PARKINSON'S DEMENTIA ALZHEIMER'S

Genetic Predisposition Testing

REPORT

Ayass bioscience LLC™
Bridging Science and Precision Patient Care

Patient Name: XXXXX XXXXXXX
Date of Birth: XX/XX/XXXX
Accession #: XXXXXXXXXXXX

Parkinson's, Dementia, Alzheimer's - Genetic Predisposition Testing Report

Patient Name:

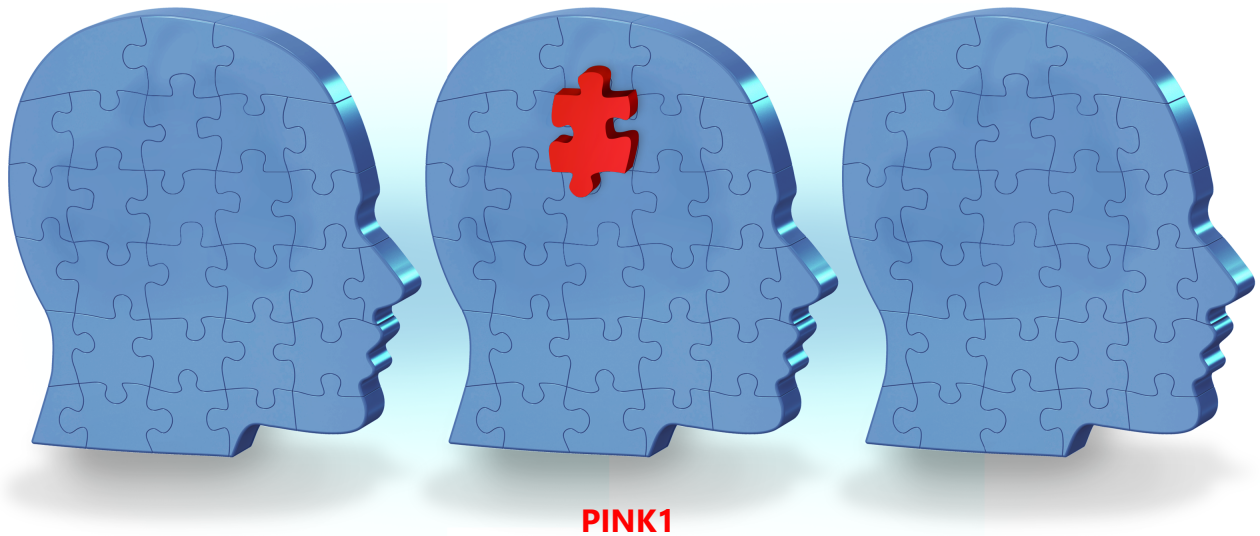
Accession #: XXXXXXXXXXX

DOB:

Collection Date:

Your Genetic Risk of Neurological Disease

Dementia **Parkinson's** **Alzheimer's**



PINK1

LEGEND:

Red: One or more pathogenic or likely pathogenic variants were detected on the indicated genes for the related condition(s)

Orange: One or more variants of unknown significance (VUS) were detected on the indicated genes for the related condition(s)

Green: No significant variants were detected on genes related to the indicated conditions.

Parkinson's, Dementia, Alzheimer's - Genetic Predisposition Testing Report

Patient Name:

Accession #: XXXXXXXXXXXXXX

DOB:

Collection Date:

RECEIVING HEALTHCARE PROVIDER

SPECIMEN

Specimen Type: **Buccal**
Collection Date:
Accession Date:
Received Date:
Report Date: **15 Nov 2019**

PATIENT

Name:
Date of Birth:
Gender:
Accession #: **XXXXXXXXXXXX**
File: **XXXXXXXXXXXX**

ORDERING PHYSICIAN:

CLINICAL BACKGROUND / INDICATIONS:

RESULT: POSITIVE - CLINICALLY SIGNIFICANT VARIANT IDENTIFIED

Note: "CLINICALLY SIGNIFICANT", as defined in the report, is a genetic change that has been directly reported to contribute to the development of disease and is associated with the potential to alter medical intervention.

