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Product Datasheet

Recombinant Human AOC3 (C-Fc) EBT-EPT235

Article Name	Recombinant Human AOC3 (C-Fc)
Biozol Catalog Number	EBT-EPT235
Supplier Catalog Number	EPT235
Alternative Catalog Number	EBT-EPT235-10
Manufacturer	ELK Biotechnology
Category	Proteine/Peptide
Product Description	Recombinant Human Membrane Primary Amine Oxidase is produced by our Mammalian expression system and the target gene encoding Arg28-Asn763 is expressed with a Fc tag at the C-terminus....
Molecular Weight	Molecular weight: 108.5 KDa. Apparent molecular weight: 120 KDa, reducing conditions
UniProt	Q16853
Purity	Greater than 95% as determined by reducing SDS-PAGE.

Application Notes	Redissolve: Always centrifuge tubes before opening. Do not mix by vortex or pipetting. It is not recommended to reconstitute to a concentration less than 100µg/ml. Dissolve the lyophilized protein in distilled water. Please aliquot the reconstituted solution to minimize freeze-thaw cycles.. Endotoxin: Less than 0.1 ng/µg (1 EU/µg) as determined by LAL test. Background: Membrane primary amine oxidase(AOC3), also known as vascular adhesion protein (VAP-1) and HPAO, this protein is a member of the semicarbazide-sensitive amine oxidase (SSAO) family. VAP-1 is a type 1 membrane-bound glycoprotein that has a distal adhesion domain and an enzymatically active amine oxidase site outside of the membrane, VAP-1 has adhesive properties, functional monoamine oxidase activity, and possibly plays a role in glucose handling, leukocyte trafficking, and migration during inflammation. This rise in metabolic products contributes to generating advanced glycation end-products and oxidative stress along with the monoamine detoxification in the organism. It is highly expressed on the endothelium of the lung and trachea, and absent from leukocytes and epithelial cells. Membrane-bound VAP-1 releases an active, soluble form of the protein, which may be conducive to increased inflammation and the progression of many vascular disorders. In particular, elevation of VAP-1 activity and the increased enzymatic-mediated deamination is proposed to play a role in renal and vascular disease, oxidative stress, acute and chronic hyperglycemia, and diabetes complications
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