Anti-Mineralocorticoid Receptor antibody [H10E4C9F] ab2774

Overview

Product name: Anti-Mineralocorticoid Receptor antibody [H10E4C9F]
Description: Mouse monoclonal [H10E4C9F] to Mineralocorticoid Receptor
Host species: Mouse
Specificity: Detects mineralocorticoid receptor (MR).
Tested applications:
Suitable for: Flow Cyt, Inhibition Assay, ELISA, IHC-P, WB, ICC/IF
Unsuitable for: IP
Species reactivity:
Reacts with: Mouse, Rat, Sheep, Rabbit, Chicken, Hamster, Cow, Dog, Human
Immunogen: Aldosterone 3. This antibody was produced using the anti idiotypic method.

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: Preservative: 0.05% Sodium azide
Constituent: PBS
Purity: Protein A purified
Clonality: Monoclonal
Clone number: H10E4C9F
Isotype: IgG1

Applications

Our Abpromise guarantee covers the use of ab2774 in the following tested applications.
The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.
Application notes

Is unsuitable for IP.

Target

Function

Receptor for both mineralocorticoids (MC) such as aldosterone and glucocorticoids (GC) such as corticosterone or cortisol. Binds to mineralocorticoid response elements (MRE) and transactivates target genes. The effect of MC is to increase ion and water transport and thus raise extracellular fluid volume and blood pressure and lower potassium levels.

Tissue specificity

Ubiquitous. Highly expressed in distal tubules, convoluted tubules and cortical collecting duct in kidney, and in sweat glands. Detected at lower levels in cardiomyocytes, in epidermis and in colon enterocytes.

Involvement in disease

Defects in NR3C2 are a cause of autosomal dominant pseudohypoaldosteronism type I (AD-PHA1) [MIM:177735]. PHA1 is characterized by urinary salt wasting, resulting from target organ unresponsiveness to mineralocorticoids. There are 2 forms of PHA1: the autosomal dominant form that is mild, and the recessive form which is more severe and due to defects in any of the epithelial sodium channel subunits. In AD-PHA1 the target organ defect is confined to kidney. Clinical expression can vary from asymptomatic to moderate. It may be severe at birth, but symptoms remit with age. Familial and sporadic cases have been reported. Defects in NR3C2 are a cause of early-onset hypertension with severe exacerbation in pregnancy (EOHSEP) [MIM:605115]. Inheritance is autosomal dominant. The disease is characterized by the onset of severe hypertension before the age of 20, and by suppression of aldosterone secretion.

Sequence similarities

Belongs to the nuclear hormone receptor family. NR3 subfamily.

Contains 1 nuclear receptor DNA-binding domain.

Domain

Composed of three domains: a modulating N-terminal domain, a DNA-binding domain and a C-terminal ligand-binding domain.
Post-translational modifications

Phosphorylated.

Cellular localization

Cytoplasm. Nucleus. Endoplasmic reticulum membrane. Cytoplasmic and nuclear in the absence of ligand; nuclear after ligand-binding. When bound to HSD11B2, it is found associated with the endoplasmic reticulum membrane.

Images

Formalin-fixed, paraffin-embedded cow kidney tissue stained for Mineralocorticoid Receptor using ab2774 at 1/200 dilution in immunohistochemical analysis.

Heat mediated Antigen retrieval using Citrate buffer, 10 mM pH 6.0 was used.

ab2774 (4µg/ml) staining mineralocorticoid receptor in human pancreas, using an automated system (DAKO Autostainer Plus). Using this protocol there is strong staining of the islets of Langerhans and some weaker staining of the exocrine cells of the pancreas.

Sections were rehydrated and antigen retrieved with the Dako 3 in 1 AR buffer EDTA pH 9.0 in a DAKO PT link. Slides were peroxidase blocked in 3% H2O2 in methanol for 10 mins. They were then blocked with Dako Protein block for 10 minutes (containing casein 0.25% in PBS) then incubated with primary antibody for 20 min and detected with Dako envision flex amplification kit for 30 minutes. Colorimetric detection was completed with Diaminobenzidine for 5 minutes. Slides were counterstained with Haematoxylin and coverslipped under DePeX. Please note that, for manual staining, optimization of primary antibody concentration and incubation time is recommended. Signal amplification may be required.
Immunocytochemistry/ Immunofluorescence - Anti-Mineralocorticoid Receptor antibody [H10E4C9F] (ab2774)

Image courtesy of an anonymous Abreview.

Immunocytochemistry/ Immunofluorescence - Anti-Mineralocorticoid Receptor antibody [H10E4C9F] (ab2774)

Overlay histogram showing HEK293 cells stained with ab2774 (red line). The cells were fixed with methanol (5 min) and then permeabilized with 0.1% PBS-Tween for 20 min. The cells were then incubated in 1x PBS / 10% normal goat serum / 0.3M glycine to block non-specific protein-protein interactions followed by the antibody (ab2774, 1/20 dilution) for 30 min at 22°C. The secondary antibody used was DyLight® 488 goat anti-mouse IgG (H+L) (ab96879) at 1/500 dilution for 30 min at 22°C. Isotype control antibody (black line) was mouse IgG1 [ICIGG1] (ab91353, 2µg/1x10^6 cells) used under the same conditions. Acquisition of >5,000 events was performed. This antibody gave a slightly decreased signal in HEK293 cells fixed with 4% paraformaldehyde (10 min) permeabilized in 0.1% PBS-Tween used under the same conditions.

