Recombinant Human Apolipoprotein E/ApoE-kyoto

Catalog Number: 11780-AE

DESCRIPTION

Source	Human embryonic kidney cell, HEK293-derived human Apolipoprotein E/ApoE protein Lys19-His318, Arg 43 Cys Accession # P02649.1
N-terminal Sequence Analysis	Lys19
Predicted Molecular Mass	34 kDa

SPECIFICATIONS

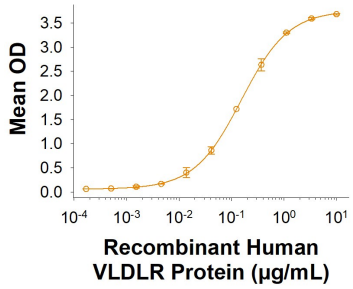
SDS-PAGE	34-40 kDa, under reducing conditions.
Activity	Measured by its binding ability in a functional ELISA. Recombinant Human Apolipoprotein E/ApoE-kyoto binds to Recombinant Human VLDLR Protein (Catalog # 8444-VL) with an ED_{50} of 0.0500-0.500 µg/mL.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>90%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
Formulation	Lyophilized from a 0.2 µm filtered solution in MOPS, NaCl and TCEP with Trehalose. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 100 µg/mL in water.
Shipping	The product is shipped at ambient temperature. 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.

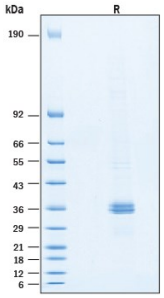
DATA

Binding Activity



Recombinant Human Apolipoprotein E/ApoE-kyoto Protein Binding Activity. Measured by its binding ability in a functional ELISA. Recombinant Human Apolipoprotein E/ApoE-kyoto Protein (Catalog # 11780-AE) binds to Recombinant Human VLDLR Protein (Catalog # 8444-VL) with an ED_{50} of 0.0500-0.500 µg/mL.

SDS-PAGE



Recombinant Human Apolipoprotein E/ApoE-kyoto Protein SDS-PAGE. 2 µg/lane of (Catalog # 11780-AE) was resolved with SDS-PAGE under reducing (R) condition and visualized by Coomassie® Blue staining, showing bands at 34-40 kDa.

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BACKGROUND

Apolipoprotein E (ApoE) is a polymorphic lipid-transport protein with three major human isoforms-ApoE2, ApoE3, and ApoE4-differing by single amino acid substitutions at residues 112 and 158, which significantly alter their structure and function [1, 2]. All isoforms share a two-domain structure: the N-terminal domain mediates receptor binding, while the C-terminal domain facilitates lipid binding and oligomerization [2]. ApoE3 (Cys112/Arg158) is the most common and functionally neutral isoform, while ApoE2 (Cys112/Cys158) has reduced affinity for LDL receptors, often leading to type III hyperlipoproteinemia in homozygotes [1, 4]. ApoE4 (Arg112/Arg158) exhibits a more compact and less stable structure due to domain interaction, predisposing it to pathological effects in the brain [2, 5]. Functionally, ApoE isoforms differentially regulate neuronal metabolism: ApoE2 enhances glycolytic activity and hexokinase expression, promoting neuroprotection, whereas ApoE4 impairs these pathways, increasing vulnerability to aging and Alzheimer's disease (AD) [1]. Recent transcriptomic and epigenomic profiling of human microglia in an AD xenotransplantation model revealed that ApoE isoforms distinctly shape gene expression and chromatin accessibility. ApoE4 drives pro-inflammatory and neurodegenerative signatures, while ApoE2 supports homeostatic microglial states [3]. Clinically, ApoE genotyping is widely used to assess AD risk, with ApoE4 being the strongest genetic risk factor and ApoE2 offering relative protection [4, 5]. Therapeutic strategies targeting ApoE include isoform-specific modulation and gene editing. Additionally, ApoE isoforms are valuable tools in translational research for drug screening, biomarker discovery, and personalized medicine [4]. ApoE2-Kyoto (Cys25/Cys112/Arg158), a rare variant of ApoE2, has been linked to lipoprotein glomerulopathy and demonstrates altered receptor-binding and lipid-handling properties [6].

References:

1. Zhang, X. *et al.* (2023) Cells **12**:410.
2. Horn, J.V.C. *et al.* (2023) Mol Cell Biochem **478**:173.
3. Murphy, K.B. *et al.* (2025) Nat Commun **16**:4883.
4. Mahley, R.W. *et al.* (2012) J Lipid Res **53**:539.
5. Liu, C.C. *et al.* (2013) Nat Rev Neurol **9**:106.
6. Matsunaga, A. *et al.* (1999) Kidney International **56**:421.

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