CLINICALLY SIGNIFICANT VARIANTS

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CLASSIFICATION	GENE	RSID	MUTATION	INTERPRETATION
Pathogenic	**PINK1**	**N/A**	**c.781G>C (p.Val261Leu)** HET	**HIGH RISK FOR PARKINSON'S DISEASE**

DETAILS ABOUT **c.781G>C (p.Val261Leu): NM_000021.3**

Functional Significance:

The heterozygote germline PINK1 variant c.781G>C is predicted to result in abnormal protein translation of the PINK1 protein at amino acid position 261 (p.Val261Leu) .
Predicted effect(s) on the protein: Missense

Clinical Significance: High RISK FOR Parkinson's

This mutation is associated with increased Parkinson's risk and should be regarded as clinically significant.

Evidence

Disease Summary: Frontotemporal dementia with parkinsonism-17 (FTDP-17) is a brain disorder. It is part of a group of conditions, called frontotemporal dementia or frontotemporal degeneration, that are characterized by a loss of nerve cells (neurons) in areas of the brain called the frontal and temporal lobes. Over time, a loss of these cells can affect personality, behavior, language, and movement.

The signs and symptoms of FTDP-17 usually become noticeable in a person's forties or fifties. Most affected people survive 5 to 10 years after

the appearance of symptoms, although a few have survived for two decades or more.

Changes in personality and behavior are often early signs of FTDP-17. These changes include a loss of inhibition, inappropriate emotional responses, restlessness, neglect of personal hygiene, and a general loss of interest in activities and events. The disease also leads to deterioration of cognitive functions (dementia), including problems with judgment, planning, and concentration. Some people with FTDP-17 develop psychiatric symptoms, including obsessive-compulsive behaviors, strongly held false beliefs (delusions), and false perceptions (hallucinations). It may become difficult for affected individuals to interact with others in a socially appropriate manner. They increasingly require help with personal care and other activities of daily living.

Many people with FTDP-17 develop problems with speech and language. They may have trouble finding words, confuse one word with another (semantic paraphasias), and repeat words spoken by others (echolalia). Difficulties with speech and language worsen over time, and most affected individuals eventually lose the ability to communicate.

FTDP-17 is also characterized by problems with movement that worsen over time. Many affected individuals develop features of parkinsonism, including tremors, rigidity, and unusually slow movement (bradykinesia). As the disease progresses, most affected individuals become unable to walk. Some people with FTDP-17 also have restricted up-and-down eye movement (vertical gaze palsy) and rapid abnormal movements of both eyes (saccades).

Population Frequency: The worldwide prevalence of FTDP-17 is unknown. In the Netherlands, where the disease prevalence has been studied, it is estimated to affect 1 in 1 million people. However, the disorder is likely underdiagnosed, so it may actually be more common than this estimate.

FTDP-17 probably accounts for a small percentage of all cases of frontotemporal dementia.

References (PubMed ID#):

