

DESCRIPTION

Species Reactivity	Rat
Specificity	Detects rat Growth Hormone R/GHR in direct ELISAs and Western blots. In direct ELISAs, approximately 30% cross-reactivity with recombinant mouse GHR and recombinant human GHR is observed.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant rat Growth Hormone R/GHR Phe19-Arg265 Accession # P16310
Endotoxin Level	<0.10 EU per 1 µg of the antibody by the LAL method.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details.

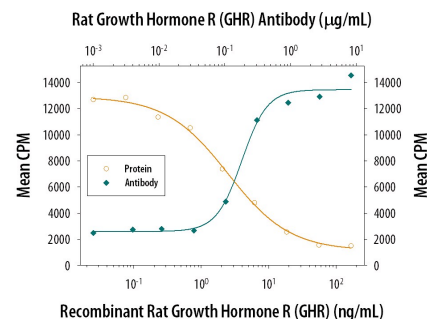
APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. [General Protocols](#) are available in the Technical Information section on our website.

	Recommended Concentration	Sample
Western Blot	0.1 µg/mL	Recombinant Rat Growth Hormone R/GHR Fc Chimera (Catalog # 1211-GR)
Immunohistochemistry	5-15 µg/mL	Perfusion fixed frozen sections of rat thymus
Neutralization	Measured by its ability to neutralize Growth Hormone R/GHR-mediated inhibition of proliferation in the Nb2-11 rat lymphoma cell line. The Neutralization Dose (ND ₅₀) is typically 0.075-0.3 µg/mL in the presence of 10 ng/mL Recombinant Rat Growth Hormone R/GHR Fc Chimera and 0.2 ng/mL Recombinant Human Growth Hormone.	

DATA

Neutralization



Growth Hormone R/GHR Inhibition of Growth Hormone-dependent Cell Proliferation and Neutralization by Rat Growth Hormone R/GHR Antibody. Recombinant Rat Growth Hormone R/GHR Fc Chimera (Catalog # [1211-GR](#)) inhibits Recombinant Human Growth Hormone (Catalog # [1067-GH](#)) induced proliferation in the Nb2-11 rat lymphoma cell line in a dose-dependent manner (orange line). Inhibition of Recombinant Human Growth Hormone (0.2 ng/mL) activity elicited by Recombinant Rat Growth Hormone R/GHR Fc Chimera (10 ng/mL) is neutralized (green line) by increasing concentrations of Goat Anti-Rat Growth Hormone R/GHR Antigen Affinity-purified Polyclonal Antibody (Catalog # AF1211). The ND₅₀ is typically 0.075-0.3 µg/mL.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.2 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 6 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

Growth hormone (GH), also known as somatotropin, is a member of a family of growth factors that includes prolactin, placental lactogens, proliferins and somatotactin (1, 2). It is synthesized primarily by somatotropes in the anterior pituitary and is released as an endocrine hormone. Other cells and tissues, including lymphoid tissues, can also produce GH (3). GH is a pleiotropic molecule which can act directly or indirectly via IGF-I, to regulate growth and metabolism as well as enhance T cell survival and thymic functions (1, 2, 4). GH exerts its biological actions by binding to the GH receptor (GHR) that is present in many cell types (1, 2). Rat GHR cDNA encodes a 638 amino acid (aa) residue type I transmembrane protein with a 18 aa signal peptide, a 247 aa extracellular domain, a 24 aa transmembrane domain and a 349 aa cytoplasmic domain. An alternatively spliced 297 aa isoform of rat GHR also exists. This 279 aa variant corresponds to the serum GH-binding protein and is identical in sequence to the extracellular domain of the transmembrane protein up to Glu262 (5). Ligation of GHR by GH has been shown to result in receptor dimerization and activation of the JAK/STAT signaling cascade (6). The soluble GHBP has been shown to interfere with GH signaling by competing with the transmembrane receptor of GH. Alternatively, the GHBP has also been shown to enhance GH action by slowing GH clearance (5, 7).

References:

1. Goffin, V. *et al.* (1996) *Endocrine Rev.* **17**:385.
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3. Clark, R. (1997) *Endocr. Rev.* **18**:157.
4. Welniak, L.A. *et al.* (2002) *J. Leukoc. Biol.* **71**:381.
5. Postel-Vinay, M.C. and J. Finidori (1995) *Eur. J. Endocrinol.* **133**:654.
6. Carter-Su, C. *et al.* (1996) *Annu. Rev. Physiol.* **58**:187.
7. Frick, G.P. *et al.* (1998) *Endocrinology* **139**:2824.