

## Product datasheet for SA1012

### Interleukin-8 / IL8 Equine Protein

#### Product data:

Product Type:	Recombinant Proteins
Description:	Interleukin-8 / IL8 equine protein, 20 µg
Species:	Equine
Expression Host:	Pichia pastoris
Predicted MW:	8.5 kDa
Purity:	>95% by SDS PAGE analysis
Buffer:	Presentation State: Purified State: Lyophilized purified Protein Buffer System: PBS
Bioactivity:	Specific: Horse.
Reconstitution Method:	Restore with 0.5 ml distilled water. Further dilutions should be made in a buffer containing carrier protein. Care should be taken during reconstitution as the protein may appear as a film at the bottom of the vial. We recommend that the vial is gently mixed after reconstitution.
Preparation:	Lyophilized purified Protein
Applications:	Functional Assays.
Protein Description:	Equine interleukin 8 (IL-8) is principally produced by macrophages and is involved in neutrophil chemotaxis. This protein has an ED50 in the range 20-25 ng/ml determined by studies of the induction of equine IL-8 on neutrophil migration in vitro.
Storage:	Prior to reconstitution store at 2-8°C. Following reconstitution store at -20°C. Avoid repeated freezing and thawing.
Stability:	Shelf life: one year from despatch.
RefSeq:	<a href="#">NP_001077420</a>



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**Summary:**

Interleukin 8, IL8 is a member of the CXC chemokine family. This family of small basic heparan-binding proteins are proinflammatory and primarily mediate the activation and migration of neutrophils into tissue from peripheral blood.

This chemokine is one of the major mediators of the inflammatory response and is secreted by several cell types in response to an inflammatory stimulus. It functions as a chemoattractant, and is also a potent angiogenic factor. IL8 attracts neutrophils, basophils, and T-cells, but not monocytes.

Cystic fibrosis (CF) is characterized by severe lung inflammation. The inflammatory process is believed to be caused by massive overproduction of the proinflammatory protein IL8, and the high levels of IL8 in the CF lung are therefore believed to be the central mechanism behind CF lung pathophysiology.