# Human Apolipoprotein E/ApoE Antibody

Monoclonal Mouse $IgG_1$ Clone # 960318

Catalog Number: MAB41443

## DESCRIPTION

| | |
|---|---|
| **Species Reactivity** | Human |
| **Specificity** | Detects human Apolipoprotein E/ApoE in direct ELISAs. |
| **Source** | Monoclonal Mouse $IgG_1$ Clone # 960318 |
| **Purification** | Protein A or G purified from hybridoma culture supernatant |
| **Immunogen** | Human Apolipoprotein E/ApoE sythetic peptide<br>Accession # P02649 |
| **Formulation** | Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details.<br>*Small pack size (-SP) is supplied either lyophilized or as a 0.2 µm filtered solution in PBS. |

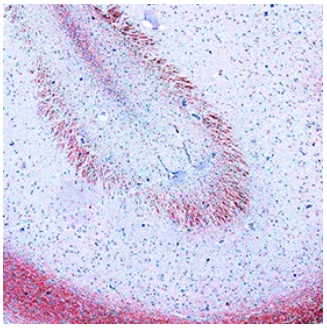
## APPLICATIONS

**Please Note:** *Optimal dilutions should be determined by each laboratory for each application. General Protocols are available in the Technical Information section on our website.*

| | Recommended Concentration | Sample |
|---|---|---|
| **Immunohistochemistry** | 5-25 µg/mL | See Below |

## DATA

**Immunohistochemistry**



**Detection of Apolipoprotein E/ApoE in Human Brain.** Apolipoprotein E/ApoE was detected in immersion fixed paraffin-embedded sections of human brain (hippocampus) using Mouse Anti-Human Apolipoprotein E/ApoE Monoclonal Antibody (Catalog # MAB41443) at 5 µg/mL for 1 hour at room temperature followed by incubation with the Anti-Mouse IgG VisUCyte™ HRP Polymer Antibody (Catalog # VC001). Tissue was stained using DAB (brown) and counterstained with hematoxylin (blue). Specific staining was localized to cytoplasm. View our protocol for IHC Staining with VisUCyte HRP Polymer Detection Reagents.

## PREPARATION AND STORAGE

| | |
|---|---|
| **Reconstitution** | Reconstitute at 0.5 mg/mL in sterile PBS. For liquid material, refer to CoA for concentration. |
| **Shipping** | Lyophilized product is shipped at ambient temperature. Liquid small pack size (-SP) is shipped with polar packs. Upon receipt, store immediately at the temperature recommended below. |
| **Stability & Storage** | **Use a manual defrost freezer and avoid repeated freeze-thaw cycles.**<br>• 12 months from date of receipt, -20 to -70 °C as supplied.<br>• 1 month, 2 to 8 °C under sterile conditions after reconstitution.<br>• 6 months, -20 to -70 °C under sterile conditions after reconstitution. |



Bio-Techne®
Global | bio-techne.com info@bio-techne.com techsupport@bio-techne.com TEL: 1.612.379.2956
USA | TEL: 800.343.7475 Canada | TEL: 855.668.8722 Europe | Middle East | Africa TEL: +44.0.1235.529449
China | info.cn@bio-techne.com TEL: 400.821.3475

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## BACKGROUND

ApoE is a major protein component of serum LDL, VLDL, HDL, and chylomicrons. It is produced predominantly by hepatocytes, macrophages, and non-neuronal cells in the CNS. ApoE-containing particles transport triglycerides and cholesterol to peripheral tissues for cellular uptake and catabolism (1-4). Mature human ApoE is a 37 kDa glycoprotein that consists of an N-terminal domain composed of four bundled α-helices, plus a hinge region and an extended α-helical C-terminal domain (2, 5). Its amphipathic nature and flexible structure enables it to adopt dramatically different conformations upon lipid association (2). ApoE is monomeric in lipid particles, although it forms oligomers when lipid-free (6). ApoE3 is the most abundant of the three common alleles in human; ApoE2 and ApoE4 differ by single aa substitutions (1). Mature human ApoE shares 71% aa sequence identity with mouse and rat ApoE. LDL receptor family proteins preferentially bind and internalize the lipid-bound form of ApoE with the exception of VLDLR which also efficiently internalizes lipid-free ApoE (7, 8). Lipoprotein uptake is facilitated by the initial binding of ApoE to cell surface heparan sulfate proteoglycans (HSPG) (9). Receptor/HSPG binding and lipid interactions primarily involve the N- and C-terminal regions of ApoE, respectively (2). Recycled lipid-free ApoE is formed into HDL particles through interactions with the lipid transporter ABCA1 (10). High cellular sterol content activates the nuclear hormone receptor LXR which promotes increased ApoE synthesis and increased sterol efflux, while low sterol content induces LDL R expression with increased sterol uptake and decreased ApoE production (11). ApoE3 dampens the TNF-α induced inflammatory response in vascular endothelial cells (12). In the CNS, ApoE blocks production of the amyloid Aβ peptide by inhibiting the γ-secretase cleavage of APP (13). It also complexes with Aβ and promotes Aβ internalization *via* LRP2 (14, 15).

**References:**

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