

DESCRIPTION

Source Mouse myeloma cell line, NS0-derived human Serpin A6 protein
Met23-Val405, with a C-terminal 6-His tag
Accession # AAH56259.1

N-terminal Sequence Analysis Met23

Predicted Molecular Mass 43 kDa

SPECIFICATIONS

SDS-PAGE 50-70 kDa, reducing conditions

Activity Measured by its binding ability in a functional ELISA.
When Cortisol-BSA Conjugate is immobilized at 5 µg/mL, 100 µL/well, Recombinant Human Serpin A6 binds with an ED₅₀ of 1.5-7.5 µg/mL.

Endotoxin Level <0.10 EU per 1 µg of the protein by the LAL method.

Purity >95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.

Formulation Lyophilized from a 0.2 µm filtered solution in Tris, NaCl and Trehalose. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 1 mg/mL in water.

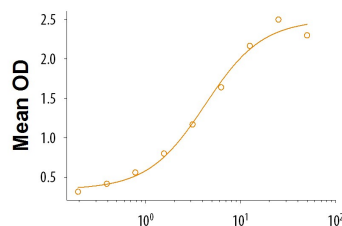
Shipping The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.

Stability & Storage Use a manual defrost freezer and avoid repeated freeze-thaw cycles.

- 12 months from date of receipt, -20 to -70 °C as supplied.
- 1 month, 2 to 8 °C under sterile conditions after reconstitution.
- 3 months, -20 to -70 °C under sterile conditions after reconstitution.

DATA

Bioactivity



Recombinant Human Serpin A6 Protein Bioactivity When Cortisol-BSA Conjugate is immobilized at 5 µg/mL, 100 µL/well, Recombinant Human Serpin A6 (Catalog # 9580-PI) binds with an ED₅₀ of 1.5-7.5 µg/mL.

BACKGROUND

Corticosteroid-binding globulin (CBG), also known as Serpin A6, is an approximately 42 kDa member of the serpin superfamily of serine protease inhibitors. CBG belongs to the clade A subgroup of serpins which include inflammatory response molecules (1). Human CBG shares 57% sequence identity to mouse CBG. CBG is one of two serpins known to bind and transport hormones. CBG is responsible for binding ~90% of cortisol in a reversible manner to regulate levels under normal physiological states (2). However, cleavage of CBG's reactive site loop by proteases present during an inflammatory response leads to a targeted and immediate release of the anti-inflammatory steroid, cortisol, from CBG (3). Reduced cleavage of CBG is implicated in the inflammatory phenotype seen in obesity, metabolic syndrome, and rheumatoid arthritis (4, 5). Cleavage of CBG can be modulated by glycosylation (6). Naturally occurring mutations of CBG lead to abnormal levels or function (7, 8). Loss of functional CBG results in low cortisol levels in patients experiencing chronic pain, fatigue, hypotension and excess weight (8).

References:

1. Law, R. *et al.* (2006) *Genome Biol.* **7**:216.
2. Lewis, J. G. *et al.* (2005) *Clin. Chem. Acta.* **359**:189.
3. Klieber M.A. *et al.* (2007) *J. Biol. Chem.* **282**:29594.
4. Nenke, M.A. *et al.* (2016) *Horm. Metab. Res.* **48**:523.
5. Nenke, M.A. *et al.* (2016) *Clin. Endocrinol.* **85**:369.
6. Sumer-Bayraktar, Z. *et al.* (2016) *J. Biol. Chem.* **291**:17727.
7. Simard, M. *et al.* (2015) *J. Clin. Endocrinol. Metab.* **100**:E129.
8. Gagliardi, L. *et al.* (2010) *Mol. Cell. Endocrinol.* **316**:24.