Alzheimer disease

BACKGROUND SCIENCE

Alzheimer disease (AD) is a devastating neurodegenerative disease and the predominant form of dementia (50-75%). In 2015, around 44 million people worldwide are estimated to have AD or a related dementia.

The disease is clinically characterized by progressive deterioration of memory and cognitive functions, leading to loss of autonomy and ultimately requiring full-time medical care. AD is pathologically defined by severe neuronal loss, aggregation of amyloid β (Aβ) in extracellular senile plaques, and formation of intraneuronal neurofibrillary tangles consisting of hyperphosphorylated tau protein.

Although the vast majority of patients develop clinical symptoms at age older than 65 years (late-onset AD), 2-10% of patients have an earlier onset of disease (early-onset AD). Rare autosomal dominant forms of AD exist, predominantly presenting as early-onset AD, although the majority of early-onset AD patients do not present with a clear autosomal pattern of inheritance. In an autosomal dominant disease, if you inherit the abnormal gene from only one parent, you can get the disease.

Typical signs and symptoms:

- MEMORY LOSS THAT DISRUPTS DAILY LIFE
- CHALLENGES IN PLANNING OR SOLVING PROBLEMS
- DIFFICULTY COMPLETING FAMILIAR TASKS AT HOME, AT WORK OR AT LEISURE
- CONFUSION WITH TIME OR PLACE
- TROUBLE UNDERSTANDING VISUAL IMAGES AND SPATIAL RELATIONSHIPS
- NEW PROBLEMS WITH WORDS IN SPEAKING OR WRITING
- MISPLACING THINGS AND LOSING THE ABILITY TO RETRACE STEPS

What do we test?

76 genetic variants linked to familial Alzheimer disease and 7 well-studied genetic variants representing the odds to get the disease.

When symptoms develop:

Late-onset AD: >60.
Early-onset AD: 30-50

How it's medically treated:

There is no cure, only treatment for symptoms. Treatment for Alzheimer's symptoms includes donepezil, memantine, and other medicines. Clinical trials are underway to test new treatments. However, in a 2016 study[1], reversal of cognitive decline in Alzheimer's disease was a significant success with diet, brain stimulation, exercise, sleep, vitamins & more.

GENETICS & ALZHEIMER

- A recent meta-study[2] reviewed all the genetic factors causing AD. The search for the genetic factors contributing to Alzheimer disease (AD) has evolved tremendously throughout the years. It started from the discovery of fully penetrant mutations in Amyloid precursor protein, Presenilin 1, and Presenilin 2 as a cause of autosomal dominant AD, the identification of the ε4 allele of Apolipoprotein E as a strong genetic risk factor for both early-onset and late-onset AD, and evolved to the more recent detection of at least 21 additional genetic risk loci for the genetically complex form of AD emerging from genome-wide association studies and massive parallel resequencing efforts. These advances in AD genetics are positioned in light of the current endeavor directing toward translational research and personalized treatment of AD.
- Many DNA testing companies test only the famous APOE ε4 gene to predict AD risks. But APOE ε4 is neither necessary nor sufficient to cause the disease. Up to 75% of individuals heterogeneous for APOE ε4 do not develop AD during life, and up to 50% of people with AD do not carry the high-risk ε4 allele.[3]

YOUR RESULTS

Gene	Your results	Effects
APP (rs63750064)	CC	No familial Alzheimer's disease detected.
APP (rs63750671)	GG	No familial Alzheimer's disease detected.
APP (rs63751039)	TT	No familial Alzheimer's disease detected.
APP (rs63749810)	CC	No familial Alzheimer's disease detected.
APP (rs63750921)	GG	No familial Alzheimer's disease detected.
APP (rs63750066)	CC	No familial Alzheimer's disease detected.
APP (rs63750643)	TT	No familial Alzheimer's disease detected.
APP (rs63750973)	GG	No familial Alzheimer's disease detected.

