

Polyclonal Antibody to XIAP



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Polyclonal Antibody to XIAP

Catalog No : IMG-5771
Formulation : 50 ul neat serum containing 0.05% sodium azide.
Sodium azide is highly toxic.
Isotype : Rabbit Ig
Clone : N/A
Purification : Neat Serum
Species React : Gerbil, Human, Mouse, Rat
Host : Rabbit

Application
Western blot analysis: 1:1000-1:2000
IHC (paraffin): 1:1000-1:5000
IHC (frozen): Users should optimize according to model and immunodetection system used (secondary reagents)
IP: 1:50-1:200

Storage
Aliquot and store at -20°C. Avoid repeated freeze-thaw cycles.

Recommended Positive Control: spleen, lymphatic tissues, prostate, colon, many cancer cell lines

Background

XIAP [human X-linked IAP, hIAP (human IAP-like protein), MIHA, BIRC4) is a member of the family of inhibitor of apoptosis proteins (IAP). IAPs suppress mitochondria-dependent and -independent apoptosis by binding to and inhibiting caspases through their BIR domains (reviewed in Liston et al, 2003; Wright and Duckett, 2005). Resistance towards apoptosis is a hallmark of cancer cells, and overexpression of IAPs can contribute to the development of cancer through inhibiting apoptosis. In addition to at least one BIR domain, some IAP members also have a RING-type finger motif at their carboxyl-terminal. The RING finger domain of several IAPs, including XIAP, have E3 ubiquitin ligase activity and target the degradation of Smac/DIABLO through ubiquitination (Morizane et al, 2005). Smac/DIABLO is a death inducer and functions by inhibiting IAP-caspase interactions, thereby promoting apoptosis. Degradation of cell death inducers like Smac/DIABLO is thought to be a conserved mechanism by which IAPs enhance their anti-apoptotic activity, thereby promoting cell survival. XIAP is highly characterized with respect to its structure and biochemical mechanisms, and has received interest as a therapeutic target (reviewed in Schimmer, 2006). Since XIAP blocks a substantial portion of the apoptosis pathway and is associated with chemoresistance in cancer cells, inhibiting XIAP has been a focus for potential therapeutics. Approaches have included antisense oligonucleotides and small molecule inhibitors. Small molecules that target the BIR2 and BIR3 domains of XIAP are considered particularly attractive. This is because the BIR domains inhibit caspase activity, and it is thought that removing the inhibition should increase the cell's ability to undergo apoptosis as well as decrease its potential for chemoresistance. IMG-5771 recognizes XIAP. Full-length human XIAP is a 497 amino acid protein and migrates at ~53 kDa on SDS-PAGE.

Antigen

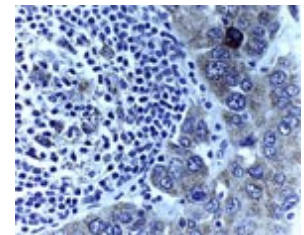
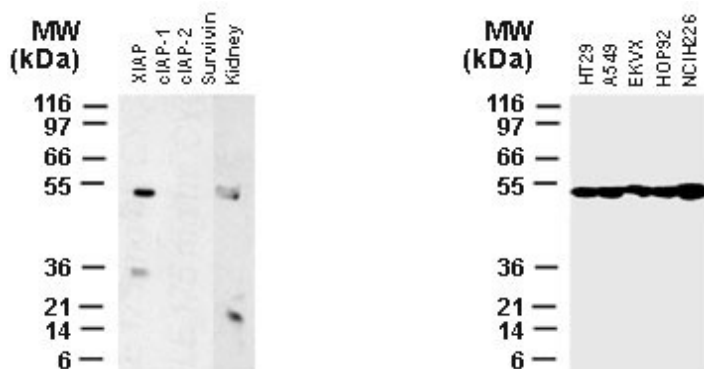
Recombinant BIR2 domain protein fragment of human XIAP was used as immunogen. The BIR2 domain used for immunogen corresponds to amino acids 163-230 of human XIAP (Deveraux et al, 1999).

Application Notes

1. The antibody recognizes epitopes in the BIR2 domain of XIAP. Therefore it can recognize full-length XIAP and XIAP cleavage fragments containing the BIR2 domain. However, XIAP cleavage fragments may be biologically unstable, and therefore cleavage fragments may be difficult to detect. Please refer to Deveraux et al, 1999 for more information on the biology of XIAP cleavage fragments.

Genebank Info (Protein)

NP_001158



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Western blot analysis of XIAP using polyclonal antibody to XIAP (IMG-5700) at 1:2000. Full-length recombinant XIAP was recognized; the antibody did not recognize the other recombinant IAP proteins (e.g. cIAP1, cIAP2, survivin). The antibody detected full-length XIAP in human kidney tissue lysate.

Western blot analysis of XIAP in various tumor cell lines recombinant IMG-5771 at 1:2000.

Immunohistochemical analysis of XIAP in formalin-fixed, paraffin-embedded human esophageal carcinoma using IMG-5771 at 1:2000. Hematoxylin-eosin counterstain.

Reference

1. Deveraux QL, E Leo, HR Stennicke, K Welsh, GS Salvesen, and JC Reed. 1999. Cleavage of human inhibitor of apoptosis protein XIAP results in fragments with distinct specificities for caspases. *EMBO* 18:5242-5251.
2. Wright CW and CS Duckett. 2005. Reawakening the cellular death program in neoplasia through the therapeutic blockade of IAP function. *J Clin Investigation*. 115:2673-2678.
3. Liston P, WG Fong and RG Korneluk. 2003. The inhibitors of apoptosis: there is more to life than Bcl2. *Oncogene*. 22:8568-8580.
4. Morizane Y, R Honda, K Fukami, and H Yasuda. 2005. X-linked inhibitor of apoptosis functions as ubiquitin ligase toward mature caspase-0 and cytosolic Smac/DIABLO. *J. Biochem*. 137:125-132.
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