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). 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 CXCL8 shares 65%-69% amino acid (aa) sequence identity with canine, feline, and porcine CXCL8 (4). There is no CXCL8 gene counterpart in rodents. N-terminal truncation by multiple proteases generates a range of shorter forms, and an alternative splice form of human CXCL8 carries an eleven aa substitution at the C-terminus (5). The bioactivity of CXCL8 is regulated by these truncations, by CXCL8 citrullination at Arg5 (N-terminal to the ELR motif) (6), and by the decoy receptor DARC (7). 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 CXCL8 interaction with CXCR1 (11). 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: