Dementia Assessment Panel

Ordering Provider: **Doogie Howser**
Accession Number: **EM-DEMO**

Patient Name: **John Doe**
Patient Gender: **Male**
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Introduction to The Dementia Assessment Panel

The Dementia Assessment Panel is a health care expert-directed genetic screening tool designed to evaluate genetic predispositions associated with the development and progression of dementia. Dementia is a complex, multifactorial neurodegenerative disorder marked by progressive memory loss, cognitive decline, and functional impairment. It is primarily driven by the abnormal accumulation of amyloid-beta plaques and tau protein tangles in the brain, changes that begin many years before clinical symptoms emerge.

This panel does not serve as a diagnostic tool but offers personalized, evidence-informed insights to support cognitive health planning and proactive risk reduction. By analyzing genetic markers across key pathways involved in amyloid processing, inflammation, neurotransmitter balance, and brain metabolism, health care experts can identify an individual's risk profile and develop tailored interventions to help support brain health and mitigate modifiable risk factors.

What The Dementia Assessment Panel Provides

This panel evaluates single nucleotide polymorphisms (SNPs) associated with various biological processes implicated in dementia, including amyloid deposition, tau pathology, metabolic regulation, inflammation, and brain structural changes. Using an additive genetic model, it categorizes genetic predispositions into High, Medium, or Low risk, based on the cumulative effect of genetic variants.

The insights from this panel allow health care experts to deliver targeted, personalized strategies related to diet, lifestyle, and brain-supportive practices. These proactive recommendations aim to support long-term cognitive function, reduce potential risk factors, and empower individuals to make informed health decisions aligned with the genetic blueprint.

Key Categories and Sub-Categories Assessed

1. **Neurodegenerative Risk Genetics:**

Analyzes genetic variations linked to susceptibility and progression of neurodegenerative conditions, with a primary focus on dementia.

Susceptibility to Dementia: Assesses common genetic variants that influence amyloid and tau-related pathways contributing to dementia development.

APOE Risk Susceptibility: Identifies risk based on APOE allele status, particularly the ε4 variant, which significantly increases the risk of late-onset dementia.

2. **Microbiome–Cognitive Health Profile**

Explores genetic factors that affect the gut-brain axis and its role in cognitive function and neurodegeneration.

Gut-Brain Resilience: Screens for genetic predispositions linking gastrointestinal disorders (e.g., IBS, GERD) with dementia, offering insights into gut-mediated brain health.

3. **Dementia's Metabolic Risk Indicators**

Assesses the genetic interplay between metabolic health and dementia, focusing on insulin signaling and lipid metabolism.

Dementia's Disease-Metabolic Interaction Susceptibility: Evaluates variants linking fasting glucose, insulin, and HDL cholesterol levels with increased dementia susceptibility.

4. **Homocysteine Regulation**

Focuses on genetic influences on homocysteine metabolism, a known risk factor for cognitive decline and vascular damage.

Hyperhomocysteinemia Susceptibility: Identifies variants associated with elevated homocysteine levels, which can accelerate neurodegeneration and oxidative stress.

5. **Neuro-Immune Health**

Examines genetic markers that affect chronic inflammation and immune system activity related to dementia pathogenesis.

Chronic Inflammation Susceptibility: Evaluates predispositions to prolonged microglial activation and neuroinflammatory responses, contributing to cognitive decline.

6. **Nutrient Metabolism Profiles**

Analyzes genetic variants that impact the metabolism of nutrients essential to brain health and immune regulation.

Zinc Metabolism Profile: Screens for genetic influences on zinc transport and utilization, relevant to neurotransmission and amyloid-beta aggregation.

Vitamin D Deficiency Susceptibility: Assesses predisposition to suboptimal vitamin D levels, which may impair neuronal protection and amyloid clearance.

Report Components

Each trait includes the following:

1. **Gene Information:** Relevant gene(s) and the known functional roles.

2. **SNP Information:** rsIDs, allele functions, and biological impact summaries.

3. **Risk Interpretation:** High, Medium, or Low risk categorization based on additive SNP scoring.

4. **Propensity Interpretation:** Categorized as High, Moderate, or Low propensity based on additive scoring of beneficial alleles.

5. **Nutritional Insight:** Evidence-based dietary suggestions where applicable.

6. **Lifestyle Insights:** Recommended lifestyle strategies tailored to the individual's genetic tendencies to support long-term health outcomes.

7. **Supplementation Insight:** Suggested nutrients and co-factors; dosage discretion lies with the physician.

8. **Lab to be Considered**: Suggested biomarkers and functional tests for follow-up and validation.

Analysis Summary

The Summary Analysis is designed to highlight the most impactful and clinically relevant insights derived from an individual's genetic and functional assessment. This summary distills the full set of category-specific recommendations by prioritizing those with the highest potential to influence health outcomes. In the summary analysis, recommendations that can be applied across more than one category are mostly emphasized, as these carry greater overall weight and broader therapeutic value. Recommendations are systematically scored and ranked based on their clinical significance, genetic relevance, and actionable benefit, ensuring that the highest-priority interventions are clearly highlighted for focused implementation. This approach enables individuals to identify the most important strategies to prioritize, making adherence more practical, targeted, and effective for long-term health improvement.

Risk Assessment

Gut-Brain Resilience	High Risk	**Hyperhomocysteinemia Susceptibility**	High Risk
Susceptibility to Dementia	Medium Risk	**APOE Risk Susceptibility**	Medium Risk
Dementia's Disease-Metabolic Interaction Susceptibility	Medium Risk	**Chronic Inflammation Susceptibility**	Medium Risk
Vitamin D Deficiency Susceptibility	Medium Risk	**Zinc Metabolism Profile**	Medium Risk

Risk Assessment & Interventions by Category

Neurodegenerative Risk Genetics	Medium Risk	**Dementia's Metabolic Risk Indicators**	Medium Risk
Susceptibility to Dementia	Medium Risk	Dementia's Disease-Metabolic Interaction Susceptibility	Medium Risk
APOE Risk Susceptibility	Medium Risk		
Neuro-Immune Health	Medium Risk	**Microbiome-Cognitive Health Profile**	High Risk
Chronic Inflammation Susceptibility	Medium Risk	Gut-Brain Resilience	High Risk
Homocysteine Regulation	High Risk	**Nutrient Metabolism Profiles**	Medium Risk

Hyperhomocysteinemia Susceptibility	High Risk
Vitamin D Deficiency Susceptibility	Medium Risk
Zinc Metabolism Profile	Medium Risk

Recommendations & Monitoring Schedule

Lifestyle Considerations

1. Include balance and coordination exercises (e.g., tai chi, yoga)- helps prevent falls and supports motor-cognitive integration.
2. Add 5 minutes of deep breathing or box breathing before bed- activates parasympathetic nervous system; improves sleep and cognitive recovery
3. Add stair climbing or hill walking- natural form of resistance training with aerobic and metabolic benefits.
4. Go for weekly outdoor walks, gardening, or open-air activities- increases vitamin D exposure and physical activity simultaneously.
5. Avoid excessive evening screen time- disrupts circadian rhythm and melatonin release

Nutritional Insights

1. Add berries (blueberries, blackberries, strawberries) daily- high in anthocyanins and antioxidants that counter oxidative stress.
2. Add extra virgin olive oil (EVOO)- monounsaturated fats + polyphenols reduce neuroinflammation.
3. Take macadamia or pecans 4x/week- monounsaturated fat–rich; supports HDL and reduces insulin resistance.
4. Add ground flaxseed or chia seeds- rich in ALA and fiber; supports Bifidobacteria and reduces gut inflammation
5. Add mixed nuts (almonds, cashews, walnuts)-provide plant-based zinc, vitamin E, and omega-3s; lower neuroinflammation risk.

