

Human IL-18 BPa Antibody

Antigen Affinity-purified Polyclonal Goat IgG Catalog Number: AF119

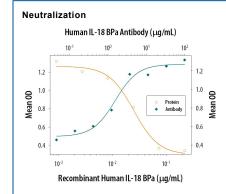
DESCRIPTION			
Species Reactivity	Human		
Specificity	Detects human IL-18 BPa in direct ELISAs and Western blots. In Western blots, less than 1% cross-reactivity with recombinant mouse (rm) IL-18 BPc and rmIL-18 BPd is observed.		
Source	Polyclonal Goat IgG		
Purification	Antigen Affinity-purified		
Immunogen	Mouse myeloma cell line NS0-derived recombinant human IL-18 BPa Thr31-Gly194 Accession # O95998		
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.

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	Recommended Concentration	Sample	
Western Blot	0.1 μg/mL	Recombinant Human IL-18 BPa Fc Chimera (Catalog # 119-BP)	
Neutralization	Measured by its ability to neutralize IL-18 BPa inhibition of IL-18/IL-1F4-induced IFN γ secretion in the KG-1 hum acute myelogenous leukemia cell line. The Neutralization Dose (ND $_{50}$) is typically 1-5 µg/mL in the presence of		
	0.1 µg/mL Recombina	int Human IL-18 BPa Fc Chimera, 40 ng/mL Recombinant Human IL-18/IL-1F4, and	
	20 ng/mL Recombina	nt Human TNF-α.	

DATA



IL-18 BPa Inhibition of IL-18/IL-1F4-induced IFN-γ 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-γ 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 (0.1 µg/mL) is neutralized (green line) by increasing concentrations of Goat Anti-H u m a n I L-18 BPa Antigen Affinity-purified Polyclonal Antibody (Catalog # AF119). The \mbox{ND}_{50} is typically 1-5 $\mu\mbox{g/mL}$ in the presence of Recombinant Human TNF- α (20 ng/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.





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BACKGROUND

Interleukin 18 binding protein (IL-18 BP) is a secreted glycoprotein, which functions as an IL-18 antagonist by binding to IL-18 and blocking its biological activity. IL-18 BP bears no amino acid sequence homology to the membrane-associated IL-18 and IL-1 receptor proteins. The gene for human IL-18 BP 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 IL-18 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 BPs share approximately 61% amino acid sequence identity. Several poxviruses also encode proteins with sequence similarity to the human and mouse IL-18 BP. 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 up-regulated 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.
- 2. Kim, S-H. et al. (2000) Proc. Nat. Acad. Sci. USA 97:1190.
- 3. Calderara, S. et al. (2001) Virology 279:22.

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