

# GR (F-10): sc-376426

## BACKGROUND

The glucocorticoid receptor (GR) is a ubiquitously expressed transcription factor that mediates the effects of glucocorticoids. The most abundant isoform is GR  $\alpha$ . GR induces or represses the expression of genes in response to glucocorticoids, mediating such processes as apoptosis, cell growth and differentiation. A significant class of genes suppressed by GR is controlled by the transcription factor AP-1. GR has also been shown to be the limiting factor in the induction of gene expression by glucocorticoids. It has been revealed that GR forms a complex with HSP 90, rendering the non-ligand bound receptor transcriptionally inactive. More importantly, mutant GRs lacking the signaling domain remain constitutively active.

## REFERENCES

1. Hollenberg, S.M., et al. 1985. Primary structure and expression of a functional human glucocorticoid receptor cDNA. *Nature* 318: 635-641.
2. Strähle, U., et al. 1992. At least three promoters direct expression of the mouse glucocorticoid receptor gene. *Proc. Natl. Acad. Sci. USA* 89: 6731-6735.

## CHROMOSOMAL LOCATION

Genetic locus: Nr3c1 (mouse) mapping to 18 B3.

## SOURCE

GR (F-10) is a mouse monoclonal antibody specific for an epitope mapping between amino acids 5-33 at the N-terminus of GR  $\alpha$  of mouse origin.

## PRODUCT

Each vial contains 200  $\mu$ g IgG<sub>2a</sub> kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin. Also available as TransCruz reagent for Gel Supershift and ChIP applications, sc-376426 X, 200  $\mu$ g/0.1 ml.

Blocking peptide available for competition studies, sc-376426 P, (100  $\mu$ g peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% stabilizer protein).

## APPLICATIONS

GR (F-10) is recommended for detection of GR  $\alpha$  and GR  $\beta$  of mouse and rat origin by Western Blotting (starting dilution 1:100, dilution range 1:100-1:1000), immunoprecipitation [1-2  $\mu$ g per 100-500  $\mu$ g of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for GR siRNA (m): sc-35506, GR shRNA Plasmid (m): sc-35506-SH and GR shRNA (m) Lentiviral Particles: sc-35506-V.

GR (F-10) X TransCruz antibody is recommended for Gel Supershift and ChIP applications.

Molecular Weight of GR  $\alpha$ : 95 kDa.

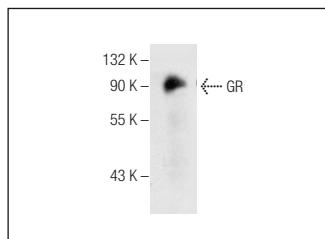
Molecular Weight of GR  $\beta$ : 90 kDa.

Positive Controls: NIH/3T3 nuclear extract: sc-2138, KNRK nuclear extract: sc-2141 or c4 whole cell lysate: sc-364186.

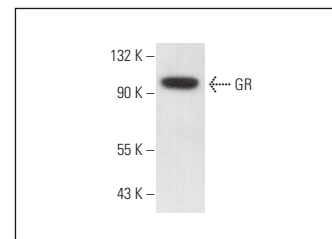
## STORAGE

Store at 4° C, **\*\*DO NOT FREEZE\*\***. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

## DATA



GR (F-10): sc-376426. Western blot analysis of GR expression in NIH/3T3 nuclear extract.



GR (F-10): sc-376426. Western blot analysis of GR expression in c4 whole cell lysate.

## SELECT PRODUCT CITATIONS

1. Zhao, L., et al. 2015. Inhibition of 11 $\beta$ -HSD1 by LG13 improves glucose metabolism in type 2 diabetic mice. *J. Mol. Endocrinol.* 55: 119-131.
2. Xiao, H., et al. 2018. Increased H3K27ac level of ACE mediates the intergenerational effect of low peak bone mass induced by prenatal dexamethasone exposure in male offspring rats. *Cell Death Dis.* 9: 638.
3. Xiao, H., et al. 2020. The low-expression programming of 11 $\beta$ -HSD2 mediates osteoporosis susceptibility induced by prenatal caffeine exposure in male offspring rats. *Br. J. Pharmacol.* 177: 4683-4700.
4. Chen, Y., et al. 2021. Sex difference in adrenal developmental toxicity induced by dexamethasone and its intrauterine programming mechanism. *Pharmacol. Res.* 174: 105942.
5. Timmermans, S., et al. 2022. Point mutation I634A in the glucocorticoid receptor causes embryonic lethality by reduced ligand binding. *J. Biol. Chem.* 298: 101574.
6. Shi, H., et al. 2022. Intrauterine programming of cartilaginous 11 $\beta$ -HSD2 induced by corticosterone and caffeine mediated susceptibility to adult osteoarthritis. *Ecotoxicol. Environ. Saf.* 239: 113624.
7. Jiang, T., et al. 2023. LOX overexpression programming mediates the osteoclast mechanism of low peak bone mass in female offspring rats caused by pregnant dexamethasone exposure. *Cell Commun. Signal.* 21: 84.
8. Shi, H., et al. 2023. Autophagy inhibition mediated by intrauterine miR-1912-3p/CTSD programming participated in the susceptibility to osteoarthritis induced by prenatal dexamethasone exposure in male adult offspring rats. *FASEB J.* 37: e23011.
9. Xiao, H., et al. 2023. Inhibition of miR-6215 rescued low subchondral bone mass caused by maternal exposure to dexamethasone in female offspring rats. *Biochem. Pharmacol.* 215: 115722.

## RESEARCH USE

For research use only, not for use in diagnostic procedures.