

**DESCRIPTION**

<b>Species Reactivity</b>	Human
<b>Specificity</b>	Detects human IL-8/CXCL8 in direct ELISAs.
<b>Source</b>	Monoclonal Mouse IgG <sub>1</sub> Clone # 1028336
<b>Purification</b>	Protein A or G purified from hybridoma culture supernatant
<b>Immunogen</b>	<i>E. coli</i> -derived human IL-8/CXCL8 Ser28-Ser99 Accession # P10145.1
<b>Formulation</b>	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.

	<b>Recommended Concentration</b>	<b>Sample</b>
<b>Western Blot</b>	3 µg/mL	THP-1 human acute monocytic leukemia cell line treated with LPS and PMA
<b>Simple Western</b>	50 µg/mL	PBMC conditioned media treated with PMA, ionomycin, and LPS

**DATA**

**Western Blot**

**Detection of Human IL-8/CXCL8 by Western Blot.** Western blot shows lysates of THP-1 human acute monocytic leukemia cell line untreated (-) or treated (+) with 200 nM PMA for 24 hours and 10 µg/mL LPS for 3 hours. PVDF membrane was probed with 3 µg/mL of Mouse Anti-Human IL-8/CXCL8 Monoclonal Antibody (Catalog # MAB2083) followed by HRP-conjugated Anti-Mouse IgG Secondary Antibody (Catalog # HAF018). A specific band was detected for IL-8/CXCL8 at approximately 11 kDa (as indicated). This experiment was conducted under reducing conditions and using Western Blot Buffer Group 1.

**Simple Western**

**Detection of Human IL-8/CXCL8 by Simple Western™.** Simple Western lane view shows PBMC conditioned media untreated (-) or treated (+) with 2 µg/mL LPS for 24 hours, 200 ng/mL PMA for 24 hours, and 10 µg/mL ionomycin for 3 hours, loaded at 0.2 mg/mL. A specific band was detected for IL-8/CXCL8 at approximately 9 kDa (as indicated) using 50 µg/mL of Mouse Anti-Human IL-8/CXCL8 Monoclonal Antibody (Catalog # MAB2083). This experiment was conducted under reducing conditions and using the 2-40 kDa separation system.

**PREPARATION AND STORAGE**

<b>Reconstitution</b>	Reconstitute at 0.5 mg/mL in sterile PBS.
<b>Shipping</b>	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
<b>Stability &amp; Storage</b>	<b>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</b> <ul style="list-style-type: none"> <li>• 12 months from date of receipt, -20 to -70 °C as supplied.</li> <li>• 1 month, 2 to 8 °C under sterile conditions after reconstitution.</li> <li>• 6 months, -20 to -70 °C under sterile conditions after reconstitution.</li> </ul>

**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 identity 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 aa 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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