- APOE epsilon-2-epsilon-4 genotype is a possible risk factor for primary progressive aphasia. (PMID: 16437577)
- Localization of frontotemporal dementia with parkinsonism in an Australian kindred to chromosome 17q21-22. (PMID: 9392579)
- Absence of rapid eye movement sleep behavior disorder in 11 members of the pallidopontonigral degeneration kindred. (PMID: 16476816)
- Association between tau H2 haplotype and age at onset in frontotemporal dementia. (PMID: 16157749)
- Reelin expression and glycosylation patterns are altered in Alzheimer's disease. (PMID: 16567613)
- Familial dementia with swollen achromatic neurons and corticobasal inclusion bodies: a clinical and pathological study. (PMID: 8926492)
- Clinical and neuropathological criteria for frontotemporal dementia. (PMID:)
- Neuropathologic diagnostic and nosologic criteria for frontotemporal lobar degeneration: consensus of the consortium for frontotemporal lobar degeneration. (PMID: 17579875)
- Pathogenic implications of mutations in the tau gene in pallido-ponto-nigral degeneration and related neurodegenerative disorders linked to chromosome 17. (PMID: 9789048)
- A family with autosomal dominant non-Alzheimer's presenile dementia. (PMID: 9088499)
- A mutation at codon 279 (N279K) in exon 10 of the tau gene causes a tauopathy with dementia and supranuclear palsy. (PMID: 10412802)
- Familial early-onset dementia with tau intron 10 +16 mutation with clinical features similar to those of Alzheimer disease. (PMID: 17923640)
- Frontotemporal dementia: clinicopathological correlations. (PMID: 16718704)
- Frontotemporal dementia and parkinsonism linked to chromosome 17: a consensus conference. (PMID: 9189031)
- Distinctive MRI findings in pallidopontonigral degeneration (PPND). (PMID: 17310038)
- Prion in progressive subcortical gliosis revisited. (Letter) (PMID: 9222220)
- Tau gene mutation in familial progressive subcortical gliosis. (PMID: 10202939)
- Comparison of family histories in FTLD subtypes and related tauopathies. (PMID: 16344531)
- Hereditary frontotemporal dementia is linked to chromosome 17q21-q22: a genetic and clinicopathological study of three Dutch families. (PMID: 9029063)
- Transgenic mouse model of tauopathies with glial pathology and nervous system degeneration. (PMID: 12165467)

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- Association of missense and 5-prime-splice-site mutations in tau with the inherited dementia FTDP-17. (PMID: 9641683)
- A distinct familial presenile dementia with a novel missense mutation in the tau gene. (PMID: 10208578)
- Age-dependent emergence and progression of a tauopathy in transgenic mice overexpressing the shortest human tau isoform. (PMID: 10595524)
- Two distinct subtypes of right temporal variant frontotemporal dementia. (PMID: 19884571)
- Pick complex: an integrative approach to frontotemporal dementia: primary progressive aphasia, corticobasal degeneration, and progressive supranuclear palsy. (PMID: 14629785)
- Familial progressive subcortical gliosis. (PMID: 7936288)
- Neuropathologic variation in frontotemporal dementia due to the intronic tau 10 +16 mutation. (PMID: 11971082)
- Neurodegenerative tauopathies. (PMID: 11520930)
- Clinical characteristics of a family with chromosome 17-linked disinhibition-dementia-parkinsonism-amyotrophy complex. (PMID: 7936241)