Supplements To Consider

1. Melatonin
2. Omega-3 supplements (fish oil or algal oil)
3. Probiotics (Lactobacillus and Bifidobacterium)
4. Curcumin
5. Zinc picolinate

(Please consult a healthcare professional or a physician before starting any supplements.)

Other Labs To Consider

1. GFAP
2. Gut microbiome test
3. Breath or stool antigen test for Helicobacter pylori
4. Serum Folate
5. Plasma Zinc

Neurodegenerative Risk Genetics

In dementia, abnormal protein buildup interferes with normal brain function, causing a decrease in neurotransmitters like acetylcholine that are vital for communication between brain cells. As these neurotransmitter levels decline, several brain regions, especially those involved in memory, begin to shrink. Early symptoms can sometimes include visual difficulties or speech problems, showing that dementia impacts more than just memory.

Susceptibility to Dementia

Dementia is a progressive neurological condition that impairs memory, thinking, and cognitive function. Its onset and progression are influenced by multiple genetic factors. Understanding an individual's genetic predisposition can provide valuable insights into personal risk. With this knowledge, the individual can take proactive measures, such as adopting healthy lifestyle habits and considering early interventions, that may help delay the onset of symptoms. This empowers the person to make informed healthcare decisions and engage in personalized strategies for prevention and long-term brain health.

High Risk

▶ Medium Risk

Low Risk

Marker : SORL1 | rsID : rsXXXX | Patient Genotype : TT

SORL1 is involved in the trafficking and recycling of amyloid precursor protein (APP), which influences the accumulation of amyloid-beta in the brain. An individual with the high-risk genotype may experience disrupted APP trafficking, potentially leading to the buildup of harmful amyloid plaques in the brain. This accumulation is associated with an increased risk of dementia-related disorders.

Marker : MS4A4A | rsID : rsXXXX | Patient Genotype : AA

MS4A4A plays a role in microglial activation and immune response regulation within the central nervous system. An individual with this genotype carries low-risk variants, which means that the brain's immune systems function effectively, helping to regulate inflammation without overreacting. This can protect brain cells and decrease the risk of developing dementia-related disorders.

Marker : EPHA1-AS1 | rsID : rsXXXX | Patient Genotype : TT

EPHA1 is a receptor tyrosine kinase involved in synaptic function and neuroinflammation, potentially modulating dementia risk. An individual with this genotype carries a low-risk variant; these processes are expected to function efficiently, helping to minimize inflammation and protect neuronal cells, thereby reducing the risk of dementia.

Marker : PLCG2 | rsID : rsXXXX | Patient Genotype : GG

PLCG2 encodes a signaling enzyme that mediates immune cell responses and is associated with neuroinflammation and microglial activity. An individual with this genotype carries low-risk variants, which means the immune systems are better equipped to respond effectively to amyloid plaques and other potentially damaging processes in the brain. This helps maintain inflammation at healthy levels, reducing the risk of neurodegeneration associated with dementia.

Marker : BIN1 | rsID : rsXXXX | Patient Genotype : CC

BIN1 regulates membrane curvature and endocytosis in neurons, which are implicated in tau pathology and dementia progression. Low-risk variants of the BIN1 gene aid essential cellular functions like cholesterol transport and amyloid protein clearance, which help preserve brain health and lower the likelihood of dementia progression.

Marker : MAPT | rsID : rsXXXX | Patient Genotype : CC

MAPT encodes the tau protein, which is critical for microtubule stability, and its mutations and variants are linked to tau aggregation and neurodegenerative disorders. An individual with this high-risk genotype may experience an increased likelihood of abnormal tau protein aggregation, leading to the formation of neurofibrillary tangles. This process can impair neuronal stability and elevate the risk of memory loss and cognitive decline related to dementia.

Marker : GAB2 | rsID : rsXXXX | Patient Genotype : AC

GAB2 acts as a signaling adaptor influencing neuroinflammatory and amyloid-related pathways. An individual with this genotype in the GAB2 gene carries a variant associated with a moderate impact on tau regulation and neuroinflammatory signaling.

Marker : PTK2B | rsID : rsXXXX | Patient Genotype : TC

PTK2B encodes a protein tyrosine kinase involved in calcium signaling and synaptic plasticity, linked to neurodegenerative processes. An individual with this genotype carries a medium-risk profile; hence, these signaling pathways generally function well, but minor disruptions may slightly increase neuronal vulnerability to damage.

Marker : CD33 | rsID : rsXXXX | Patient Genotype : AC

CD33 is an immune receptor expressed on microglia, modulating amyloid-beta clearance and contributing to dementia. An individual with this genotype exhibits a moderate effect on microglial regulation. While some impairment in immune response may occur, the individual's brain's ability to clear debris and control inflammation is likely to remain sufficiently balanced, resulting in a modest increase in dementia risk.

Marker : ACE | rsID : rsXXXX | Patient Genotype : TT

Carrying low-risk variants of the ACE gene allows an individual to effectively regulate blood pressure and support brain function. This helps to maintain healthy brain blood flow and metabolism of amyloid-beta peptides, reducing the risk of amyloid plaque formation and dementia.

Marker : PICALM | rsID : rsXXXX | Patient Genotype : AA

The PICALM gene plays a vital role in how an individual's cells manage certain proteins and fats in the body. It helps clear amyloid proteins from the brain, which can build up and contribute to dementia. With a high-risk version of this gene, these processes may be disrupted, potentially affecting brain health and metabolism. This disruption could increase the individual's risk of dementia while also impacting factors such as blood sugar levels, insulin levels, and good cholesterol (HDL).

Marker : MS4A4A | rsID : rsXXXX | Patient Genotype : TT

The MS4A gene family plays a crucial role in immune responses and cellular signaling, particularly in an individual's brain's microglial cells. Low-risk variants in the MS4A gene family ensure that an individual's immune response and cellular signaling are well-regulated, particularly in microglial cells. This regulation promotes effective amyloid clearance and minimizes inflammation, contributing to a lower risk of dementia for an individual with this genotype.

