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AHSG

Description: The human Alpha-2-HS-Glycoprotein was purified from the human plasma.

Catalog #:PRPS-425

Synonyms: Alpha-2-HS-glycoprotein, Fetuin-A, Alpha-2-Z-globulin, Ba-alpha-2-glycoprotein, AHSG, FETUA, AHS, A2HS, HSGA, PRO2743.

For research use only.

Source: Purified from the human plasma.

Physical Appearance: Filtered White lyophilized (freeze-dried) powder.

Purity: Greater than 95% as determined by SDS-PAGE.

Formulation:
Filtered (0.4

Stability:
Store the lyophilized protein at -20°C. Aliquot the product after reconstitution to avoid repeated freezing/thawing cycles. Reconstituted protein can be stored at 4°C for a limited period of time; it does not show any change after two weeks at 4°C.

Usage:
NeoBiolab's products are furnished for LABORATORY RESEARCH USE ONLY. The product may not be used as drµgs, agricultural or pesticidal products, food additives or household chemicals.

Solubility:
It is recommended to add deionized water to prepare a working stock solution of approximately 0.5mg/ml and let the lyophilized pellet dissolve completely. Product is not sterile! Please filter the product by an appropriate sterile filter before using it in the cell culture.

Introduction:
Fetuin is a liver-produced negative acute phase protein composed of two subunits, the A and B chains.Fetuin homologs have been identified in several species including rat, sheep, pig, rabbit, guinea pig, cattle, mouse and human. Multiple physiological roles for these homologs have been sµggested, including ability to bind to hydroxyapatite crystals and to specifically inhibit the tyrosine kinase (TK) activity of the insulin receptor (IR).Fetuin-A (alpha2-Heremans-Schmid glycoprotein; AHSG) is an important circulating inhibitor of calcification in vivo, and is downregulated during the acute-phase response.Sera from patients on long-term dialysis with low AHSG concentrations showed impaired ex-vivo capacity to inhibit CaxPO4 precipitation.Fetuin may influence the resolution of inflammation by modulating the phagocytosis of apoptotic cells by macrophages.ASHG blocks TGF-beta-dependent signaling in osteoblastic cells, and mice lacking ASHG display growth plate defects, increased bone formation with age, and enhanced cytokine-dependent osteogenesis.

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