- Nomenclature for neuropathologic subtypes of frontotemporal lobar degeneration: consensus recommendations. (PMID: 19015862)
- Nomenclature and nosology for neuropathologic subtypes of frontotemporal lobar degeneration: an update. (PMID: 19924424)
- The molecular genetics and neuropathology of frontotemporal lobar degeneration: recent developments. (PMID: 17805587)
- Clinical and pathological diagnosis of frontotemporal dementia: report of the Work Group on Frontotemporal Dementia and Pick's Disease. (PMID: 11708987)
- Accuracy of the clinical evaluation for frontotemporal dementia. (PMID: 17562930)
- The genetic and pathological classification of familial frontotemporal dementia. (PMID: 11708988)
- Familial multiple-system tauopathy with presenile dementia is localized to chromosome 17. (PMID: 9345089)
- Ubiquitinated TDP-43 in frontotemporal lobar degeneration and amyotrophic lateral sclerosis. (PMID: 17023659)
- Familial progressive subcortical gliosis: presence of prions and linkage to chromosome 17. (PMID: 7783864)
- Dementia with prominent frontotemporal features associated with L113P presenilin 1 mutation. (PMID: 11094121)
- The neuropathology of a chromosome 17-linked autosomal dominant parkinsonism and dementia ('pallido-ponto-nigral degeneration'). (PMID: 9630238)
- The heritability and genetics of frontotemporal lobar degeneration. (PMID: 19884572)
- Familial occurrence of amyotrophic lateral sclerosis, parkinsonism, and dementia. (PMID: 6524873)
- Distinct genetic forms of frontotemporal dementia. (PMID: 18703462)
- Differences in tau and apolipoprotein E polymorphism frequencies in sporadic frontotemporal lobar degeneration syndromes. (PMID: 11939896)
- Genome-wide association study reveals genetic risk underlying Parkinson's disease. (Letter) (PMID: 19915575)
- Familial multiple system tauopathy with presenile dementia: a disease with abundant neuronal and glial tau filaments. (PMID: 9108114)
- Frontotemporal lobar degeneration--tau as a pied piper? (PMID: 12481984)
- Clinical features and disease haplotypes of individuals with the N279K tau gene mutation: a comparison of the pallidopontonigral degeneration kindred and a French family. (PMID: 12056930)
- Frameshift proteins in autosomal dominant forms of Alzheimer disease and other tauopathies. (PMID: 16432153)
- Apolipoprotein E gene in frontotemporal dementia: an association study and meta-analysis. (PMID: 12107813)
- Association between the extended tau haplotype and frontotemporal dementia. (PMID: 12056929)
- Atrophy patterns in IVS10+16, IVS10+3, N279K, S305N, P301L, and V337M MAPT mutations. (PMID: 19786698)
- Localization of the gene for rapidly progressive autosomal dominant parkinsonism and dementia with pallido-ponto-nigral degeneration to chromosome 17q21. (PMID: 8789453)
- 17q-linked frontotemporal dementia-amyotrophic lateral sclerosis without tau mutations with tau and alpha-synuclein inclusions. (PMID: 15023818)
- Localization of disinhibition-dementia-parkinsonism-amyotrophy complex to 17q21-22. (PMID: 7977375)
- Rapidly progressive autosomal dominant parkinsonism and dementia with pallido-ponto-nigral degeneration. (PMID: 1416801)
- Linkage of frontotemporal dementia to chromosome 17: clinical and neuropathological characterization of phenotype. (PMID: 8940276)

