

**DESCRIPTION**

<b>Species Reactivity</b>	Human
<b>Specificity</b>	Detects human IL-6 in direct ELISAs.
<b>Source</b>	Recombinant Monoclonal Rabbit IgG Clone # 2828D
<b>Purification</b>	Protein A or G purified from cell culture supernatant
<b>Immunogen</b>	<i>E. coli</i> -derived human IL-6 protein Pro29-Met212 Accession # Q75MH2
<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.

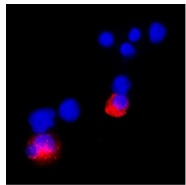
**APPLICATIONS**

**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.

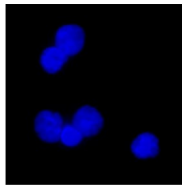
	<b>Recommended Concentration</b>	<b>Sample</b>
<b>Immunocytochemistry</b>	8-25 µg/mL	Immersion fixed human peripheral blood mononuclear cells (PBMCs) treated with PHA
<b>Immunohistochemistry</b>	3-25 µg/mL	Immersion fixed paraffin-embedded sections of human tonsil

**DATA**

**Immunocytochemistry**



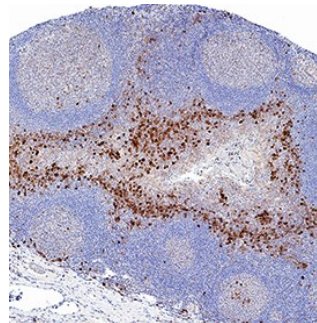
Positive (hPBMC + PHA)



Negative (hPBMC)

**IL-6 in Human PBMCs.** IL-6 was detected in immersion fixed human peripheral blood mononuclear cells (PBMCs) treated with PHA (positive staining) and untreated PBMCs (negative control) using Rabbit Anti-Human IL-6 Monoclonal Antibody (Catalog # MAB95402) at 8 µg/mL for 3 hours at room temperature. Cells were stained using the NorthernLights™ 557-conjugated Anti-Rabbit IgG Secondary Antibody (red; Catalog # NL004) and counterstained with DAPI (blue). Specific staining was localized to cell cytoplasm. Staining was performed using our protocol for Fluorescent ICC Staining of Non-adherent Cells.

**Immunohistochemistry**



**IL-6 in Human Tonsil.** IL-6 was detected in immersion fixed paraffin-embedded sections of human tonsil using Rabbit Anti-Human IL-6 Monoclonal Antibody (Catalog # MAB95402) at 3 µg/mL for 1 hour at room temperature followed by incubation with the Anti-Rabbit IgG VisUCyte™ HRP Polymer Antibody (Catalog # VC003). Before incubation with the primary antibody, tissue was subjected to heat-induced epitope retrieval using Antigen Retrieval Reagent-Basic (Catalog # CTS013). Tissue was stained using DAB (brown) and counterstained with hematoxylin (blue). Specific staining was localized to lymphocytes. Staining was performed using our protocol for IHC Staining with VisUCyte HRP Polymer Detection Reagents.

**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-6 (IL-6) is a pleiotropic,  $\alpha$ -helical, 22-28 kDa phosphorylated and variably glycosylated cytokine that plays important roles in the acute phase reaction, inflammation, hematopoiesis, bone metabolism, and cancer progression (1-5). Mature human IL-6 is 183 amino acids (aa) in length and shares 39% aa sequence identity with mouse and rat IL-6 (6). Alternative splicing generates several isoforms with internal deletions, some of which exhibit antagonistic properties (7-10). IL-6 induces signaling through a cell surface heterodimeric receptor complex composed of a ligand binding subunit (IL-6 R  $\alpha$ ) and a signal transducing subunit (gp130). IL-6 binds to IL-6 R  $\alpha$ , triggering IL-6 R  $\alpha$  association with gp130 and gp130 dimerization (11). gp130 is also a component of the receptors for CLC, CNTF, CT-1, IL-11, IL-27, LIF, and OSM (12). Soluble forms of IL-6 R  $\alpha$  are generated by both alternative splicing and proteolytic cleavage (5). In a mechanism known as trans-signaling, complexes of soluble IL-6 and IL-6 R  $\alpha$  elicit responses from gp130-expressing cells that lack cell surface IL-6 R  $\alpha$  (5). Trans-signaling enables a wider range of cell types to respond to IL-6, as the expression of gp130 is ubiquitous, while that of IL-6 R  $\alpha$  is predominantly restricted to hepatocytes, monocytes, and resting lymphocytes (2, 5). Soluble splice forms of gp130 block trans-signaling from IL-6/IL-6 R  $\alpha$  but not from other cytokines that use gp130 as a co-receptor (5, 13). IL-6, along with TNF- $\alpha$  and IL-1, drives the acute inflammatory response and the transition from acute inflammation to either acquired immunity or chronic inflammatory disease (1-5). When dysregulated, it contributes to chronic inflammation in obesity, insulin resistance, inflammatory bowel disease, arthritis, sepsis, and atherosclerosis (1, 2, 5). IL-6 can also function as an anti-inflammatory molecule, as in skeletal muscle where it is secreted in response to exercise (2). In addition, it enhances hematopoietic stem cell proliferation and the differentiation of Th17 cells, memory B cells, and plasma cells (1, 14).

**References:**

1. Mansell, A. and B.J. Jenkins (2013) Cytokine Growth Factor Rev. **24**:249.
2. Schuett, H. *et al.* (2009) Thromb. Haemost. **102**:215.
3. Erta, M. *et al.* (2012) Int. J. Biol. Sci. **8**:1254.
4. Garbers, C. *et al.* (2012) Cytokine Growth Factor Rev. **23**:85.
5. Mihara, M. *et al.* (2012) Clin. Sci. (Lond.) **122**:143.
6. Hirano, T. *et al.* (1986) Nature **324**:73.
7. Kestler, D.P. *et al.* (1995) Blood **86**:4559.
8. Kestler, D.P. *et al.* (1999) Am. J. Hematol. **61**:169.
9. Bihl, M.P. *et al.* (2002) Am. J. Respir. Cell Mol. Biol. **27**:48.
10. Alberti, L. *et al.* (2005) Cancer Res. **65**:2.
11. Murakami, M. *et al.* (1993) Science **260**:1808.
12. Muller-Newen, G. (2003) Sci. STKE **2003**:PE40.
13. Mitsuyama, K. *et al.* (2006) Clin. Exp. Immunol. **143**:125.
14. Cerutti, A. *et al.* (1998) J. Immunol. **160**:2145.