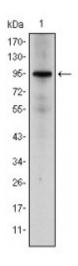


Anti-GR antibody



Description Mouse monoclonal to GR.

Model STJ98115

Host Mouse

Reactivity Human

Applications ELISA, FC, IF, IHC, WB

Immunogen Purified recombinant fragment of human GR expressed in E. Coli.

Gene ID 2908

Gene Symbol NR3C1

Dilution range WB 1:500-1:2000IHC 1:200-1:1000IF 1:200-1:1000FC 1:200-1:400ELISA

1:10000

Specificity GR Monoclonal Antibody detects endogenous levels of GR protein.

Tissue Specificity Widely expressed including bone, stomach, lung, liver, colon, breast, ovary,

pancreas and kidney. In the heart, detected in left and right atria, left and right ventricles, aorta, apex, intraventricular septum, and atrioventricular node as well as whole adult and fetal heart. Isoform Beta: Widely expressed including brain, bone marrow, thymus, spleen, liver, kidney, pancreas, lung, fat, skeletal muscle, heart, placenta and blood leukocytes. Isoform Alpha-2: Expressed at

low level.

Purification Affinity purification

Clone ID 6000000

Note For Research Use Only (RUO).

Protein Name Glucocorticoid receptor GR Nuclear receptor subfamily 3 group C member 1

Clonality Monoclonal

Conjugation Unconjugated

Isotype IgG1

Formulation Ascitic fluid containing 0.03% sodium azide.

Storage Instruction Store at -20°C, and avoid repeat freeze-thaw cycles.

Database Links HGNC:7978OMIM:138040

Alternative Names Glucocorticoid receptor GR Nuclear receptor subfamily 3 group C member 1

Function Receptor for glucocorticoids (GC). Has a dual mode of action: as a

transcription factor that binds to glucocorticoid response elements (GRE), both for nuclear and mitochondrial DNA, and as a modulator of other transcription factors. Affects inflammatory responses, cellular proliferation and differentiation in target tissues. Involved in chromatin remodeling . Plays a role in rapid mRNA degradation by binding to the 5' UTR of target mRNAs and interacting with PNRC2 in a ligand-dependent manner which recruits the RNA helicase UPF1 and the mRNA-decapping enzyme DCP1A, leading to RNA decay. Could act as a coactivator for STAT5-dependent transcription upon growth hormone (GH) stimulation and could reveal an essential role of hepatic GR in the control of body growth . Isoform Alpha: Has transcriptional activation and repression activity. Mediates glucocorticoid-induced apoptosis . Promotes accurate chromosome segregation during mitosis . May act as a tumor suppressor. May play a negative role in adipogenesis through the regulation of lipolytic and antilipogenic gene expression . Isoform Beta: Acts as a dominant negative inhibitor of isoform Alpha . Has intrinsic transcriptional activity independent of isoform Alpha when both isoforms are coexpressed. Loses this transcription modulator function on its own. Has no hormone-binding activity. May play a role in controlling glucose metabolism by maintaining insulin sensitivity. Reduces hepatic gluconeogenesis through down-regulation of PEPCK in an isoform Alpha-dependent manner. Directly regulates STAT1 expression in isoform Alpha-independent manner . Isoform Alpha-2: Has lower transcriptional activation activity than isoform Alpha. Exerts a dominant negative effect on isoform Alpha trans-repression mechanism .; Isoform GR-P: Increases activity of isoform Alpha. Isoform Alpha-B: More effective than isoform Alpha in transcriptional activation, but not repression activity. Isoform 10: Has transcriptional activation activity. Isoform Alpha-C1: Has transcriptional activation activity. Isoform Alpha-C2: Has transcriptional activation activity. Isoform Alpha-C3: Has highest transcriptional activation activity of all isoforms created by alternative initiation. Has transcriptional repression activity. Mediates glucocorticoidinduced apoptosis. Isoform Alpha-D1: Has transcriptional activation activity. Isoform Alpha-D2: Has transcriptional activation activity. Isoform Alpha-D3: Has lowest transcriptional activation activity of all isoforms created by alternative initiation. Has transcriptional repression activity.

Sequence and Domain Family

Composed of three domains: a modulating N-terminal domain, a DNA-binding domain and a C-terminal ligand-binding domain . The ligand-binding domain is required for correct chromosome segregation during mitosis although ligand binding is not required .

Cellular Localization

Isoform Alpha: Cytoplasm Nucleus Mitochondrion Cytoplasm, cytoskeleton, spindle Cytoplasm, cytoskeleton, microtubule organizing center, centrosome. After ligand activation, translocates from the cytoplasm to the nucleus. Isoform Beta: Nucleus Cytoplasm. Expressed predominantly in the nucleus with some expression also detected in the cytoplasm. Isoform Alpha-B: Nucleus Cytoplasm. After ligand activation, translocates from the cytoplasm to the nucleus.

Post-translational Modifications

Acetylation by CLOCK reduces its binding to glucocorticoid response elements and its transcriptional activity. Increased proteasome-mediated degradation in response to glucocorticoids. Isoform Alpha-B appears to be more susceptible to proteolytic degradation than isoform Alpha. Phosphorylated in the absence of hormone; becomes hyperphosphorylated in the presence of glucocorticoid. The Ser-203, Ser-226 and Ser-404phosphorylated forms are mainly cytoplasmic, and the Ser-211phosphorylated form is nuclear. Phosphorylation at Ser-211 increases transcriptional activity. Phosphorylation at Ser-203, Ser-226 and Ser-404 decreases signaling capacity. Phosphorylation at Ser-404 may protect from glucocorticoid-induced apoptosis. Phosphorylation at Ser-203 and Ser-211 is not required in regulation of chromosome segregation. May be dephosphorylated by PPP5C, attenuates NR3C1 action. Sumoylation at Lys-277 and Lys-293 negatively regulates its transcriptional activity. Sumovlation at Lys-703 positively regulates its transcriptional activity in the presence of RWDD3. Sumoylation at Lys-277 and Lys-293 is dispensable whereas sumoylation at Lys-703 is critical for the stimulatory effect of RWDD3 on its transcriptional activity. Heat shock increases sumoylation in a RWDD3-dependent manner. Ubiquitinated; restricts glucocorticoid-mediated transcriptional signaling.

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