References (PubMed ID#):

- The structure of the presenilin 1 (S182) gene and identification of six novel mutations in early onset AD families. (PMID: 7550356)
- A novel presenilin-1 mutation (leu85pro) in early-onset Alzheimer disease with spastic paraparesis. (PMID: 15534188)

Hereditary Neurological Screening Result

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- A case of Alzheimer's disease with unusual neurological disturbances. (PMID:)
- Worldwide distribution of PSEN1 Met146Leu mutation: a large variability for a founder mutation. (PMID: 20164095)
- A large pedigree with early-onset Alzheimer's disease: clinical, neuropathologic, and genetic characterization. (PMID: 7824141)
- A variant of Alzheimer's disease with spastic paraparesis and unusual plaques due to deletion of exon 9 of presenilin 1. (PMID: 9546792)
- A novel p.Leu(381)Phe mutation in presenilin 1 is associated with very early onset and unusually fast progressing dementia as well as lysosomal inclusions typically seen in Kufs disease. (PMID: 24121961)
- Chromosomal fragility associated with familial Alzheimer's disease. (PMID: 8053655)
- A presenilin 1 R278I mutation presenting with language impairment. (PMID: 15534260)
- Pick bodies in a family with presenilin-1 Alzheimer's disease. (PMID: 15622541)
- Early-onset Alzheimer's disease due to mutations of the presenilin-1 gene on chromosome 14: a 7-year follow-up of a patient with a mutation at codon 139. (PMID: 9728730)
- Lewy body pathology in familial Alzheimer disease: evidence for disease- and mutation-specific pathologic phenotype. (PMID: 16533963)
- Clinical features of early-onset Alzheimer disease in a large kindred with an E280A presenilin-1 mutation. (PMID: 9052708)
- Dementia, pyramidal system involvement, and leukoencephalopathy with a presenilin 1 mutation. (PMID: 16401857)
- Molecular evidence of presenilin 1 mutation in familial early onset dementia. (PMID: 11920851)
- Novel insertional presenilin 1 mutation causing Alzheimer disease with spastic paraparesis. (PMID: 15159497)
- A locus for familial early-onset Alzheimer's disease on the long arm of chromosome 14, proximal to the alpha-1-antichymotrypsin gene. (PMID: 1303291)
- The A431E mutation in PSEN1 causing familial Alzheimer's disease originating in Jalisco state, Mexico: an additional fifteen families. (Letter) (PMID: 16897084)
- Linkage of familial Alzheimer disease to chromosome 14 in two large early-onset pedigrees: effects of marker allele frequencies on lod scores. (PMID: 8357039)
- Presenilin-1 mutation (E280G), spastic paraparesis, and cranial MRI white-matter abnormalities. (PMID: 12370477)
- Apolipoprotein E-epsilon-4 modifies Alzheimer's disease onset in an E280A PS1 kindred. (PMID: 12891668)
- Association of a presenilin 1 S170F mutation with a novel Alzheimer disease molecular phenotype. (PMID: 17502474)
- Molecular diagnosis of autosomal dominant early onset Alzheimer's disease: an update. (Letter) (PMID: 16033913)
- Presenilin 1 mutation in an African American family presenting with atypical Alzheimer dementia. (PMID: 12810495)
- PS1 Alzheimer's disease family with spastic paraplegia: the search for a gene modifier. (PMID: 14557582)
- Genetic linkage evidence for a familial Alzheimer's disease locus on chromosome 14. (PMID: 1411576)
- Chromosome 14 and late-onset familial Alzheimer disease (FAD). (PMID: 8352272)
- Cloning of a gene bearing mis-sense mutations in early-onset familial Alzheimer's disease. (PMID: 7596406)
- Missense mutation of S182 gene in Italian families with early-onset Alzheimer's disease. (Letter) (PMID: 7623584)
- Genetic evidence for a novel familial Alzheimer's disease locus on chromosome 14. (PMID: 1303289)
- Molecular genetics of Alzheimer disease amyloid. (PMID: 1939107)
- Mapping of a gene predisposing to early-onset Alzheimer's disease to chromosome 14q24.3. (PMID: 1303290)
- Failure of familial Alzheimer's disease to segregate with the A4-amyloid gene in several European families. (PMID: 3306405)
- Founder effect for the ala431glu mutation of the presenilin 1 gene causing early-onset Alzheimer's disease in Mexican families. (PMID: 16628450)

No additional pathogenic/likely pathogenic variants found in the panel genes tested (refer to the Test Methodology section of this report for a complete list of the genes).

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ADDITIONAL INFORMATION

Assay was performed in a CLIA certified laboratory (CLIA#45D2034851).

METHODOLOGY AND LIMITATIONS

This test is a performed using mass array genotyping methodology for genetic profiling of 47 genes associated with Parkinson's-Dementia-Alzheimer's predisposition. This array-based assay detects alleles, including targeted variants with known clinical significance at analytical sensitivity and specificity >99%. Variants in this panel are not FDA approved. Variants were classified following recent guidelines of American College of Medical Genetics and Genomics and the Association for Molecular Pathology.

The genes in this panel are:

Parkinson Dementia Alzheimer's

AAAS, ACE, APOE, APP, ATP13A2, AUP1, BAIAP2L2, CSF1R, DCTN1, DNMT1, EIF4G1, FBXO7, GBA, GBF1, GCH1, GRN, HTRA2, LOC105375056, LOC105377329, LOXL3, LRRK2, MAPT, MPO, NOTCH3, PACRG, PARK7, PICALM, PINK1, PINK1-AS, PLA2G6, POLG, PRKN, PRKRA, PRNP, PSEN1, PSEN2, SLC6A3, SNCA, SNCA-AS1, SNCB, TAF1, TH, TREM2, TREML1, TYROBP, UCHL1, VPS35

Alzheimer's Disease, Parkinson's Disease, and dementia are conditions that affect the brain and spinal cord. There is no cure for Alzheimer's disease, Parkinson's disease, or dementia; however, there are treatments available to provide temporary relief from symptoms.

