

## DESCRIPTION

<b>Species Reactivity</b>	Human
<b>Specificity</b>	Detects human Complement Component C3a in direct ELISAs and Western blots.
<b>Source</b>	Polyclonal Goat IgG
<b>Purification</b>	Antigen Affinity-purified
<b>Immunogen</b>	<i>E. coli</i> -derived recombinant human C3a Ser672-Arg748 Accession # P01024
<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 as a 0.2 µm filtered solution in PBS.

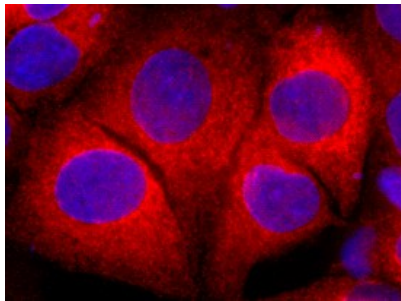
## APPLICATIONS

**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>	0.1 µg/mL	Recombinant Human Complement Component C3a (Catalog # 3677-C3)
<b>Immunocytochemistry</b>	5-15 µg/mL	See Below

## DATA

### Immunocytochemistry



#### Complement Component C3a in HepG2 Human Cell Line.

Complement Component C3a was detected in immersion fixed HepG2 human hepatocellular carcinoma cell line using Goat Anti-Human Complement Component C3a Antigen Affinity-purified Polyclonal Antibody (Catalog # AF3677) at 10 µg/mL for 3 hours at room temperature. Cells were stained using the NorthernLights™ 557-conjugated Anti-Goat IgG Secondary Antibody (red; Catalog # NL001) and counterstained with DAPI (blue). Specific staining was localized to cytoplasm. View our protocol for [Fluorescent ICC Staining of Cells on Coverslips](#).

## PREPARATION AND STORAGE

<b>Reconstitution</b>	Reconstitute at 0.2 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**

C3a is an anaphylotoxin polypeptide comprising amino acids (aa) 672-748 of the Complement C3 precursor protein (1-4). Anaphylatoxins are proteolytically generated from the C3, C4 and C5 alpha chains by convertases formed by other complement fragments (2). They share 30 - 36% aa identity, and mediate inflammatory responses that vary in strength in the order C5a > C3a > C4a (2). Like C4a and C5a, the 77 aa, 9 kDa human C3a contains six conserved cysteine residues that form a knot structure and possess an overall basic charge (4, 5). It is not glycosylated (4). The C-terminal regions of C3a and C4a, but not C5a, shows antimicrobial activity (5). Human C3a shows 67-69% aa identity with mouse, rat, guinea pig, bovine, porcine and canine C3a. C3a formation is common to all three pathways of complement activation: classical (antibody-mediated), lectin and alternative (1, 2). It binds the G-protein coupled C3a receptor (C3aR) on myeloid peripheral blood leukocytes, and on activated lymphocytes, endothelial and internal organ epithelial cells (7, 10). C3a contributes to both innate and adaptive immunity. It activates mast cells and neutrophils, triggering robust mast cell degranulation in airways during asthmatic allergen challenges (9). It enhances lipopolysaccharide-induced prostaglandin, cytokine and chemokine secretion by macrophages and other cells (1, 6-8). It assists in Th2-type inflammatory reactions and stimulates smooth muscle contraction and leukocyte chemotaxis (8, 9). Endogenous carboxypeptidase-N can remove the arginine at the C-terminus of the anaphylatoxins to create desArg forms (1). C3adesArg, also called ASP (Acylation-Stimulating Protein) is an adipocyte-derived protein that binds the C5L2 (GPR77) receptor and stimulates adipose tissue triglyceride synthesis (2, 10, 11). The anaphylactic activity of ASP is weaker than that of C3a (6, 10). C5L2 is also involved in C3a and C5a activity (11).

**References:**

1. Markiewski, M. and J.D. Lambris (2007) *Am. J. Pathol.* **171**:715.
2. Haas, P.-J. and J. van Strijp (2007) *Immunol. Res.* **37**:161.
3. DeBrujin, M.H. and G.H. Fey (1985) *Proc. Natl. Acad. Sci. USA* **82**:708.
4. Hugli, T.E. (1975) *J. Biol. Chem.* **250**:8293.
5. Pasupuleti, M. *et al.* (2007) *J. Biol. Chem.* **282**:2520.
6. Fischer, W.H. *et al.* (1999) *J. Immunol.* **162**:453.
7. Thurman, J.M. *et al.* (2007) *J. Immunol.* **178**:1819.
8. Honczarenko, M. *et al.* (2005) *J. Immunol.* **175**:3698.
9. Ali, H. and R.A. Panettieri (2005) *Respir. Res.* **6**:19.
10. Kalant, D. *et al.* (2005) *J. Biol. Chem.* **280**:23936.
11. Chen, N.-J. *et al.* (2007) *Nature* **446**:203.