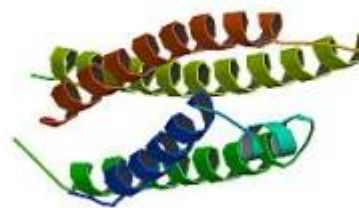


Apolipoprotein E4



Apolipoprotein E (ApoE) is a **glycoprotein** with a major role in **brain lipoprotein metabolism**. It has three isoforms encoded by distinct **alleles**: APOEε2, APOEε3 and **APOEε4**.

Risk factors for **Dementia**: advanced **age**, **family history** of dementia, and apolipoprotein E-4 **allele**.

The presence of this **genotype** portends a worse prognosis following **traumatic brain injury** ¹⁾

Furthermore, the incidence of **severe traumatic brain injury** in individuals with the apoE-4 allele greatly exceeds the rate of the allele in the general population ²⁾. This allele is also a risk factor for **Alzheimer's disease** ^{3) 4) 5)} as well as for **chronic traumatic encephalopathy**.

Among patients with lobar hemorrhage, those with the apoE ε4 allele typically have their first hemorrhage >5 yrs earlier than noncarriers (73 ± 8 yrs vs. 79 ± 7 yrs) ⁶⁾.

Findings suggested that APOEε4 allele is a risk factor to brain function aggravation in the early stage of **aneurysmal subarachnoid hemorrhage**, and it may contribute to early brain injury after **SAH** ⁷⁾.

Finding also suggests that the patients with APOEε4 allele predispose to **cerebral vasospasm** after spontaneous SAH ⁸⁾.

The presence of APOE ε4, an elevated **international normalized ratio**, and a higher **glucose** level (≥ 10 mmol/L) are predictors of progressive **traumatic intracerebral hemorrhage**. Additionally, APOE ε4 is not associated with traumatic coagulopathy and patient outcome ⁹⁾.

APOE ε4 and ε2 alleles appear to affect lobar **ICH** risk variably by race/ethnicity, associations that are confirmed in white individuals but can be shown in Hispanic individuals only when the excess burden of hypertension is propensity score-matched; further studies are needed to explore the interactions between APOE alleles and environmental exposures that vary by race/ethnicity in representative populations at risk for ICH ¹⁰⁾.

APOEε4 may induce **cerebral perfusion** impairment in the early phase, contributing to early brain

injury (EBI) following [aneurysmal subarachnoid hemorrhage](#) (aSAH), and assessment of APOE genotypes could serve as a useful tool in the prognostic evaluation and therapeutic management of aSAH ¹¹⁾.

The APOE ϵ 4 polymorphism was analysed in 147 patients with aSAH. Allele and genotype frequencies were compared to those found in a gender- and area-matched control group of healthy individuals (n = 211). Early [cerebral vasospasm](#) (CVS) was identified and treated according to neurointensive care unit (NICU) guidelines. [Neurological deficit\(s\)](#) at admittance and at 1-year follow-up visit was recorded. Neurological outcome was assessed by the National Institute of Health Stroke Scale, Barthel Index and the Extended Glasgow Outcome Scale.

APOE ϵ 4 and non-APOE ϵ 4 allele frequencies were similar in aSAH patients and healthy individuals. The presence of APOE ϵ 4 was not associated with the development of early CVS. We could not find an influence of the APOE polymorphism on 1-year neurological outcome between groups. Subgroup analyses of patients treated with surgical clipping vs endovascular coiling did not reveal any associations.

The APOE ϵ 4 polymorphism has no major influence on risk of aSAH, the occurrence of CVS or long-term neurological outcome after aSAH ¹²⁾.

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