Human Holo-Transferrin

Catalog Number: 2914-HT

DESCRIPTION

Source	Human plasma-derived The human plasma used for the isolation of this product were certified by the supplier to be HIV-1 and HBsAg negative at the time of shipment. Human blood products should always be treated in accordance with universal handling precautions.

SPECIFICATIONS

SDS-PAGE	76-81 kDa, reducing conditions
Activity	Measured in a serum-free cell proliferation assay using MDCK canine kidney epithelial cells. Taub, M. *et al.* (1979) PNAS **76**:3338. The ED_{50} for this effect is 0.075-0.375 µg/mL. **Optimal concentration depends on cell type as well as the application or research objectives.**
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>90%, by SDS-PAGE under reducing conditions and visualized by silver stain.
Formulation	Lyophilized from a 0.2 µm filtered solution in NH_4HCO_3. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 20 mg/mL in sterile, deionized water.
Shipping	The product is shipped with dry ice or equivalent. 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.

BACKGROUND

Human Transferrin (Tf) is a single chain, 80 kDa member of the anion-binding superfamily of proteins (1 - 5). It is a bilobed molecule that is the product of an ancient gene duplication event (1, 6). Transferrin is synthesized as a 698 amino acid (aa) precursor that is divided into a 19 aa signal sequence plus a 679 aa mature segment that contains 19 intrachain disulfide bonds. The crystal structure of Tf reveals a protein with two flanking 340 aa globular domains. Each are composed of a β-sheet surrounded by series of α-helices (1, 7). The N- and C-terminal flanking regions (or domains) will bind ferric iron through the interaction of an obligate anion (usually bicarbonate) and four amino acids (His, Asp, and two Tyr) (7, 8). Apotransferrin (or iron-free) will initially bind one atom of iron at the C-terminus, and this is followed by subsequent iron binding by the N-terminus to form holotransferrin (diferric Tf) (8, 9). Through its C-terminal iron-binding domain, holotransferrin will interact with the type I Tf receptor (TfR) on the surface of cells where it is internalized into acidified endosomes. Iron dissociates from the Tf molecule within these endosomes, and is transported into the cytosol as ferrous iron. At physiological pH, iron-free apotransferrin is not bound by TfR. But at acidic pH, such as exists in the endosome, apotransferrin has considerable affinity for TfR. Thus, it remains bound to TfR and is recycled back to the cell surface where a neutral pH environment dissociates ligand from receptor. Each Tf molecule recycles 100 - 150 times during its lifetime (8 - 11). In addition to TfR, transferrin is reported to bind to cubulin, IGFBP3, microbial iron-binding proteins and liver-specific TfR2 (7, 12, 13, 14). Transferrin is variably glycosylated and the degree of sialylation is suggestive of certain clinical conditions (15). Finally, Tf is highly allelic and the gene codominant, with many single aa changes noted. Three general forms are known, based on standard electrophoretic mobility. Fast Tf is known as transferrin B, slow transferrin is transferrin D, and the middle migrating transferrin is type/variant C, thre most common (16, 17). Mature human TF is 73% aa identical to both mouse and rat Tf, and 68% and 71% aa identical to bovine and equine Tf, respectively.

References:

1. Brus, C.M. *et al.* (2001) Nat. Struct. Biol. **4**:919.
2. Schaeffer, E. *et al.* (1987) Gene **56**:109.
3. MacGillivray, R.T.A. *et al.* (1983) J. Biol. Chem. **258**:3543.
4. Yang, F. *et al.* (1984) Proc. Natl. Acad. Sci. USA **81**:2752.
5. Uzan, G. *et al.* (1984) Biochem. Biophys. Res. Commun. **119**:273.
6. Zak, O. *et al.* (2002) Biochemistry **41**:7416.
7. Gomme, P.T. and K. B. McCann (2005) Drug Discov. Today **10**:267.
8. Liu, R. *et al.* (2003) Biochemistry **42**:12447.
9. Pakdaman, R. *et al.* (1999) J. Mol. Biol. **293**:1273.
10. Hemadi, M. *et al.* (2004) Biochemistry **43**:1736.
11. Aisen, P. *et al.* (2001) Int. J. Biochem. Cell Biol. **33**:940.
12. Kozyraki, R. *et al.* (2001) Proc. Natl. Acad. Sci. USA **98**:12941.
13. Boulton, I.C. *et al.* (1998) Biochem. J. **334**:269.
14. Robb, A. and M. Wessling-Resnick (2004) Blood **104**:4294.
15. Landberg, E. *et al.* (1995) Biochem. Biophys. Res. Commun. **210**:267.
16. Gorg, A. *et al.* (1983) Hum. Genet. **64**:222.
17. Bean, P. and J.B. Peter (1994) Clin. Chem. **40**:2078.

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