for pituitary gland growth hormone involved in regulating postnatal body growth. On ligand binding, couples to the JAK2/STAT5 pathway. The soluble form (GHBP) acts as a reservoir of growth hormone in plasma and may be a modulator/inhibitor of GH signaling. Isoform 2 up-regulates the production of GHBP and acts as a negative inhibitor of GH signaling.

**Tissue specificity**
Expressed in various tissues with high expression in liver and skeletal muscle. Isoform 4 is predominantly expressed in kidney, bladder, adrenal gland and brain stem. Isoform 1 expression in placenta is predominant in chorion and decidua. Isoform 4 is highly expressed in placental villi. Isoform 2 is expressed in lung, stomach and muscle. Low levels in liver.

**Involvement in disease**
Defects in GHR are a cause of Laron syndrome (LARS) [MIM:262500]. A severe form of growth hormone insensitivity characterized by growth impairment, short stature, dysfunctional growth hormone receptor, and failure to generate insulin-like growth factor I in response to growth hormone.
Defects in GHR may be a cause of idiopathic short stature autosomal (ISSA) [MIM:604271]. Short stature is defined by a subnormal rate of growth.

**Sequence similarities**
Belongs to the type I cytokine receptor family. Type 1 subfamily. Contains 1 fibronectin type-III domain.

**Domain**
The WSXWS motif appears to be necessary for proper protein folding and thereby efficient intracellular transport and cell-surface receptor binding. The box 1 motif is required for JAK interaction and/or activation. The extracellular domain is the ligand-binding domain representing the growth hormone-binding protein (GHBP). The ubiquitination-dependent endocytosis motif (UbE) is required for recruitment of the ubiquitin conjugation system on to the receptor and for its internalization.

**Post-translational modifications**
The soluble form (GHBP) is produced by phorbol ester-promoted proteolytic cleavage at the cell surface (shedding) by ADAM17/TACE. Shedding is inhibited by growth hormone (GH) binding to the receptor probably due to a conformational change in GHR rendering the receptor inaccessible to ADAM17. On GH binding, phosphorylated on tyrosine residues in the cytoplasmic domain by JAK2. On ligand binding, ubiquitinated on lysine residues in the cytoplasmic domain. This ubiquitination is not sufficient for GHR internalization.

**Cellular localization**
Secreted; Cell membrane. On growth hormone binding, GHR is ubiquitinated, internalized, down-regulated and transported into a degradative or non-degradative pathway and Cell membrane. Remains fixed to the cell membrane and is not internalized.

**Images**
All lanes: Anti-Growth hormone receptor antibody (ab65304) at 1 µg/ml

Lane 1: A549 (Human lung adenocarcinoma epithelial cell line) Whole Cell Lysate
Lane 2: HeLa (Human epithelial carcinoma cell line) Whole Cell Lysate
Lane 3: DU 145 (Human prostate carcinoma cell line) Whole Cell Lysate

Lysates/proteins at 10 µg per lane.

Secondary

All lanes: Goat polyclonal to Rabbit IgG - H&L - Pre-Adsorbed (HRP) at 1/3000 dilution

Developed using the ECL technique.

Performed under reducing conditions.

Predicted band size: 71 kDa
Observed band size: 76 kDa

why is the actual band size different from the predicted?

Additional bands at: 56 kDa. We are unsure as to the identity of these extra bands.

Exposure time: 12 minutes

Growth hormone receptor contains a number of potential glycosylation sites (SwissProt) which may explain its migration at a higher molecular weight than predicted.
ICC/IF image of ab65304 stained HepG2 cells. The cells were 4% PFA fixed (10 min) and then incubated in 1%BSA / 10% normal goat serum / 0.3M glycine in 0.1% PBS-Tween for 1h to permeabilise the cells and block non-specific protein-protein interactions. The cells were then incubated with the antibody (ab65304, 5µg/ml) overnight at +4°C. The secondary antibody (green) was ab96899, DyLight® 488 goat anti-rabbit IgG (H+L) used at a 1/250 dilution for 1h. Alexa Fluor® 594 WGA was used to label plasma membranes (red) at a 1/200 dilution for 1h. DAPI was used to stain the cell nuclei (blue) at a concentration of 1.43µM. This antibody also gave a positive result in 4% PFA fixed (10 min) HeLa, Hek293 and MCF7 cells at 5µg/ml, and in 100% methanol fixed (5 min) HeLa, Hek293, HepG2 and MCF7 cells at 5µg/ml.

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