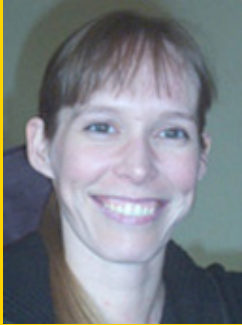


The Memory Practice Newsletter

Issue 27, April 2009



In this issue Dr. Michelon tells you about Alzheimer's Disease and genetic risk factors.

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Alzheimer's Disease and Genetics: The latest news on APOE-ε4

Alzheimer's Disease (AD) is a progressive brain disease. It has two types: early-onset and late-onset. Both types have genetic links.

Early-onset AD is a rare form of AD that develops in people 30 to 65 years of age. Of all people having AD only 5% suffer from early onset.

Some cases of early-onset, called familial AD, are inherited. These cases are caused by gene mutations on chromosomes 21, 14, and 1. These mutations cause abnormal proteins to be formed in the brain.

If one parent has one of these gene mutations (and thus AD), offspring in the same generation have a 50/50 chance of developing AD.

Late-onset AD is the most common form of AD that develops after age 60. Mutations seen in early-onset cases are not involved in late-onset cases.

No gene has been found so far to be the cause of late-onset AD.

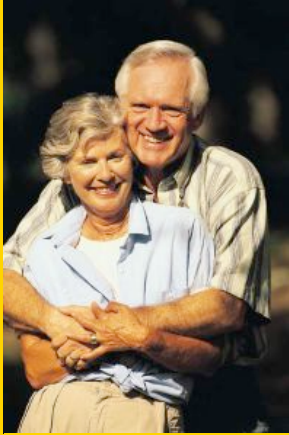
However a predisposing genetic factor does appear to increase a person's risk of developing AD. This increased risk is related to the apolipoprotein E (APOE) gene found on chromosome 19.

APOE comes in several different forms: 3 forms (or alleles) are the most common: APOE ε2, APOE ε3, and APOE ε4.

APOE ε4 occurs in about 40 percent of all people who have late-onset AD.

It is present in about 25 to 30 percent of the population. People with AD are more likely to have an APOE ε4 form than people who do not develop AD.

However, many people with AD do not have an APOE ε4 allele...



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Previous studies have shown that the brain of middle-aged and elderly individuals who have APOE ϵ 4 does not seem to work the same way compared to the brain of individuals who do not carry APOE ϵ 4, especially during memory tasks.

New study with young APOE ϵ 4 carriers

In a recent study the brains of 18 young adults who carried APOE ϵ 4 were compared to the brains of 18 young adults who did not carry APOE ϵ 4 (Filippini, et al., 2009). The mean age of these 36 adults was 28.6 years old. The brains of the two groups did not differ in terms of volume, white matter and cerebrospinal fluid.

The brains of the participants were scanned (using functional neuroimaging) both at rest and while trying to recognize previously memorized pictures.

The two groups did just as well in terms of memory performance.

However, the activity in their brain was different! Especially in regions such as the hippocampus that are critical for memory formation.

Young APOE ϵ 4 carriers had more activity in their hippocampus both while at rest and while performing the memory task.

The authors of the study suggest that this increased activity may reflect processes to compensate for reduced plasticity, reduced neuronal growth or low-efficient memory consolidation processes.

Why does this study matters?

This result is exciting because it shows changes in the brain long before any cognitive decline is apparent.

It suggests that carrying APOE ϵ 4 makes the memory system more vulnerable.

It may also open the door to new early diagnostic tools.

References

Filippini, N., MacIntosh, B., Hough, M., ... & Mackay, C. (2009). Distinct patterns of brain activity in young carriers of the APOE- ϵ 4 allele, *PNAS*, published on-line before print.