Immunotag™ MCR Polyclonal Antibody

Antibody Specification	
Catalog No.	ITN2320
Product Description	Immunotag™ MCR 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	MCR
Clonality	Polyclonal
Storage/Stability	-20°C/1 year
Application	WB,ELISA
Recommended Dilution	WB 1:500-2000 ELISA 1:5000-20000
Concentration	1 mg/ml
Reactive Species	Human,Rat,Mouse
Host Species	Rabbit
Immunogen	Synthesized peptide derived from human protein, at AA range: 10-90
Specificity	MCR Polyclonal Antibody detects endogenous levels of protein.
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen
Form	Liquid in PBS containing 50% glycerol, and 0.02% sodium azide.
Gene Name	NR3C2 MCR MLR
Accession No.	P08235 Q8VII8 P22199

Antibody Specification	
Description	nuclear receptor subfamily 3 group C member 2(NR3C2) Homo sapiens This gene encodes the mineralocorticoid receptor, which mediates aldosterone actions on salt and water balance within restricted target cells. The protein functions as a ligand-dependent transcription factor that binds to mineralocorticoid response elements in order to transactivate target genes. Mutations in this gene cause autosomal dominant pseudohypoaldosteronism type I, a disorder characterized by urinary salt wasting. Defects in this gene are also associated with early onset hypertension with severe exacerbation in pregnancy. Alternative splicing results in multiple transcript variants. [provided by RefSeq, Oct 2009],
Cell Pathway/ Category	Aldosterone-regulated sodium reabsorption,
Protein Expression	Brain, Heart, Kidney, Leukocyte, PCR rescued clones,
Subcellular Localization	nucleus,nucleoplasm,endoplasmic reticulum membrane,receptor complex,
Protein Function	Additional isoforms seem to exist, disease:Defects in NR3C2 are a cause of autosomal dominant pseudohypoaldosteronism type I (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 autosomal dominant 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., disease:Defects in NR3C2 are a cause of early onset hypertension with severe exacerbation in pregnancy [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., domain:Composed of three domains: a modulating N-terminal domain, a DNA-binding domain and a C-terminal steroid-binding domain, 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., online information:Mineralocorticoid receptor entry, PTM:Phosphorylated., similarity:Belongs to the nuclear hormone receptor family., similarity:Belongs to the nuclear hormone receptor family, similarity:Contains 1 nuclear receptor DNA-binding domain., subcellular location: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., subunit:Heteromultimeric cytoplasmic complex with HSP90, HSP70, and FKBP4, in the absence of ligand. After ligand binding, it translocates to the n
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