Parkinson's

Approximately 15 percent of people with Parkinson's disease have a family history of this disorder. Familial cases of Parkinson's disease can be caused by mutations in the LRRK2, PARK7, PINK1, PRKN, or SNCA gene, or by alterations in genes that have not been identified.

Alterations in certain genes, including GBA and UCHL1, do not cause Parkinson's disease but appear to modify the risk of developing the condition in some families. Variations in other genes that have not been identified may also contribute to Parkinson disease risk.

It is not fully understood how genetic changes cause Parkinson's disease or influence the risk of developing the disorder. Many Parkinson's disease symptoms occur when nerve cells die or become impaired. Normally, these cells produce a chemical messenger called dopamine, which transmits signals within the brain to produce smooth physical movements. When these dopamine-producing neurons are damaged or die, communication between the brain and muscles weakens. Eventually, the brain becomes unable to control muscle movement.

Some gene mutations appear to disturb the cell machinery that breaks down unwanted proteins in dopamine-producing neurons. As a result, unwanted proteins accumulate, leading to the impairment or death of these cells. Other mutations may affect the function of mitochondria, the energy-producing structures within cells. As a byproduct of energy production, mitochondria make unstable molecules called free radicals that can damage cells. Cells normally counteract the effects of free radicals before they cause damage, but mutations can disrupt this process. As a result, free radicals may accumulate and impair or kill dopamine-producing neurons.

Dementia

Genes and frontotemporal dementia

Frontotemporal dementia (FTD), originally called Pick's disease, is a rarer form of dementia mostly affecting people under the age of 65. The symptoms of FTD can be quite varied but include changes that predominantly affect behavior or language. There are different types of FTD, and these are likely to have different causes.

Some people with FTD have a family history of dementia and the condition may be inherited in some of these families. For behavioral variant FTD, a third to half of people could have a family history. Several genes have been found that can cause these inherited forms of FTD, including:

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- tau (MAPT)
- progranulin (GRN)
- PSEN1

Genes and vascular dementia

Vascular dementia is caused when blood flow to the brain is reduced, damaging nerve cells. This can happen as a result of a stroke or damage to blood vessels deep in the brain. The majority of cases of vascular dementia are not caused by abnormalities in genes, though an individual might carry genes that affect their risk of stroke, heart disease, or other diseases that may contribute to vascular dementia. However, lifestyle factors such as smoking, lack of exercise, obesity, alcohol and diet are also important.

There are rare genetic disorders that can cause vascular dementia by damaging blood vessels in the brain. One is called CADASIL (cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy) and can be passed down through families.

Genes and dementia with Lewy bodies

Dementia with Lewy bodies is caused by a build-up of abnormal proteins in the brain and may have symptoms similar to those seen in Parkinson's disease. Age is currently the biggest known risk factor for dementia with Lewy bodies, although mutations in two genes (SNCA and SNCB) also play a role. The SNCA and SNCB genes provide instructions for making proteins in the brain, called alpha-synuclein and beta-synuclein, respectively. Alpha-synuclein plays a role in communication between nerve cells, to regulate the release of chemical messengers. Beta-synuclein is involved in a process that allows neurons to change and adapt over time, which is necessary for learning and memory. Beta-synuclein may also prevent harmful accumulation of alpha-synuclein in neurons.

Early Onset Familial Alzheimer's Disease

APP,PSEN1,PSEN2

Early onset familial Alzheimer's disease (eFAD) is hereditary and marked by Alzheimer's disease symptoms that appear at an unusually early age. Symptoms can start in a person's thirties, forties, and fifties (and very rarely in the late twenties). Genetics researchers studied eFAD families to discover the three known genes that cause familial AD: amyloid precursor protein (APP), presenilin-1 (PS1), and presenilin-2 (PS2). Of these, PS1 mutations account for most eFAD, while APP and PS2 are rarer. Having a pathogenic mutation in one of these three genes virtually guarantees that one will develop early onset Alzheimer's disease.

