

DESCRIPTION

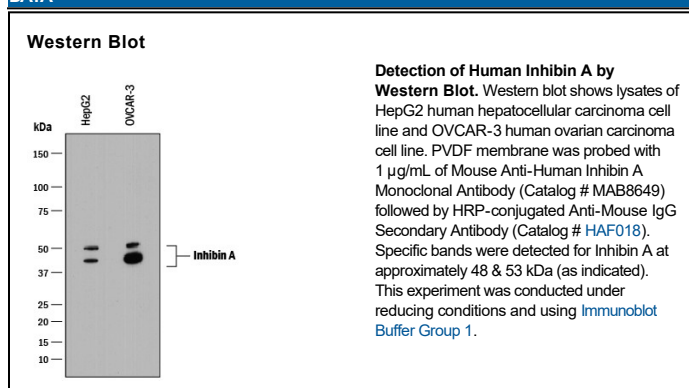
Species Reactivity	Human
Specificity	Detects human Inhibin A in direct ELISAs. In direct ELISAs, no cross-reactivity with recombinant human Activin A is observed.
Source	Monoclonal Mouse IgG ₁ Clone # 58608
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Peptide on N-terminus of human Inhibin A protein Accession # P08476
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 either lyophilized or 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	1 µg/mL	See Below

DATA



PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.5 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	<p>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</p> <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

Activin and Inhibin are members of the TGF- β superfamily of cytokines and are involved in a wide range of biological processes including tissue morphogenesis and repair, fibrosis, inflammation, neural development, hematopoiesis, reproductive system function, and carcinogenesis (1-7). Activin and Inhibin are produced as precursor proteins. Their amino terminal propeptides are proteolytically cleaved and facilitate formation of disulfide-linked dimers of the bioactive proteins (8, 9). Activins are nonglycosylated homodimers or heterodimers of various β subunits (β A, β B, β C, and β E in mammals), while Inhibins are heterodimers of a unique α subunit and one of the β subunits. Activin A is a widely expressed homodimer of two β A chains. The β A subunit can also heterodimerize with a β B or β C subunit to form Activin AB and Activin AC, respectively (10). The 14 kDa mature human β A chain shares 100% amino acid sequence identity with bovine, feline, mouse, porcine, and rat β A. Activin A exerts its biological activities by binding to the type 2 serine/threonine kinase Activin RIIA which then noncovalently associates with the type 1 serine/threonine kinase Activin RIB/ALK-4 (7, 11). Signaling through this receptor complex leads to Smad activation and regulation of activin-responsive gene transcription (7, 11). The bioactivity of Activin A is regulated by a variety of mechanisms (11). BAMBI, Betaglycan, and Cripto are cell-associated molecules that function as decoy receptors or limit the ability of Activin A to induce receptor complex assembly (12-14). The intracellular formation of Activin A can be prevented by the incorporation of the β A subunit into Activin AC or Inhibin A (3, 10). And the bioavailability of Activin A is restricted by its incorporation into inactive complexes with α 2-Macroglobulin, Follistatin, and FLRG (15, 16).

References:

1. Kumanov, P. *et al.* (2005) *Reprod. Biomed. Online* **10**:786.
2. Maeshima, A. *et al.* (2008) *Endocr. J.* **55**:1.
3. Rodgarkia-Dara, C. *et al.* (2006) *Mutat. Res.* **613**:123.
4. Werner, S. and C. Alzheimer (2006) *Cytokine Growth Factor Rev.* **17**:157.
5. Xu, P. and A.K. Hall (2006) *Dev. Biol.* **299**:303.
6. Shav-Tal, Y. and D. Zipori (2002) *Stem Cells* **20**:493.
7. Chen, Y.G. *et al.* (2006) *Exp. Biol. Med.* **231**:534.
8. Gray, A.M. and A.J. Mason (1990) *Science* **247**:1328.
9. Mason, A.J. *et al.* (1996) *Mol. Endocrinol.* **10**:1055.
10. Thompson, T.B. *et al.* (2004) *Mol. Cell. Endocrinol.* **225**:9.
11. Harrison, C.A. *et al.* (2005) *Trends Endocrinol. Metab.* **16**:73.
12. Onichtchouk, D. *et al.* (1999) *Nature* **401**:480.
13. Gray, P.C. *et al.* (2002) *Mol. Cell. Endocrinol.* **188**:254.
14. Kelber, J.A. *et al.* (2008) *J. Biol. Chem.* **283**:4490.
15. Phillips, D.J. *et al.* (1997) *J. Endocrinol.* **155**:65.
16. Schneyer, A. *et al.* (2003) *Endocrinology* **144**:1671.