in familial early-onset AD, or decreased Aβ clearance in brain. The lack of compelling evidence that Aβ production occurs in the more common late-onset forms of AD suggests that insufficient Aβ clearance may drive Aβ deposition and amyloid plaque formation as well.

[0006] The apolipoprotein E (ApoE) gene remains the most widely replicated genetic risk factor for late-onset AD, with carriers of the E4 allelle having a 3-15-fold greater risk as well as an earlier age of disease onset. In brain, ApoE is mainly synthesized and secreted by astrocytes and microglia which are found to surround amyloid plaques. The present invention provides ApoE antibodies effective at reducing amyloid plaque load in AD mouse models.

SUMMARY ~~OF THE INVENTION~~

[0006a] In one aspect, there is provided the use of at least one anti-ApoE antibody in the manufacture of a medicament for reversing at least one symptom or sign of Aβ plaque associated symptoms in a subject having Alzheimer's disease, Lewy body dementia, or cerebral amyloid angiopathy.

[0006b] In one aspect, there is provided the use of at least one anti-ApoE antibody in the manufacture of a medicament for decreasing the amyloid plaque load in the brain of a subject having Alzheimer's disease, Lewy body dementia, or cerebral amyloid angiopathy.

[0007] Disclosed is~~One aspect of the invention encompasses~~ a method of effectively treating at least one clinically detectable Aβ plaque associated symptom which comprises administering an effective amount of an anti-ApoE antibody to a living human subject. Also disclosed is~~In another aspect, the invention encompasses~~ an antibody useful in such treatment. For instance, an antibody that therapeutically attenuates the toxic effects of the Aβ peptide in a living mammal.

[0008] Disclosed isAnother aspect of the invention encompasses a composition comprising at least one anti-ApoE antibody. In one embodimentan aspect, the disclosureinvention encompasses a composition comprising at least one anti-ApoE antibody produced from hybridoma HJ6.1, HJ6.2, HJ6.3, HJ6.4 or a combination thereof. In one embodimentan aspect, the disclosureinvention encompasses a medicinal composition comprising at least one anti-ApoE antibody. In one embodimentan aspect, the disclosureinvention encompasses a medicinal composition comprising at least one anti-ApoE antibody produced from hybridoma HJ6.1, HJ6.2, HJ6.3, HJ6.4 or a combination thereof.

[0009] Yet another embodimentaspect of the disclosureinvention encompasses a medicinal composition useful to treat at least one clinically detectable Aβ plaque associated symptom. The composition comprises a medicinally effective amount of an anti-ApoE antibody adapted for administration to a living human subject. In one embodimentan aspect, an antibody useful in such

treatment includes an antibody that therapeutically attenuates the toxic effects of the Aβ peptide in a living mammal. In one embodiment~~an aspect~~, the medicinal composition is effectively administered to a living subject.

[0010] Still another embodiment~~aspect~~ the disclosure~~invention~~ encompasses a medicinal kit comprising a container containing a functional therapeutic medicinal composition of a medicinally effective amount of an anti-ApoE antibody adapted for administration to a living human subject and any medical devices to be used for said administration. In one embodiment~~an aspect~~, an antibody useful in such treatment includes an antibody that therapeutically attenuates the toxic effects of the Aβ peptide in a living mammal.

[0011] Other aspects and iterations of the disclosure~~invention~~ are detailed below.

BRIEF DESCRIPTION OF THE FIGURES

[0012] **FIG. 1** graphically depicts a strong effect of anti-ApoE antibodies in decreased amyloid plaque load in samples of hippocampus from PS/APP mice treated intraperitoneally once weekly with 10 mg/kg beginning at 3 months of age until 7 months of age with either an anti-Aβ antibody (HJ3.4) or one of two anti-ApoE antibodies (HJ6.2 or HJ6.3). The control group was treated with phosphate buffered saline (PBS). Animal numbers assessed are N=7 (PBS), N=16 (HJ3.4), N=16 (HJ6.2), N=16).

[0013] **FIG. 2** graphically depicts a strong effect of anti-ApoE antibodies in decreased amyloid plaque load in samples of brain cortex from PS/APP mice treated intraperitoneally once weekly with 10 mg/kg with either an anti-Aβ antibody (HJ3.4) or one of two anti-ApoE antibodies (HJ6.2 or HJ6.3). The control group was treated with PBS. Animal numbers assessed are N=7 (PBS), N=16 (HJ3.4), N=16 (HJ6.2), N=16).

[0014] **FIG. 3** graphically depicts binding of anti-apoE antibodies HJ6.1, HJ6.2, HJ6.3 and HJ6.4 to human apoE using an ELISA assay.

[0015] **FIG. 4** graphically depicts (**A**) binding of anti-apoE antibodies HJ6.1, HJ6.2, HJ6.3 and HJ6.4 to human apoE in plasma, and (**B**) levels of cholesterol in the same mice that received the HJ6.1, HJ6.2, HJ6.3 and HJ6.4 antibodies.

[0016] **FIG. 5** graphically depicts the presence of anti-apoE antibodies HJ6.1, HJ6.2, HJ6.3 and HJ6.4 in the central nervous system after peripheral administration.