Marker : LRAT | rsID : rsXXXX | Patient Genotype : TT

LRAT is vital for vitamin A metabolism, essential for brain and cellular health. An individual with this genotype carries low-risk variants of the LRAT gene, which support normal vitamin A metabolism and retinoid signaling. This helps maintain healthy neuronal function and reduces the risk of dementia-related cognitive decline.

Marker : BIN1, NIFKP9 | rsID : rsXXXX | Patient Genotype : AG

An individual with this BIN1 genotype may experience moderate cholesterol regulation and amyloid processing. While some disruption is possible, its impact on dementia risk is likely to be moderate and influenced by other factors.

Marker : ATXN2L | rsID : rsXXXX | Patient Genotype : TC

ATXN2L is involved in critical cellular processes like RNA metabolism and protein translation. An individual with this genotype carries a medium-risk variant in ATXN2L, which generally supports these functions but may introduce minor inefficiencies in protein processing. These subtle disruptions could moderately increase the risk of dementia progression.

Marker : MS4A2 | rsID : rsXXXX | Patient Genotype : AA

The MS4A family is essential for immune responses and cellular signaling in an individual's brain's microglial cells. An individual with this genotype carries a high-risk variant in these genes, which may alter immune responses by affecting the levels of soluble TREM2 (sTREM2), a protein critical in dementia. These changes can impair amyloid clearance and increase inflammation, significantly raising the risk of dementia progression.

Risk Assessment

The individual's genetic profile shows a moderate risk of dementia-related disorders. This implies a potential likelihood of earlier development or faster progression compared to average population risk.

Lifestyle Considerations

Instead of isolated walks, go for walks with a friend, neighbor, or pet- boosts oxytocin, stress resilience, and verbal interaction

Nutritional Insights

1. Add cooked lentils or beans- low glycemic and rich in fiber; stabilizes blood sugar and reduces insulin spikes
2. Add steel-cut oats- beta-glucans support brain-friendly gut bacteria and cholesterol regulation
3. Remove processed meats (sausages, bacon)-contain nitrosamines; linked to neurodegeneration
4. Replace sugary yogurt with plain Greek yogurt + blueberries-removes added sugars and enhances antioxidant intake

Supplements To Consider

1. Curcumin
2. Resveratrol
3. EGCG
4. Flavonoids (Baicalein, Quercetin, Luteolin, Cinnamon extract, Grape seed extract, Gallic acid, Ginkgo biloba)
5. Souble fiber (beta glucan)

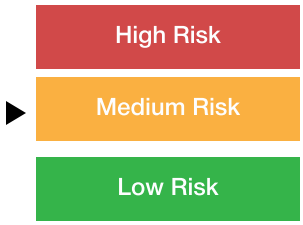
(Please consult a healthcare professional or your physician before starting any supplements)

Other Labs To Consider

1. hs-CRP
2. IL-6
3. Homocysteine
4. Advanced Lipid Panel

APOE Risk Susceptibility

The APOE (Apolipoprotein E) gene plays a significant role in brain health, particularly in lipid transport and neuronal repair processes. It has three common alleles: ε2, ε3, and ε4, which combine to form six possible genotypes (ε2/ε2, ε2/ε3, ε2/ε4, ε3/ε3, ε3/ε4, and ε4/ε4). Each allele influences the risk of developing dementia differently, with ε4 increasing the risk, ε3 being neutral, and ε2 offering a degree of protection.



Marker : APOE | rsID : rsXXXX | Patient Genotype : TC

APOE is a gene that regulates fat transport and amyloid-beta clearance, with certain variants linked to increased dementia risk. An individual with the ε3/ε4 genotype is placed in the medium-risk category for dementia. This may lead to moderately reduced efficiency in clearing amyloid-beta, increasing the susceptibility to dementia with age.

Marker : APOE | rsID : rsXXXX | Patient Genotype : TC

APOE plays a role in managing brain fat metabolism and eliminating amyloid-beta, with specific variants influencing dementia susceptibility. An individual with the ε3/ε4 combination falls into the moderate-risk group. This may somewhat impair amyloid-beta clearance, raising the potential for cognitive decline as one ages.

Risk Assessment

The individual's APOE profile suggests a moderately increased lifetime risk of dementia relative to the general population. While this does not mean dementia will develop, preventive lifestyle and nutritional measures may help reduce modifiable risks.

Lifestyle Considerations

1. Include balance and coordination exercises (e.g., tai chi, yoga)- helps prevent falls and supports motor-cognitive integration.
2. Limit long sedentary periods (sit <60 mins without movement break)- movement breaks help circulation and prevent metabolic slowdown.
3. Opt for outdoor physical activity when possible- combines exercise with natural light and vitamin D synthesis.

Nutritional Insights

1. Add berries (blueberries, blackberries, strawberries) daily- high in anthocyanins and antioxidants that counter oxidative stress.
2. Add leafy greens (spinach, kale, arugula)- provide folate and lutein, linked to better memory and slower cognitive decline.
3. Add extra virgin olive oil (EVOO)- monounsaturated fats + polyphenols reduce neuroinflammation.
4. Remove trans fats and partially hydrogenated oils- strongly associated with neurodegeneration and memory decline.
5. Replace fruit juice with whole fruit or infused water- less sugar, more fiber and nutrients for brain and metabolic health.

Supplements To Consider

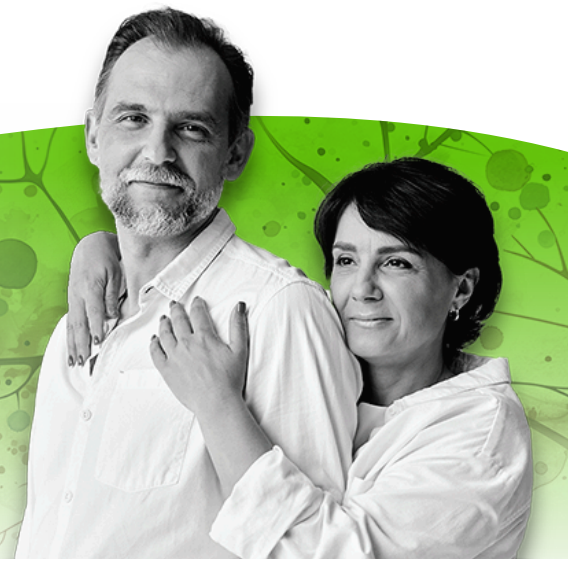
1. Omega-3 supplements (fish oil or algal oil)
2. Vitamin C
3. Vitamin E

(Please consult a healthcare professional or your physician before starting any supplements)

Other Labs To Consider

1. Total, Direct, and Indirect bilirubin
2. LDL cholesterol
3. GFAP

Dementia's Metabolic Risk Indicators



This category explores the link between metabolic processes and the risk of dementia. It examines genetic variations that affect how metabolic factors, such as insulin resistance, HDL, cholesterol levels, and fasting glucose, interact with neurodegenerative changes. This provides a comprehensive understanding of susceptibility to dementia connected to metabolic health.

