

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects mouse IGFBP-1 in direct ELISAs and Western blots. In direct ELISAs, less than 10% cross-reactivity with recombinant human IGFBP-1 and less than 1% cross-reactivity with recombinant mouse (rm) IGFBP-4, and rmIGFBP-5 is observed.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant mouse IGFBP-1 Ala26-Asn272 Accession # P47876
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied as a 0.2 µm filtered solution in PBS.

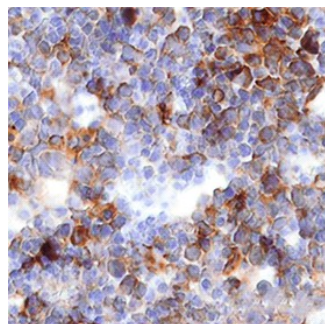
APPLICATIONS

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 Mouse IGFBP-1 (Catalog # 1588-B1)
Immunohistochemistry	5-15 µg/mL	See Below

DATA

Immunohistochemistry



IGFBP-1 in Mouse Embryo. IGFBP-1 was detected in immersion fixed frozen sections of mouse embryo (13 d.p.c.) using Goat Anti-Mouse IGFBP-1 Antigen Affinity-purified Polyclonal Antibody (Catalog # AF1240) at 15 µg/mL overnight at 4 °C. Tissue was stained using the Anti-Goat HRP-DAB Cell & Tissue Staining Kit (brown; Catalog # CTS008) and counterstained with hematoxylin (blue). Specific staining was localized to developing liver. View our protocol for [Chromogenic IHC Staining of Frozen Tissue Sections](#).

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. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> ● 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

The insulin-like growth factor binding protein (IGFBP) family consists of six structurally related proteins that bind IGF with high affinity (1, 2). These proteins share conserved cysteine-rich N- and C- terminal regions that participate in IGF binding. IGFBPs regulate the bioavailability of IGFs and modulate their biological activities, both positively and negatively. Some IGFBPs also have intrinsic bioactivity that is IGF-independent. Post-translational modifications of the IGFBPs, including glycosylation, phosphorylation and proteolysis, influence IGF binding affinities and tissue localization, affecting both the IGF-dependent and independent functions (1, 2).

Mouse IGFBP-1 cDNA encodes a 272 amino acid (aa) residue precursor protein with a putative 25 aa signal peptide (3). Mature mouse IGFBP-1 contains potential phosphorylation and proteolytic cleavage sites, an Arg-Gly-Asp (RGD) integrin receptor recognition sequence, but lacks potential N-linked glycosylation sites. IGFBP-1 binds equally well to IGF-I and IGF-II. Phosphorylation of human IGFBP-1, but not rat IGFBP-1, increases IGF affinity. Mouse IGFBP-1 shares 67% and 93% aa sequence identity with human and rat IGFBP-1, respectively. IGFBP-1 is expressed in liver, decidua, and kidneys and is the most abundant IGFBP in amniotic fluid. Hepatic production of IGFBP-1 is down-regulated by insulin and up-regulated by glucocorticoids. Circulating IGFBP-1 levels are elevated under various catabolic conditions including bacterial or viral infections, trauma and diabetes (4). IGFBP-1 has been shown to either potentiate or inhibit the activities of IGF in a variety of cells. IGFBP-1, through its RDG motif, also interacts with $\alpha 5 \beta 1$ integrin to stimulate changes in cell adhesion and migration in the absence of IGFs (1, 2).

References:

1. Firth, S.M. and R.C. Baxter (2002) *Endocrine Reviews* **23**:824.
2. Rajaram, S. *et al.* (1997) *Endocrine Reviews* **18**:801.
3. Schuller, A.G. *et al.* (1994) *Mol. Cell. Endocrinol.* **104**:57.
4. Lang, C.H. *et al.* (2003) *Endocrinology* **144**:3922.