

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human Azurocidin/CAP37/HBP in direct ELISAs and Western blots.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant human Azurocidin/CAP37/HBP Ile27-Pro250 Accession # P20160
Conjugate	Alexa Fluor 594 Excitation Wavelength: 590 nm Emission Wavelength: 617 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

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.

Western Blot	Optimal dilution of this antibody should be experimentally determined.
Immunoprecipitation	Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

BACKGROUND

Azurocidin, also known as cationic antimicrobial protein 37 (CAP37) and heparin-binding protein (HBP), is a member of the serine protease family that includes Cathepsin G, neutrophil elastase (NE), and proteinase 3 (PR3). These proteases are found in the specialized azurophilic granules of neutrophils (1, 2). Human Azurocidin 1 is encoded by the AZU1 gene located in a cluster with NE and PR3 on chromosome 19pter (2). The open reading frame predicts a 251 amino acid (aa) protein with an N-terminal 26 aa signal sequence and a 7 aa propeptide. There are also eight cysteine residues and 3 putative N-linked glycosylation sites (1).

Although Azurocidin 1 shares a significant degree of aa sequence identity with Cathepsin G, NE, and PR3, it lacks serine protease activity due to mutations at two of the three residues in the catalytic triad (His41Ser and Ser175Gly) (1, 3). Crystallographic analysis suggests that the antibacterial activity of Azurocidin is mediated by a hydrophobic pocket (residues 20 to 44) that binds Gram-negative bacteria lipid A. These structural data also imply that the heparin binding capacity is mediated by non-specific electrostatic interactions between the negatively charged heparin molecule and a large patch of positively charged residues near the lipid A binding site (3).

Azurocidin has also been identified as a modulator of endothelial permeability. Neutrophils arriving first at sites of inflammation release Azurocidin, which acts in a paracrine fashion on endothelial cells causing the development of intercellular gaps and allowing leukocyte extravasation. These findings imply that Azurocidin may be a reasonable therapeutic target for a variety of inflammatory disease conditions (4).

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