Dementia's Disease-Metabolic Interaction Susceptibility

Dementia's Disease-Metabolic Interaction Susceptibility [DMI] refers to the genetic predisposition that links Dementia with certain metabolic traits, including fasting glucose, fasting insulin, and HDL cholesterol levels. Recent studies have identified significant genetic correlations between these metabolic factors and dementia, suggesting that shared genetic variants may contribute to both conditions. Understanding DMI Susceptibility can provide valuable insights into the underlying biological mechanisms connecting dementia with metabolic health, potentially leading to more personalized preventive strategies and interventions that address both cognitive and metabolic health.

- High Risk
- ▶ Medium Risk
- Low Risk

Marker : MADD | rsID : rsXXXX | Patient Genotype : GG

The MADD gene plays an important role in both insulin and TNF (tumor necrosis factor) signaling pathways, which are essential for metabolic regulation and inflammatory response. An individual with a high-risk variant may experience impairment in these pathways, potentially leading to disrupted glucose metabolism, increased oxidative stress, and heightened brain inflammation. These effects are closely linked to neurodegeneration and may significantly raise the risk of developing dementia for this individual.

Marker : PICALM | rsID : rsXXXX | Patient Genotype : AG

PICALM supports synaptic vesicle recycling and Aβ clearance, with additional roles in modulating lipid and glucose metabolism. An individual with a medium-risk variant may experience partial inefficiency in these cellular processes under stress or aging conditions. This may contribute to mild impairments in metabolic regulation and an increased risk for neurodegeneration over time.

Marker : CLU | rsID : rsXXXX | Patient Genotype : CC

The CLU gene, also known as Clusterin, plays a key role in managing inflammation and clearing waste from brain cells. It also supports lipid metabolism, particularly cholesterol transport and amyloid clearance. An individual with a high-risk version of this gene may face challenges in managing these critical processes. This could lead to altered cholesterol metabolism and reduced control of inflammation, increasing susceptibility to dementia.

Marker : GCKR | rsID : rsXXXX | Patient Genotype : CC

GCKR (Glucokinase Regulator) plays a vital role in glucose metabolism and lipid regulation. An individual with low-risk variants of the GCKR gene supports healthy glucose metabolism and lipid regulation. This helps maintain metabolic balance, reducing the risk of inflammation and dementia.

Marker : GCKR | rsID : rsXXXX | Patient Genotype : CC

GCKR (Glucokinase Regulator) supports stable glucose metabolism and efficient lipid regulation. An individual with low-risk variants of the GCKR gene may experience effective metabolic control and homeostasis. This helps minimize inflammation, contributing to a lower risk of dementia.

Risk Assessment

The individual has a moderate genetic predisposition to Dementia's Disease–Metabolic Interaction (DMI) Susceptibility, indicating a genetic connection between dementia and certain metabolic traits. Shared genetic variants may contribute to an increased risk for both cognitive decline and metabolic dysfunction.

Lifestyle Considerations

1. Add stair climbing or hill walking- natural form of resistance training with aerobic and metabolic benefits.
2. Do walking lunges while carrying groceries or light weights- turns daily activities into functional strength training
3. Add balance training (e.g., standing on one foot while brushing teeth)- supports proprioception and brain-body integration

Nutritional Insights

1. Add 1 tsp matcha powder or cacao nibs to smoothies- boosts EGCG or flavanols; enhances cerebral blood flow and insulin sensitivity.
2. Include sweet potato or purple yam- provides resistant starch + antioxidants for gut-brain health and glucose balance.
3. Take macadamia or pecans 4x/week- monounsaturated fat–rich; supports HDL and reduces insulin resistance.
4. Replace high-sugar granola with sprouted buckwheat + seeds + cinnamon- low sugar and full of polyphenols and fiber.
5. Avoid microwave popcorn (with artificial butter/oils)- high in trans fats and perfluorochemicals that harm brain health..

Supplements To Consider

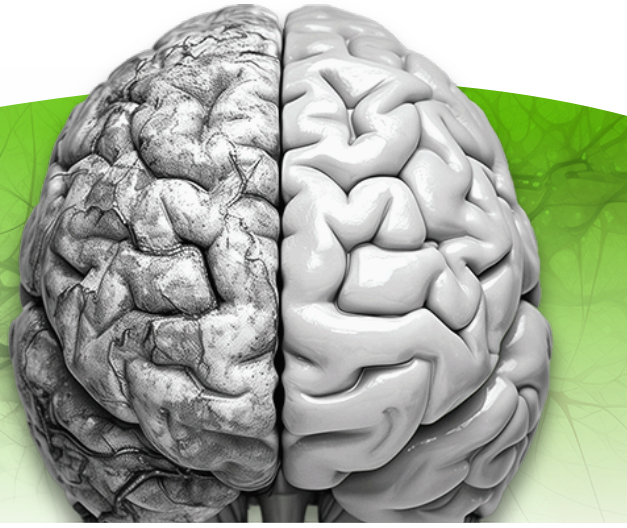
1. Vitamin C
2. CoQ10
3. Selenium
4. Polyphenols
5. Probiotics
6. MCTs

(Please consult a healthcare professional or your physician before starting any supplements.)

Other Labs To Consider

1. Fasting Insulin
2. HbA1C
3. Lipid Profile

Neuro-Immune Health



The relationship between the immune system and brain health is central to this category, which examines genetic markers linked to chronic inflammation. Understanding these predispositions helps identify risks for neuroinflammation, a key driver of many neurodegenerative diseases.

Chronic Inflammation Susceptibility

Inflammatory markers are substances that signal the presence of inflammation in the body. While inflammation is a natural part of the immune response, chronic inflammation can become harmful over time. In dementia, ongoing brain inflammation—often driven by immune cells called microglia—is believed to play a central role in the progression of neurodegeneration. Microglia respond to changes in the brain, such as protein buildup, by releasing pro-inflammatory molecules that may exacerbate neuronal damage and accelerate cognitive decline. Genetic variants can influence how the body regulates inflammation, and certain variants may be associated with elevated inflammatory activity, potentially contributing to the development or progression of dementia.

- High Risk
- ▶ Medium Risk
- Low Risk

Marker : MFHAS1, CLDN23 | rsID : rsXXXX | Patient Genotype : CC

MFHAS1 and CLDN23 ensure balanced immune signaling and support the structural integrity of the blood-brain barrier. An individual with this genotype carries low-risk variants that promote proper control of TLR, TNF, and JAK-STAT pathways, limiting excessive inflammatory activity. This functional stability helps preserve BBB function and provides protection against neuroinflammatory processes, reducing the likelihood of developing dementia.