APP (rs63750734)	CC	No familial Alzheimer's disease detected.
APP (rs63750399)	TT	No familial Alzheimer's disease detected.
APP (rs63750851)	AA	No familial Alzheimer's disease detected.
APP (rs63749964)	AA	No familial Alzheimer's disease detected.
APP (rs63750847)	CC	No familial Alzheimer's disease detected.
PSEN1 (rs63749824)	CC	No familial Alzheimer's disease detected.
PSEN1 (rs63750599)	TT	No familial Alzheimer's disease detected.
PSEN1 (rs63750815)	GG	No familial Alzheimer's disease detected.
PSEN1 (rs63751141)	GG	No familial Alzheimer's disease detected.
PSEN1 (rs63750831)	GG	No familial Alzheimer's disease detected.
PSEN1 (rs63750601)	GG	No familial Alzheimer's disease detected.
PSEN1 (rs63750852)	GG	No familial Alzheimer's disease detected.
PSEN1 (rs63751475)	II	No familial Alzheimer's disease detected.
PSEN1 (rs63750450)	AA	No familial Alzheimer's disease detected.
PSEN1 (rs63750550)	CC	No familial Alzheimer's disease detected.
PSEN1 (rs63750800)	GG	No familial Alzheimer's disease detected.
PSEN1 (rs63750353)	AA	No familial Alzheimer's disease detected.
PSEN1 (rs63751278)	AA	No familial Alzheimer's disease detected.
PSEN1 (rs41345849)	CC	No familial Alzheimer's disease detected.
PSEN1 (rs63751037)	AA	No familial Alzheimer's disease detected.
PSEN1 (rs63750522)	GG	No familial Alzheimer's disease detected.
PSEN1 (rs63750322)	AA	No familial Alzheimer's disease detected.
PSEN1 (rs63751071)	TT	No familial Alzheimer's disease detected.
PSEN1 (rs63750907)	CC	No familial Alzheimer's disease detected.
PSEN1 (rs63751441)	CC	No familial Alzheimer's disease detected.
PSEN1 (rs63750588)	TT	No familial Alzheimer's disease detected.
PSEN1 (rs63750590)	AA	No familial Alzheimer's disease detected.
PSEN1 (rs63751010)	TT	No familial Alzheimer's disease detected.
PSEN1 (rs63751484)	GG	No familial Alzheimer's disease detected.
PSEN1 (rs63751458)	II	No familial Alzheimer's disease detected.
PSEN1 (rs63750418)	TT	No familial Alzheimer's disease detected.
PSEN1 (rs63751210)	CC	No familial Alzheimer's disease detected.
PSEN1 (rs63750577)	CC	No familial Alzheimer's disease detected.
PSEN1 (rs63750963)	TT	No familial Alzheimer's disease detected.
PSEN1 (rs63750299)	TT	No familial Alzheimer's disease detected.
PSEN1 (rs63751144)	CC	No familial Alzheimer's disease detected.

PSEN1 (rs63751025)	TT	No familial Alzheimer's disease detected.
PSEN1 (rs63749911)	TT	No familial Alzheimer's disease detected.
PSEN1 (rs63750155)	TT	No familial Alzheimer's disease detected.
PSEN1 (rs63751068)	GG	No familial Alzheimer's disease detected.
PSEN1 (rs63750311)	AA	No familial Alzheimer's disease detected.
PSEN1 (rs63750569)	GG	No familial Alzheimer's disease detected.
PSEN1 (rs63751309)	TT	No familial Alzheimer's disease detected.
PSEN1 (rs63751003)	CC	No familial Alzheimer's disease detected.
PSEN1 (rs63749987)	CC	No familial Alzheimer's disease detected.
PSEN1 (rs63750009)	AA	No familial Alzheimer's disease detected.
PSEN1 (rs63751072)	GG	No familial Alzheimer's disease detected.
PSEN1 (rs63750487)	CC	No familial Alzheimer's disease detected.
PSEN1 (rs63749961)	TT	No familial Alzheimer's disease detected.
PSEN1 (rs63749970)	AA	No familial Alzheimer's disease detected.
PSEN1 (rs63749836)	GG	No familial Alzheimer's disease detected.
PSEN1 (rs63750799)	CC	No familial Alzheimer's disease detected.
PSEN1 (rs63751024)	TT	No familial Alzheimer's disease detected.
PSEN1 (rs63751130)	CC	No familial Alzheimer's disease detected.
PSEN1 (rs63749835)	TT	No familial Alzheimer's disease detected.
PSEN1 (rs63750526)	CC	No familial Alzheimer's disease detected.
PSEN1 (rs63751163)	TT	No familial Alzheimer's disease detected.
PSEN1 (rs63751420)	CC	No familial Alzheimer's disease detected.
PSEN1 (rs63750301)	CC	No familial Alzheimer's disease detected.
PSEN1 (rs63751229)	CC	No familial Alzheimer's disease detected.
PSEN1 (rs63751019)	CC	No familial Alzheimer's disease detected.
PSEN1 (rs63750900)	GG	No familial Alzheimer's disease detected.
PSEN1 (rs63750680)	TT	No familial Alzheimer's disease detected.
PSEN1 (rs63751139)	CC	No familial Alzheimer's disease detected.
PSEN1 (rs63751235)	CC	No familial Alzheimer's disease detected.
PSEN2 (rs63750048)	CC	No familial Alzheimer's disease detected.
PSEN2 (rs63749851)	AA	No familial Alzheimer's disease detected.
PSEN2 (rs63750812)	GG	No familial Alzheimer's disease detected.
PSEN2 (rs63750880)	AA	No familial Alzheimer's disease detected.
PSEN2 (rs28936379)	AA	No familial Alzheimer disease detected.
PSEN2 (rs63749884)	GG	No familial Alzheimer disease detected.

