Human IL-18 BPa Antibody

Monoclonal Mouse IgG₁ Clone # 136007

Catalog Number: MAB1191

DESCRIPTION	
Species Reactivity	Human
Specificity	Detects human IL-18 BPa in direct ELISAs and Western blots. In direct ELISAs and Western blots, no cross-reactivity with recombinant mouse (rm) IL-18 BPc or rmIL-18 BPd is observed.
Source	Monoclonal Mouse IgG ₁ Clone # 136007
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant human IL-18 BPa Thr29-Gly192 Accession # AAD17190
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. *Small pack size (-SP) is supplied as a 0.2 µm filtered solution in PBS.

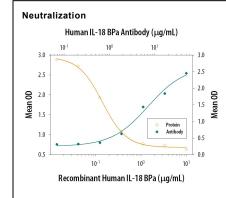
APPLICATIONS

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.

Neutralization

Measured by its ability to neutralize IL-18 BPa inhibition of IL-18/IL-1F4-induced IFN- γ secretion in the KG-1 human acute myelogenous leukemia cell line. The Neutralization Dose (ND₅₀) is typically 5-20 µg/mL in the presence of 1 µg/mL Recombinant Human IL-18 BPa Fc Chimera, 40 ng/mL Recombinant Human IL-18/IL-1F4, and 20 ng/mL Recombinant Human TNF- α .

DATA



IL-18 BPa Inhibition of IL-18/IL-1F4-induced IFN-v Secretion and Neutralization by Human IL-18 BPa Antibody. Recombinant Human IL-18 BPa Fc Chimera (Catalog # 119-BP) inhibits Recombinant Human IL-18/IL-1F4 induced IFN-v secretion in the KG-1 human acute myelogenous leukemia cell line in a dose-dependent manner (orange line), as measured by the Human IFN-y Quantikine ELISA Kit (Catalog # DIF50). Inhibition of Recombinant Human IL-18/IL-1F4 (40 ng/mL) activity elicited by Recombinant Human IL-18 BPa Fc Chimera (1 $\mu g/mL$) is neutralized (green line) by increasing concentrations of Mouse Anti-Human IL-18 BPa Monoclonal Antibody (Catalog # MAB1191). The ND₅₀ is typically 5-20 µg/mL.

• 6 months, -20 to -70 °C under sterile conditions after reconstitution.

PREPARATION AND STORAGE

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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	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.





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BACKGROUND

Interleukin 18 binding protein (IL-18 BP or IL-18 BPa) is a secreted glycoprotein, which functions as an IL-18 antagonist by binding to IL-18 and blocking its biological activity. IL-18 BPa bears no amino acid sequence homology to the membrane-associated IL-18 and IL-1 receptor proteins. The gene for human IL-18 BPa has been localized to chromosome 11q13. It encodes for at least four isoforms by alternative splicing. The IL-18 BP isoforms a and c each contain one immunoglobulin (Ig)-like C2-type domain while isoforms b and d lack a complete Ig domain. The complete Ig domain has been shown to be essential to the binding and neutralizing properties of the binding proteins. Two isoforms of mouse IL18 BP (c and d) containing the complete Ig domain have also been isolated and shown to neutralize IL-18 bioactivity. Human and mouse IL-18 BPa share approximately 61% amino acid sequence identity. Several poxviruses also encode proteins with sequence similarity to the human and mouse IL-18 BPa. Viral IL-18 BPs have been shown to bind and inhibit IL-18 responses and may be involved in modulating host immune responses. The expression of IL-18 BP is markedly upregulated by IFN-γ, suggesting that IL-18 activity is modulated by a negative feedback mechanism mediated by IL-18 BP.

References:

- 1. Mühl, H. et al. (2000) Biochem. Biophys. Res. Commun. 267:960.
- Kim, S-H. et al. (2000) Proc. Nat. Acad. Sci. USA 97:1190.
- Calderara, S. et al. (2001) Virology 279:22.

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