Recombinant Human Azurocidin/CAP37/HBP

Catalog Number: 2200-SE/CF

DESCRIPTION

Source	Mouse myeloma cell line, NS0-derived Ile27-Pro250, with a C-terminal 10-His tag Accession # P20160
N-terminal Sequence Analysis	Ile27
Predicted Molecular Mass	26 kDa

SPECIFICATIONS

SDS-PAGE	39 kDa, reducing conditions
Activity	Measured by its ability to enhance LPS-induced TNF-α secretion from human monocytes. Rasmussen, P.B. *et al.* (1996) FEBS Letters **390**:109. The ED_{50} for this effect is 0.5-3 µg/mL.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE under reducing conditions and visualized by silver stain.
Formulation	Lyophilized from a 0.2 µm filtered solution in HEPES and NaCl. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 200 µg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** ● 12 months from date of receipt, -70 °C as supplied. ● 3 months, -20 to -70 °C under sterile conditions after reconstitution.

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).

References:

1. Morgan, J.G. *et al.* (1991) J. Immunol. **147**:3210.
2. Zimmer, M. *et al.* (1992) Proc. Natl. Acad. Sci. USA **89**:8215.
3. Iverson, L.F. *et al.* (1997) Nat. Struct. Biol. **4**:265.
4. Gautam, N. *et al.* (2001) Nat. Med. **7**:1123.

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