APOE	E3/E3	Decreased Alzheimer disease risk.
TREM2	CC	Typical risk for AD.
SOLR1	GG	Typical risk for AD.
BIN1	GG	1.5 fold higher risk for AD.
CR1	AG	1.2 fold higher risk for AD.
CLU	CG	1.2 fold higher risk for AD.

YOUR OVERALL ALZHEIMER RISK SCORE :

Slightly higher risk

You have a slightly higher AD risk than average.

WHAT CAN YOU DO TO PREVENT THIS CONDITION ?

If your GenetiConcept results indicate a higher odds for this condition, here are a few natural strategies you can follow. Note that this is not a diagnostic test. In a 2016 study[1], reversal of cognitive decline in Alzheimer's disease was a significant success with diet, brain stimulation, exercise, sleep, vitamins & more.

- Alpha-GPC supplement has been shown in various studies to improve brain in people with Alzheimer's within three to six months. Those types of positive results are getting people in Europe excited about the possibilities that this supplement offers.
  There is a chemical within the brain known as acetycholine that the brain uses to transmit information between neurons and receptors. The Choline family of nootropics is a very popular supplement that is designed to help memory and improve cognitive thinking and is a component of acetycholine. When you take Alpha GPC, all of the compounds are separated and synthesized by your body. The acetycholine from the Alpha GPC is stored by the brain and used when needed to enhance memory.

  As a person ages, the amount of choline in the brain starts to naturally deteriorate. This is what begins to cause memory loss as we get older. This process is accelerated due to conditions such as Alzheimer's and dementia, which can both be combated by an increase in acetycholine. Alpha GPC helps to not only maintain the proper level of acetycholine the brain needs to function, but offers the brain the ability to store acetycholine and utilize it as needed.
- A high circulating concentration of the amino acid homocysteine is an independent risk factor for stroke. Alzheimer's disease (AD) commonly co-occurs with stroke. Epidemiological studies[4] found significant associations between high levels of homocysteine and both histologically confirmed AD and disease progression. Interestingly, homocysteine level is dependant of your folate metabolism efficiency and therefore it can be easily controlled through diet and supplementation. You can find more information about this topic in your nutritional section (Nutrition > Folate Metabolism).
- A recent University of Wisconsin School of Medicine and Public Health study[5] showed that life experiences that engage the brain, such as higher educational attainment in this case, protect against biological changes in the brain that underlie Alzheimer's. So stay mentally active and exercise your mind !
- Growing evidence suggests a link between heart health and brain health. Taking care of your heart by controlling your blood pressure, weight, cholesterol, and diabetes may also help decrease your risk for Alzheimer's. A healthy, low-fat diet can not only help you maintain healthy weight and cholesterol, but can also help reduce your risk of Alzheimer's.

SCIENTIFIC REFERENCES

[1] Dale E. Bredesen, Edwin C. Amos, Jonathan Canick, Mary Ackerley, Cyrus Raji, Milan Fiala, and Jamila Ahdidan; 05/30/16; "Reversal of cognitive decline in Alzheimer's disease"

[2] Caroline Van Cauwenberghe, Christine Van Broeckhoven, Kristel Sleegers, Genetics in Medicine (2016), "The genetic landscape of Alzheimer disease: clinical implications and perspectives"

[3] Farrer LA, Cupples LA, Haines JL, et al. Effects of age, sex, and ethnicity on the association between apolipoprotein E genotype and Alzheimer disease. A meta-analysis. APOE and Alzheimer Disease Meta Analysis Consortium. JAMA 1997;278:1349-1356.

[4] Lancet Neurol. 2003 Jul;2(7):425-8. "Homocysteine and Alzheimer's disease"

[5] JAMA Neurol. 2015 Jun;72(6):699-706. doi: 10.1001/jamaneurol.2015.0098. "Effect of Cognitive Reserve on Age-Related Changes in Cerebrospinal Fluid Biomarkers of Alzheimer Disease."

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