

Human AHSP / ERAF Protein

Catalog Number: 14391-HNAE



Sino Biological Inc.

Biological Solution Specialist

General Information

Gene Name Synonym:

ERAF, AHSP, EDRF

Protein Construction:

A DNA sequence encoding human ERAF (Q9NZD4) (Met1-Ser102) was expressed.

Source: Human

Expression Host: E. coli

QC Testing

Purity: > 90 % as determined by SDS-PAGE

Endotoxin:

Please contact us for more information.

Stability:

Samples are stable for up to twelve months from date of receipt at -70 °C

Predicted N terminal: Met

Molecular Mass:

The recombinant human ERAF consists of 102 amino acids and predicts a molecular mass of 11.8 KDa. It migrates as an approximately 12 KDa band in SDS-PAGE under reducing conditions.

Formulation:

Lyophilized from sterile PBS, pH 7.4.

Normally 5 % - 8 % trehalose and mannitol are added as protectants before lyophilization. Specific concentrations are included in the hardcopy of COA. Please contact us for any concerns or special requirements.

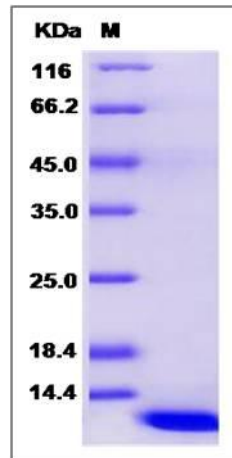
Usage Guide

Storage:

Store it under sterile conditions at -20°C to -80°C upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

Avoid repeated freeze-thaw cycles.

SDS-PAGE:



Reconstitution:

Detailed reconstitution instructions are sent along with the products.

Protein Description

AHSP, also known as ERAF, is a conserved mammalian erythroid protein which belongs to the AHSP family. It is expressed in blood and bone marrow. AHSP facilitates the production of Hemoglobin A by stabilizing free α -globin. It rapidly binds to ferrous α with association ($k(\text{AHSP})$) and dissociation ($k(\text{AHSP})$) rate constants of $\approx 10 \mu\text{M}^{-1} \text{s}^{-1}$ and 0.2s^{-1} , respectively, at pH 7.4 at 22 °C. A small slow phase was observed when AHSP binds to excess ferrous αCO . This slow phase appears to be due to cis to trans prolyl isomerization of the Asp(29)-Pro(30) peptide bond in wild-type AHSP because it was absent when αCO was mixed with P30A and P30W AHSP, which are fixed in the trans conformation. This slow phase was also absent when met($\text{Fe}(3+)$)- α reacted with wild-type AHSP, suggesting that met- α is capable of rapidly binding to either Pro(30) conformer. Both wild-type and Pro(30)-substituted AHSPs drive the formation of a met- α hemichrome conformation following binding to either met- or oxy($\text{Fe}(2+)$)- α . The dissociation rate of the met- α -AHSP complex ($k(\text{AHSP}) \approx 0.002 \text{s}^{-1}$) is ~ 100 -fold slower than that for ferrous α -AHSP complexes, resulting in a much higher affinity of AHSP for met- α . Thus, in vivo, AHSP acts as a molecular chaperone by rapidly binding and stabilizing met- α hemichrome folding intermediates. The low rate of met- α dissociation also allows AHSP to have a quality control function by kinetically trapping ferric α and preventing its incorporation into less stable mixed valence Hemoglobin A tetramers. Reduction of AHSP-bound met- α allows more rapid release to β subunits to form stable fully, reduced hemoglobin dimers and tetramers.

References

1. Miele G. et al., 2001, Nat Med. 7 (3): 361-4.
2. Kihm AJ. et al., 2002, Nature. 417 (6890): 758-63.
3. Florio P. et al., 2004, Steroids. 68 (10-13): 801-7.
4. Miele G. et al., 2001, Nat Med. 7 (3): 361-4.
5. Mollan TL. et al., 2012, J Biol Chem. 287 (14): 11338-50.