Flow Cytometry - Anti-Mineralocorticoid Receptor antibody [H10E4C9F] (ab2774)

Immunohistochemistry was performed on both normal and cancer biopsies of deparaffinized Human colon carcinoma tissues. To expose target proteins heat induced antigen retrieval was performed using 10mM sodium citrate (pH6.0) buffer microwaved for 8-15 minutes. Following antigen retrieval tissues were blocked in 3% BSA-PBS for 30 minutes at room temperature. Tissues were then probed at a dilution of 1:100 with a mouse monoclonal antibody recognizing Mineralocorticoid Receptor ab2774 or without primary antibody (negative control) overnight at 4°C in a humidified chamber. Tissues were washed extensively with PBST and endogenous peroxidase activity was quenched with a peroxidase suppressor. Detection was performed using a biotin-conjugated secondary antibody and SA-HRP followed by colorimetric detection using DAB. Tissues were counterstained with hematoxylin and
Immunohistochemistry was performed on both normal and cancer biopsies of deparaffinized Human tonsil tissue tissues. To expose target proteins heat induced antigen retrieval was performed using 10mM sodium citrate (pH6.0) buffer microwaved for 8-15 minutes. Following antigen retrieval tissues were blocked in 3% BSA-PBS for 30 minutes at room temperature. Tissues were then probed at a dilution of 1:100 with a mouse monoclonal antibody recognizing Mineralocorticoid Receptor ab2774 or without primary antibody (negative control) overnight at 4°C in a humidified chamber. Tissues were washed extensively with PBST and endogenous peroxidase activity was quenched with a peroxidase suppressor. Detection was performed using a biotin-conjugated secondary antibody and SA-HRP followed by colorimetric detection using DAB. Tissues were counterstained with hematoxylin and prepped for mounting.

Immunohistochemistry was performed on both normal and cancer biopsies of deparaffinized Human kidney tissue tissues. To expose target proteins heat induced antigen retrieval was performed using 10mM sodium citrate (pH6.0) buffer microwaved for 8-15 minutes. Following antigen retrieval tissues were blocked in 3% BSA-PBS for 30 minutes at room temperature. Tissues were then probed at a dilution of 1:200 with a mouse monoclonal antibody recognizing Mineralocorticoid Receptor ab2774 or without primary antibody (negative control) overnight at 4°C in a humidified chamber. Tissues were washed extensively with PBST and endogenous peroxidase activity was quenched with a peroxidase suppressor. Detection was performed using a biotin-conjugated secondary antibody and SA-HRP followed by colorimetric detection using DAB. Tissues were counterstained with hematoxylin and prepped for mounting.
Immunofluorescent analysis of Mineralocorticoid Receptor using Mineralocorticoid Receptor Monoclonal antibody (H10E4C9F) ab2774 shows staining in HEK293 cells. Mineralocorticoid Receptor staining (green) F-Actin staining with Phalloidin (red) and nuclei with DAPI (blue) is shown. Cells were grown on chamber slides and fixed with formaldehyde prior to staining. Cells were probed without (control) or with or an antibody recognizing Mineralocorticoid Receptor ab2774 at a dilution of 1:20-1:200 over night at 4 °C washed with PBS and incubated with a DyLight-488 conjugated secondary antibody. Images were taken at 60X magnification.

Immunofluorescent analysis of Mineralocorticoid Receptor using Mineralocorticoid Receptor Monoclonal antibody (H10E4C9F) ab2774 shows staining in HeLa cells. Mineralocorticoid Receptor staining (green) F-Actin staining with Phalloidin (red) and nuclei with DAPI (blue) is shown. Cells were grown on chamber slides and fixed with formaldehyde prior to staining. Cells were probed without (control) or with or an antibody recognizing Mineralocorticoid Receptor ab2774 at a dilution of 1:20-1:200 over night at 4 °C washed with PBS and incubated with a DyLight-488 conjugated secondary antibody. Images were taken at 60X magnification.

Immunofluorescent analysis of Mineralocorticoid Receptor using Mineralocorticoid Receptor Monoclonal antibody (H10E4C9F) ab2774 shows staining in HepG2 cells. Mineralocorticoid Receptor staining (green) F-Actin staining with Phalloidin (red) and nuclei with DAPI (blue) is shown. Cells were grown on chamber slides and fixed with formaldehyde prior to staining. Cells were probed without (control) or with or an antibody recognizing Mineralocorticoid Receptor ab2774 at a dilution of 1:20-1:200 over night at 4 °C washed with PBS and incubated with a DyLight-488 conjugated secondary antibody. Images were taken at 60X magnification.

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