Marker : BMPR2 | rsID : rsXXXX | Patient Genotype : CC

BMPR2 facilitates normal neurodevelopment and immune modulation through its role in TGF-β signaling, aiding brain tissue maintenance and regulating immune responses. An individual with this genotype carries low-risk variants that support effective anti-inflammatory signaling and suppress excessive TLR pathway activation. This promotes immune stability in the brain and reduces the likelihood of chronic neuroinflammation and dementia.

Marker : IL1RN- IL1F10 | rsID : rsXXXX | Patient Genotype : GG

An individual with this genotype possesses variants of IL1RN and IL1F10 that support balanced inflammation. This helps limit JAK-STAT activation, keeping neuroinflammation low and reducing the risk of dementia.

Marker : TNF | rsID : rsXXXX | Patient Genotype : GG

TNF supports inflammatory responses that defend against threats but can be harmful when overactive. An individual with this genotype carries a low-risk variant that helps maintain balanced immune signaling in the brain. This reduces microglial stress, supports blood-brain barrier stability, and lowers the risk of inflammation-related dementia.

Marker : TLR4 | rsID : rsXXXX | Patient Genotype : AA

The TLR genes encode innate immune receptors that detect threats and trigger inflammation. A high-risk variant is linked to heightened immune sensitivity, causing excessive microglial activation, sustained cytokine release, and chronic neuroinflammation. This disrupts neuronal repair, increases oxidative stress, and significantly elevates the risk of dementia for an individual with this genotype.

Marker : TGF | rsID : rsXXXX | Patient Genotype : AG

TGF-β1 plays a key role in regulating neuroinflammation and supporting neuronal repair. An individual with this genotype may exhibit moderate TGF signaling, characterized by largely functional anti-inflammatory control but mild inefficiencies under stress. This can lead to periodic neuroinflammation and reduced repair capacity following injury or aging. Over time, these imbalances may gradually elevate the risk of cognitive decline and dementia.

Marker : ADCY9 | rsID : rsXXXX | Patient Genotype : TT

ADCY9 (Adenylate Cyclase 9) plays a critical role in cyclic AMP (cAMP) signaling, which regulates various cellular processes, including energy balance. An individual with a high-risk ADCY9 variant may experience impaired cAMP signaling, disrupting energy regulation and promoting chronic inflammation. This dysfunction can activate the JAK-STAT pathway, contributing to neurodegeneration and increasing the individual's risk of dementia.

Markers : CD33, LOC107985327 | rsID : rsXXXX | Patient Genotype : CC

CD33 is an immune receptor expressed on microglia that modulates the activation and plays a role in clearing cellular debris and inflammatory signals in the brain. An individual with this genotype carries a high-risk variant associated with increased expression or impaired receptor function, leading to dysregulated microglial activity. This impairment reduces the efficiency of clearing toxic substances and dampening inflammation, resulting in sustained neuroinflammatory responses. Over time, this chronic immune activation elevates the risk of developing dementia.

Marker : HPR-TXNL4B | rsID : rsXXXX | Patient Genotype : GG

HPR and TXNL4B support proper regulation of immune and stress responses. An individual with this genotype carries low-risk variants that help control TLR signaling and limit chronic inflammation. This promotes microglial stability and neuroimmune balance. As a result, the risk of inflammation-driven dementia is reduced.

Marker : ERCC2 | rsID : rsXXXX | Patient Genotype : TG

ERCC2 is essential for DNA repair and maintaining genomic stability in an individual's cells. An individual with this genotype carries a medium-risk genotype in ERCC2, which generally supports DNA repair processes but may introduce minor inefficiencies. These subtle disruptions could moderately increase oxidative damage in neurons, slightly elevating the risk of neurodegeneration linked to dementia.

Marker : TRIB1AL-TRIB1 | rsID : rsXXXX | Patient Genotype : GG

TRIB1AL and TRIB1 are genes crucial for lipid metabolism and regulating inflammatory pathways. An individual with this genotype carries low-risk variants of these genes, which support healthy lipid metabolism and inflammatory regulation. This helps maintain metabolic balance and reduces the risk of dementia.

Marker : IL6R | rsID : rsXXXX | Patient Genotype : CC

IL6R plays a role in controlling immune signaling through the IL-6 and JAK-STAT pathway. An individual with this genotype carries low-risk variants that help maintain regulated inflammatory responses. This reduces excessive neuroimmune signaling and preserves neuronal function. As a result, the risk of inflammation-related cognitive decline is lower.

Risk Assessment

The individual's genetic profile indicates a moderate predisposition to elevated inflammation. While not strongly predictive, inflammation may still exert some influence on brain health during the aging process.

Lifestyle Considerations

1. Replace elevator use with stairs when feasible- incorporates NEAT (non-exercise activity thermogenesis)
2. Avoid excessive evening screen time- disrupts circadian rhythm and melatonin release

Nutritional Insights

1. Replace white bread with sprouted or sourdough whole grain bread- better gut tolerance and nutrient density
2. Remove microwave popcorn in bags with artificial flavorings- often contains trans fats and harmful packaging chemicals
3. Add high-polyphenol spices (turmeric, rosemary, oregano, thyme)- flavonoids protect neurons and reduce oxidative damage
4. Add beets, carrots, and sweet potatoes- fiber + anti-inflammatory pigments + vascular support

Supplements To Consider

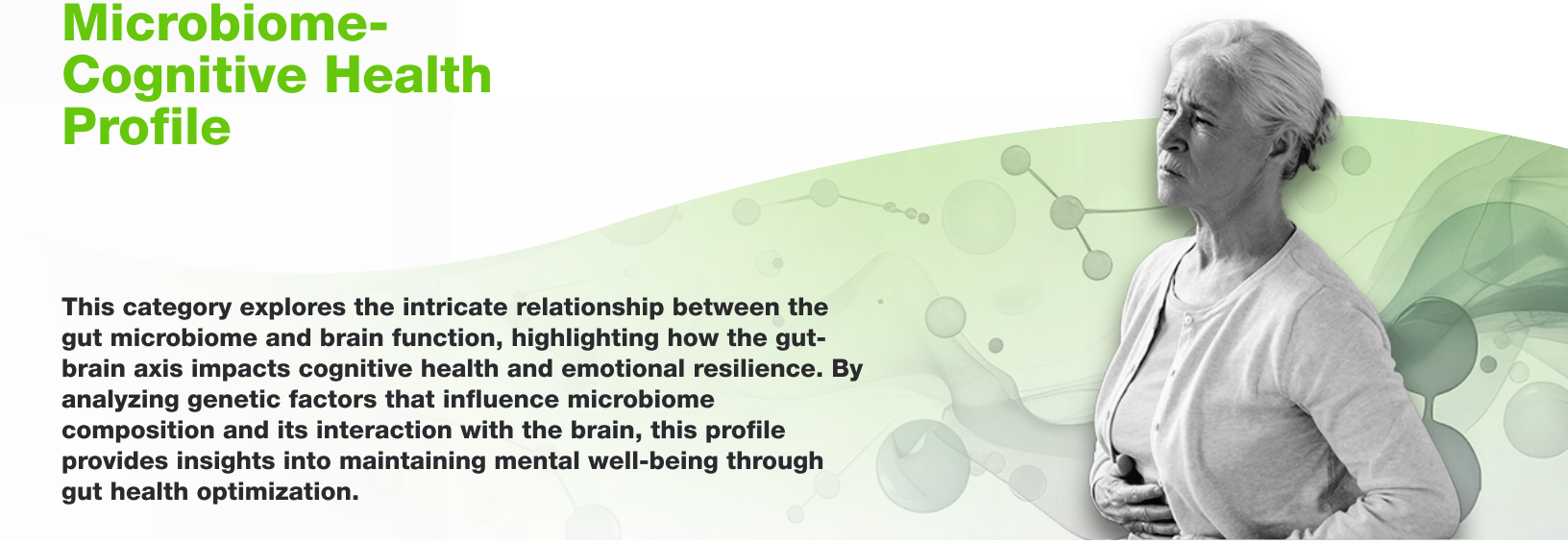
1. Green tea extract
2. Plant Sterols
3. Curcumin
4. Resveratrol
5. EGCG
6. Beta - Glucan

