

**Glucocorticoid Receptor (D4X9S) Mouse
mAb****Orders:** 877-616-CELL (2355)
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Applications: W, IP, IHC-P, IF-IC, FC-FP	Reactivity: H Mk	Sensitivity: Endogenous	MW (kDa): 80, 91, 94	Source/Isotype: Mouse IgG1	UniProt ID: #P04150	Entrez-Gene Id: 2908
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**Product Usage
Information****Application**

Western Blotting
Immunoprecipitation
Immunohistochemistry (Paraffin)
Immunofluorescence (Immunocytochemistry)
Flow Cytometry (Fixed/Permeabilized)

Dilution

1:1000
1:100
1:300
1:400 - 1:1600
1:200

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at -20°C. Do not aliquot the antibody.

Specificity/Sensitivity

Glucocorticoid Receptor (D4X9S) Mouse mAb recognizes endogenous levels of total glucocorticoid receptor protein.

Source / Purification

Monoclonal antibody is produced by immunizing animals with recombinant protein specific to the amino terminus of human glucocorticoid receptor protein.

Background

Glucocorticoid hormones control cellular proliferation, inflammation, and metabolism through their association with the glucocorticoid receptor (GR)/NR3C1, a member of the nuclear hormone receptor superfamily of transcription factors (1). GR is composed of several conserved structural elements, including a carboxy-terminal ligand-binding domain (which also contains residues critical for receptor dimerization and hormone-dependent gene transactivation), a neighboring hinge region containing nuclear localization signals, a central zinc-finger-containing DNA-binding domain, and an amino-terminal variable region that participates in ligand-independent gene transcription. In the absence of hormone, a significant population of GR is localized to the cytoplasm in an inactive form via its association with regulatory chaperone proteins, such as HSP90, HSP70, and FKBP52. On hormone binding, GR is released from the chaperone complex and translocates to the nucleus as a dimer to associate with specific DNA sequences termed glucocorticoid response elements (GREs), thereby enhancing or repressing transcription of specific target genes (2). It was demonstrated that GR-mediated transcriptional activation is modulated by phosphorylation (3-5). Although GR can be basally phosphorylated in the absence of hormone, it becomes hyperphosphorylated upon binding receptor agonists. It has been suggested that hormone-dependent phosphorylation of GR may determine target promoter specificity, cofactor interaction, strength and duration of receptor signaling, receptor stability, and receptor subcellular localization (3).

Background References

1. Yamamoto, K.R. (1985) *Annu. Rev. Genet* 19, 209-52.
2. Necela, B.M. and Cidlowski, J.A. (2003) *Trends Pharmacol. Sci.* 24, 58-61.
3. Wang, Z. et al. (2002) *J. Biol. Chem.* 277, 26573-80.
4. Rogatsky, I. et al. (1998) *J. Biol. Chem.* 273, 14315-21.
5. Krstic, M. D. et al. (1997) *Mol. Cell. Biol.* 17, 3947-54.

Species Reactivity

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

Western Blot Buffer

IMPORTANT: For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS, 0.1% Tween@ 20 at 4°C with gentle shaking, overnight.

Applications Key

W: Western Blotting **IP:** Immunoprecipitation **IHC-P:** Immunohistochemistry (Paraffin) **IF-IC:** Immunofluorescence (Immunocytochemistry) **FC-FP:** Flow Cytometry (Fixed/Permeabilized)

Cross-Reactivity Key

H: Human **Mk:** Monkey

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