

Human IL-8/CXCL8 Antibody

Monoclonal Mouse IgG_{2B} Clone # 1028311 Catalog Number: MAB2082

DESCRIPTION	
Species Reactivity	Human
Specificity	Detects human IL-8/CXCL8 in direct ELISAs.
Source	Monoclonal Mouse IgG _{2B} Clone # 1028311
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	E. coli-derived human IL-8/CXCL8 Ser28-Ser99 Accession # P10145.1
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

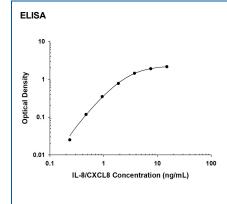
Please Note: Optimal dilutions should be determined by each laboratory for each application. General Protocols are available in the Technical Information section on our website.

ELISA

This antibody functions as an ELISA detection antibody when paired with Mouse Anti-Human IL-8/CXCL8 Monoclonal Antibody (Catalog # MAB2081).

This product is intended for assay development on various assay platforms requiring antibody pairs. We recommend the Human IL-8/CXCL8 DuoSet ELISA Kit (Catalog # DY208) for convenient development of a sandwich ELISA or the Human IL-8/CXCL8 Quantikine ELISA Kit (Catalog # D8000C) for a complete optimized ELISA.

DATA



Human IL-8/CXCL8 ELISA Standard Curve. Recombinant Human IL-8/CXCL8 protein was serially diluted 2-fold and captured by Mouse Anti-Human IL-8/CXCL8 Monoclonal Antibody (Catalog # MAB2081) coated on a Clear Polystyrene Microplate (Catalog # DY990). Mouse Anti-Human IL-8/CXCL8 Monoclonal Antibody (Catalog # MAB2082) was biotinylated and incubated with the protein captured on the plate. Detection of the standard curve was achieved by incubating Streptavidin-HRP (Catalog # DY998) followed by Substrate Solution (Catalog # DY999) and stopping the enzymatic reaction with Stop Solution (Catalog # DY994).

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 Use a manua

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-8 (IL-8), also known as CXCL8, GCP-1, and NAP-1, is a widely expressed proinflammatory member of the CXC family of chemokines. Near its N-terminus, this 8-9 kDa chemokine contains an ELR motif which is important for its angiogenic properties (1). IL-8/CXCL8 can associate into a homodimer or a heterodimer with CXCL4/PF4 (2), and it can also interact with matrix and cell surface glycosaminoglycans (3). Mature human IL-8/CXCL8 shares 65%-69% amino acid (aa) sequence identiity with canine, feline, and porcine IL-8/CXCL8 (4). There is no IL-8/CXCL8 gene counterpart in rodent. N-terminal truncation by multiple proteases generates a range of shorter forms, and an alternative splice form of human IL-8/CXCL8 carries an eleven as substitution at the C-terminus (5). The bioactivity of IL-8/CXCL8 is regulated by these truncations, by IL-8/CXCL8 citrullination at Arg5 (N-terminal to the ELR motif) (6), and by the decoy receptor DARC (7). IL-8/CXCL8 effects are mediated through CXCR1/IL-8 RA, which is also used by CXCL6, and through CXCR2/IL-8 RB, which is used by multiple CXC chemokines (1). CXCR1 and CXCR2 associate into functional homodimers and heterodimers with each other (8). Through both CXCR1 and CXCR2, CXCL8 promotes neutrophil adhesion to the vascular endothelium and migration to sites of inflammation (9). It triggers the antimicrobial activation of neutrophils through CXCR1 (10). CXCL8 also binds to Serpin A1/alpha-1 Antitrypsin, and this prevents IL-8/CXCL8 interaction with CXCR1 (11). IL-8/CXCL8 is upregulated in atherosclerotic lesions and other cardiac pathologies where it exacerbates inflammatory tissue damage (12). In addition, it induces VEGF expression, vascular endothelial cell proliferation, angiogenesis, and tumor cell invasiveness (13-16).

References:

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