(Please consult a healthcare professional or your physician before starting any supplements)

Other Labs To Consider

1. Hs-CRP
2. Erythrocyte Sedimentation Rate (ESR)
3. Homocysteine
4. Serum Amyloid A (SAA)
5. IL - 6

Microbiome-Cognitive Health Profile



This category explores the intricate relationship between the gut microbiome and brain function, highlighting how the gut-brain axis impacts cognitive health and emotional resilience. By analyzing genetic factors that influence microbiome composition and its interaction with the brain, this profile provides insights into maintaining mental well-being through gut health optimization.

Gut-Brain Resilience

This trait reflects a genetic predisposition to a potential relationship between gastrointestinal tract (GIT) disorders and dementia. Recent studies suggest shared genetic factors may exist between conditions such as gastroesophageal reflux disease (GERD), peptic ulcer disease (PUD), and irritable bowel syndrome (IBS), and the development of dementia. A higher resilience in this area may indicate a lower genetic susceptibility to the gut-brain connection, potentially influencing both digestive health and cognitive function with age. Exploring these genetic links can provide valuable insights into maintaining overall well-being and reducing the risk of GIT-related and neurodegenerative conditions.

▶ High Risk

Medium Risk

Low Risk

Marker : FUT2 | rsID : rsXXXX | Patient Genotype : AA

FUT2 influences mucosal glycan modification, mucosal immunity, and gut-brain communication. An individual with a high-risk non-secretor variant experiences a reduction in fucosylated glycans, which limits Bacteroides colonization and microbiome diversity. This impairment can negatively affect gut-brain signaling and increase intestinal permeability. The resulting dysbiosis promotes systemic and neuroinflammation, subsequently raising the risk of dementia.

Risk Assessment

The individual's genetic profile suggests a higher susceptibility to the potential connection between gastrointestinal tract (GIT) disorders and dementia. This indicates an increased likelihood of experiencing GIT conditions such as gastroesophageal reflux disease (GERD), peptic ulcer disease (PUD), or irritable bowel syndrome (IBS) alongside an elevated risk for dementia.

Lifestyle Considerations

1. Add 5 minutes of deep breathing or box breathing before bed- activates parasympathetic nervous system; improves sleep and cognitive recovery
2. Avoid smoking or exposure to secondhand smoke- tobacco toxins accelerate brain aging and deplete antioxidants like vitamin C and glutathione
3. Replace evening alcohol or caffeine with herbal tea (e.g., chamomile, tulsi, or lemon balm)- promotes relaxation and helps regulate circadian rhythm without neurostimulation

Nutritional Insights

1. Add plain yogurt (unsweetened) as a snack or base- delivers Bifidobacterium strains; supports mood, gut lining, and immune modulation
2. Add rolled or steel-cut oats- provides beta-glucan (soluble fiber)
3. Add ground flaxseed or chia seeds- rich in ALA and fiber; supports Bifidobacteria and reduces gut inflammation
4. Consume ripe banana- resistant starch and inulin; supports SCFA production and E. rectale growth
5. Remove refined grains (e.g., white bread, pasta)- low in fiber, disrupt gut balance, and promote inflammation
6. Remove alcohol (especially hard liquor and beer)- disrupts microbial balance and promotes leaky gut
7. Remove deep-fried snacks (chips, pakoras, fries)- high in trans fats and AGEs; promote oxidative stress and gut barrier disruption
8. Replace potato chips with roasted chickpeas or mixed seeds- adds fiber, protein, and prebiotics while reducing trans fats
9. Replace white bread with sprouted grain or whole grain bread- increases fiber and lowers glycemic load

Supplements To Consider

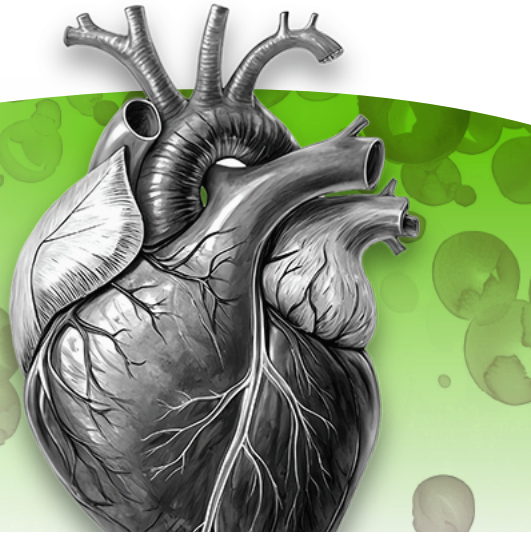
1. Probiotics (Lactobacillus and Bifidobacterium)
2. Vitamins (ADEK)
3. B- complex vitamins
4. Omega 3 fatty acids
5. Curcumin
6. Resveratrol
7. Melatonin

(Please consult a healthcare professional or your physician before starting any supplements.)

Other Labs To Consider

1. Gut microbiome test
2. Liver function test
3. Kidney function test
4. Vitamin B12
5. Folate
6. CRP
7. Breath or stool antigen test for Helicobacter pylori

Homocysteine Regulation



Homocysteine levels are a critical biomarker for cardiovascular and neurological health. This category identifies genetic factors that influence homocysteine metabolism, shedding light on predispositions to elevated levels, which are associated with increased risks of neurodegeneration and cardiovascular conditions.

Hyperhomocysteinemia Susceptibility

Homocysteine is an amino acid in the blood that, when elevated, has been linked to an increased risk of dementia related disorder. High levels of homocysteine can contribute to neurodegenerative processes by promoting inflammation, oxidative stress, and vascular damage, all of which can damage brain cells and accelerate cognitive decline. In dementia, elevated homocysteine levels are thought to exacerbate the development and progression of the disease by influencing amyloid-beta deposition and tau phosphorylation.