Late Onset Alzheimer's Disease

APOE e4

Late-onset Alzheimer's disease is a condition characterized by memory loss, cognitive decline, and personality changes developing after the age of 65. One in ten Americans age 65 and older is affected by Alzheimer's disease. The most important risk factor for Alzheimer's disease is a gene called Apolipoprotein E, or APOE. This gene has a number of different alleles, called ε2, ε3, and ε4. ε2 and ε3 protect against Alzheimer's, whereas ε4 increases your risk of developing it. Every individual has two copies of this allele, and the combination you have determines how likely you are to develop the disease; ε2/ε2 is the lowest risk, and ε4/ε4 is the highest. There is a lot of evidence that suggests that APOE E4 isoform contributes to Alzheimer's disease pathogenesis through multiple pathways including facilitated amyloid-Î² peptide deposition, lipid transport Neuroinflammation, and cerebrovascular defects. The association of one or two copies of the APOE allele ε4 (i.e., genotypes ε2/ε4, ε3/ε4, ε4/ε4) with late-onset Alzheimer's disease is well documented. The strongest association between the APOE ε4 allele and late onset Alzheimer's disease, relative to the normal control population, is observed with the ε4/ε4 genotype. This genotype occurs in 1% of the normal control population and in 19% of the familial late onset Alzheimer's disease population. The association between APOE e4 and Alzheimer disease is greatest when the individual has a positive family history of dementia.

About Non-Clinically Significant Variants: All individuals carry DNA changes (i.e., variants) and most variants do not increase an individual's risk of Parkinson's-Dementia-Alzheimer's predisposition or other diseases. When identified, variants of uncertain significance (VUS) are reported. Likely benign variants (Favor Polymorphisms) and benign variants (Polymorphisms) are not reported and available data indicate that

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these variants most likely do not cause increased Parkinson's-Dementia-Alzheimer's risk. Present evidence does not suggest that non-clinically significant variant findings be used to modify patient medical management beyond what is indicated by the personal and family history and any other significant clinical findings.

Limitations: This test will not detect all the existing/known alleles that result in altered or inactive tested genes. It only looks at selected variants picked and curated by researchers at Ayass Bioscience. This test does not account for all individual variations in the individual tested. Absence of a detectable gene mutation does not rule out the possibility that a patient has different phenotypes due to the presence of an undetected polymorphism or due to other factors such as drug-gene interactions, co morbidities and lifestyle habits.

Disclaimer: The information presented on this report is provided as general educational health information. The Content is not intended to be a substitute for professional medical advice, diagnosis, or treatment. Ayass Lung Clinic, PLLC developed this test and its performance characteristics. This test has not been cleared or approved by the U.S. Food and Drug Administration (FDA). Ayass Lung Clinic, PLLC is accredited by CLIA for performance of high complexity testing. The Parkinson's-Dementia-Alzheimer's report is one of multiple pieces of information that clinicians should consider in providing medical advice for each patient. This test should be interpreted in context with other clinical findings. It remains the responsibility of the health-care provider to interpret the results of the test.

These assays do not detect polymorphisms other than those outside of the panel. Rare false positive or false negative results may occur. These assays have been developed and performance characteristics determined by Ayass Lung Clinic, PLLC; and therefore, are classified as Laboratory Developed Tests. These assays have not been cleared or approved by the U.S. Food and Drug Administration. The FDA has determined that such clearance is not necessary. Ayass Lung Clinic, PLLC is accredited by CLIA for performance of high complexity testing. These tests are used for clinical purposes and should not be considered as investigational. Results of genotyping should be interpreted in the full context of the patient's clinical history, family history, life style, co-administration of other drugs, and other pre-existing conditions.

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Additional Resources:

https://parkinson.org/
https://www.ninds.nih.gov/
https://www.apdaparkinson.org/
https://www.alz.org/
https://www.alzforum.org/
http://www.alz.org/research/clinical_trials/find_clinical_trials_trialmatch.asp
http://www.brainhealthregistry.org/?utm_source=alzforum&utm_medium=article&utm_campaign=intro_bhr
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