- ▶ High Risk
- Medium Risk
- Low Risk

Marker : HNF1A | rsID : rsXXXX | Patient Genotype : AC

HNF1A supports key metabolic processes involving glucose control and homocysteine regulation. An individual with this genotype reflects moderate risk, with generally intact function but some potential inefficiencies in nutrient-related pathways. These inefficiencies may lead to mildly increased homocysteine levels under stress or poor diet. Over time, this can modestly raise an individual's risk of vascular-related cognitive decline.

Marker : MTHFR | rsID : rsXXXX | Patient Genotype : AG

MTHFR supports folate activation and homocysteine clearance, contributing to healthy methylation and neurological function. An individual with this genotype has a moderate genetic risk, with partially reduced MTHFR activity. This may lead to mildly elevated homocysteine levels, particularly when folate or B-vitamin intake is insufficient. Over time, this can modestly increase the risk of cognitive and cardiovascular concerns.

Marker : MTHFR C677T | rsID : rsXXXX | Patient Genotype : AG

MTHFR helps convert dietary folate into its active form and supports detoxification and DNA methylation through homocysteine regulation. An individual with this genotype has a moderate genetic risk, with slightly compromised enzyme activity that may lead to inefficient folate use. While not severely impaired, this can cause mild homocysteine elevation under conditions such as poor diet or stress. This may increase the vulnerability to subtle metabolic and cognitive imbalances over time.

Marker : MTR | rsID : rsXXXX | Patient Genotype : GG

MTR encodes an enzyme that uses vitamin B12 to convert homocysteine into methionine, supporting methylation and brain health. An individual with this genotype carries a high-risk variant that may reduce enzyme efficiency and lead to elevated homocysteine levels. This can disrupt methylation-dependent processes like neurotransmitter synthesis and neural repair. Over time, this increases the risk of cognitive decline and dementia.

Marker : MTRR A66G | rsID : rsXXXX | Patient Genotype : AG

MTRR helps sustain methylation by reactivating MTR through B12 recycling. An individual with this genotype indicates moderate risk, with mild enzyme inefficiency and possible homocysteine elevation under low nutrient intake. This may subtly affect brain methylation and memory processes. While the impact is modest, it could contribute to cognitive aging if unaddressed.

Risk Assessment

The individual has a high genetic predisposition to elevated homocysteine levels, which may increase the likelihood of developing dementia. Elevated homocysteine is associated with neurodegeneration through mechanisms such as inflammation, oxidative stress, and vascular damage, potentially contributing to long-term brain changes.

Lifestyle Considerations

1. Go for bi-annual or quarterly lab testing for homocysteine, B12, folate, and inflammatory markers- early tracking can prevent silent progression and guide targeted intervention
2. Add kettlebell swings or farmer carries 2–3x/week- functional strength training; supports mitochondrial health and insulin regulation.

Nutritional Insights

1. Consume eggs (especially yolks)- rich in B12 and choline; supports methylation and brain structure
2. Add wild-caught salmon or sardines (2x/week)- high in B12, omega-3s, and anti-inflammatory nutrients
3. Include spinach or Swiss chard- excellent source of folate and antioxidants
4. Remove processed meats (e.g., bacon, sausage, deli meats)- contain nitrates and advanced glycation end products; linked to brain aging
5. Remove sugary cereals and baked goods- refined carbs increase insulin resistance and inflammation
6. Replace white rice with quinoa or lentils- higher in folate and fiber; supports steady blood sugar and methylation

Supplements To Consider

1. Vitamin B6
2. Methylfolate

(Please consult a healthcare professional or your physician before starting any supplements.)

Other Labs To Consider

1. Homocysteine
2. Vitamin B12

Nutrient Metabolism Profiles

This category evaluates genetic factors that affect the metabolism of essential nutrients critical for cognitive and overall health. By focusing on variations impacting zinc metabolism and vitamin D utilization, it provides insights into maintaining immunity, bone health, and optimal brain function.

Vitamin D Deficiency Susceptibility

Vitamin D deficiency has been associated with an increased risk of dementia-related disorders due to its critical role in brain health. Vitamin D participates in key neurobiological processes, including the regulation of amyloid-β (Aβ) plaque formation and tau protein dynamics. Inadequate levels can impair cognitive function and heighten susceptibility to neurodegeneration. Maintaining sufficient vitamin D levels is essential for preserving neuronal health and may help reduce the risk of dementia. Genetic factors that influence vitamin D metabolism and absorption can affect susceptibility to deficiency and its potential impact on cognitive decline.

- High Risk
- ▶ Medium Risk
- Low Risk

Marker : VDR | rsID : rsXXXX | Patient Genotype : GG

VDR supports efficient vitamin D signaling, promoting brain health through inflammation control and calcium homeostasis. An individual with this genotype carries a low-risk variant that allows for effective receptor function even under suboptimal vitamin D conditions. This supports microglial balance and neuronal resilience. As a result, the individual's genetic risk for dementia due to impaired vitamin D signaling is low.

Marker : ZPR1 | rsID : rsXXXX | Patient Genotype : CC

ZPR1 maintains cellular signaling and supports proper vitamin D function, which is critical for systemic and neurological health. An individual with this genotype carries a low-risk variant that enables stable vitamin D signaling and cellular communication. This promotes neuronal homeostasis and offers protection from oxidative or inflammatory stress. As a result, the risk of neurodegenerative effects linked to impaired vitamin D pathways is low.

Marker : UGT2B7 - SPOPLP1 | rsID : rsXXXX | Patient Genotype : TT

UGT2B7 supports balanced metabolism of compounds, including vitamin D processing, while SPOPLP1 does not appear to interfere with this function. An individual with this genotype carries low-risk variants that help maintain stable vitamin D levels and metabolic efficiency. This supports optimal brain immune regulation and antioxidant defense. As a result, the risk of dementia related to vitamin D imbalance is low.

Marker : CYP24A1 | rsID : rsXXXX | Patient Genotype : TC

CYP24A1 controls vitamin D breakdown, ensuring balance for systemic and neurological functions. An individual with this genotype carries a medium-risk profile, which may lead to moderately reduced active vitamin D levels. This could limit vitamin D's role in immune modulation and neuronal maintenance under certain conditions. Over time, this may contribute to mild neuroinflammation and increased vulnerability to dementia.

Marker : CYP2R1 | rsID : rsXXXX | Patient Genotype : AA

CYP2R1 supports the initial metabolic step in vitamin D activation, maintaining systemic vitamin D availability. An individual with this genotype carries a low-risk variant that supports efficient conversion of vitamin D to its circulating form. This helps maintain stable vitamin D signaling in the brain. As a result, that individual's risk of vitamin D–related cognitive decline is low.

Marker : ALDH1A2 | rsID : rsXXXX | Patient Genotype : AG

ALDH1A2 plays a role in generating retinoic acid, which supports neuronal differentiation and complements vitamin D function. An individual with this genotype reflects a moderate reduction in enzyme efficiency, potentially affecting vitamin A activity under stress or nutrient deficiency. This may subtly impact neuronal maintenance and immune balance. Over time, it may contribute to mild cognitive vulnerability.

Marker : ALDH1A2, LIPC | rsID : rsXXXX | Patient Genotype : TC

ALDH1A2 and LIPC regulate key components of fat-soluble vitamin signaling and metabolism. An individual with this genotype carries moderate-risk variants that may lead to mild inefficiencies in vitamin A and D pathways. This could modestly reduce neuroimmune balance and stress resilience in the brain. Over time, this may slightly increase the risk for neurodegeneration.

Risk Assessment

The individual's genetic makeup indicates a moderate likelihood of vitamin D deficiency, which may subtly influence brain health over time. Adequate vitamin D supports cognitive function and may help slow pathological processes linked to dementia, such as abnormal protein accumulation.

Lifestyle Considerations

1. Go for weekly outdoor walks, gardening, or open-air activities- increases vitamin D exposure and physical activity simultaneously.
2. Adopt smoking cessation support if applicable- smoking reduces serum vitamin D and accelerates brain aging.

Nutritional Insights

1. Add daily egg yolk- contains vitamin D, choline, and lutein supports memory and neuronal signaling.
2. Avoid low-fat obsession if it excludes fat-soluble vitamin sources- vitamin D requires fat for absorption; fat-free diets impair bioavailability.
3. Replace soda or juice with fortified plant-based milk or kefir- provides vitamin D, B12, and probiotics instead of empty sugar.

Supplements To Consider

1. Omega 3
2. B vitamins

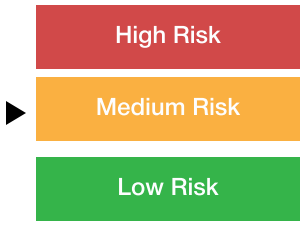
(Please consult a healthcare professional or your physician before starting any supplements)

Other Labs To Consider

1. Vitamin D (serum 25(OH)D)
2. Insulin-like growth factor 1 (IGF-1)
3. Testosterone

Zinc Metabolism Profile

Zinc, a crucial element in brain function, influences neurotransmission by modulating NMDA and AMPA receptors. Particularly noteworthy is its involvement in the aggregation of amyloid-β (Aβ) peptides, a key feature in dementia pathology, leading to the formation of neurotoxic species associated with the condition. Imbalances in zinc levels can contribute to cognitive impairments and memory deficits observed in dementia. Understanding the impact of zinc on dementia pathogenesis is vital for therapeutic considerations and further genetic assessment, including exploring the genetic predisposition to zinc deficiency and its potential relevance in dementia related disease risk assessment.



Marker : PPCDC | rsID : rsXXXX | Patient Genotype : TT

PPCDC ensures proper coenzyme A production and supports zinc-dependent cellular functions critical for brain health. An individual with this genotype carries a low-risk variant that helps maintain efficient zinc metabolism and metabolic resilience. This supports stable neurotransmitter activity and reduces oxidative damage in the brain. As a result, that individual's risk of dementia related to zinc imbalance is low.

Marker : NBDY | rsID : rsXXXX | Patient Genotype : TT

NBDY encodes a regulatory microprotein that modulates mRNA degradation and cellular stress adaptation. An individual with this genotype carries a high-risk variant that may impair the ability of the cells to respond to stress and manage micronutrients like zinc. This could lead to reduced zinc availability, affecting cellular repair and immune balance in the brain. Chronic zinc deficiency may increase oxidative damage and inflammation, contributing to cognitive decline and dementia risk.

Marker : CA1 | rsID : rsXXXX | Patient Genotype : AG

An individual with this genotype carries a moderate-risk variant of the CA1 gene, which may partially affect the zinc balance and enzyme efficiency. While the system generally manages pH and metabolic stability well, periods of nutritional imbalance could still challenge the brain's resilience, slightly increasing vulnerability to neuroinflammation over time.

Risk Assessment

The individual's genetic profile shows a moderate tendency toward lower zinc levels, which may influence brain health over time. Zinc supports cognitive performance and helps protect against the buildup of neurotoxic proteins associated with dementia.

Lifestyle Considerations

1. Add routine screening and management for gut conditions (IBS, leaky gut, IBD)-chronic gut issues can impair zinc and B vitamin absorption.
2. Remove overuse of antacids or PPIs without necessity-low stomach acid significantly reduces zinc and B12 absorption.
3. Replace frying or grilling with steaming, pressure cooking, or stewing-reduces nutrient loss and phytate content; retains zinc and fiber.
4. Remove chronic stress without regulation tools-impairs gut health and increases nutrient depletion (especially magnesium and zinc).

Nutritional Insights

1. Add oysters or shellfish (if non-vegetarian)-among the richest sources of zinc; support brain, immune, and skin health.
2. Add eggs -moderate zinc source + choline and lutein for neuroprotection.
3. Add mixed nuts (almonds, cashews, walnuts)-provide plant-based zinc, vitamin E, and omega-3s; lower neuroinflammation risk.
4. Remove high-sugar snacks and desserts-can increase inflammation and interfere with nutrient metabolism (including zinc).
5. Replace white pasta with whole grain or buckwheat noodles-increases zinc, fiber, and antioxidant intake; supports better metabolic and cognitive health.

Supplements To Consider

Zinc picolinate
(Please consult a healthcare professional or your physician before starting any supplements.)

Other Labs To Consider

Plasma Zinc

Explanation Video

https://vimeo.com/1107672774

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Methodology and Limitations Disclaimer

Testing for genetic variation/mutation on listed genes was performed using the Illumina Global Screening Array (DNA genotyping microarray). All genetic testing is performed by ExtendingME Inc, 750 William D Fitch Pkwy Ste 420, College Station, TX 77845. This test will not detect all the known alleles that result in altered or inactive tested genes. This test does not account for all individual variations in the individual tested. Test results do not rule out the possibility that this individual could be a carrier of other mutations/variations not detected by this gene mutation/variation panel. Rare mutations surrounding these alleles may also affect our detection of genetic variations. Thus, the interpretation is given as a probability. Therefore, this genetic information shall be interpreted in conjunction with other clinical findings and familial history. Patients should receive appropriate genetic counseling to explain the implications of these test results. The calculations and supplement recommendations presented in this report are not suitable for children under the age of 16. The analytical and performance characteristics of this laboratory developed test were determined by ExtendingME laboratory (Laboratory Director: James Jacobson, PhD) pursuant to Clinical Laboratory Improvement Amendments (CLIA) requirements (CLIA #: 10D2316806).

Medical Disclaimer

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