Review of the science of plasmalogens and Plasmalogen Replacement Therapy (PRT), including the favorable effects on longevity as well as on diseases of aging including cardiovascular disease, cancer, and neurodegenerative diseases (Alzheimer's disease, dementia, Parkinson's disease, Multiple Sclerosis)

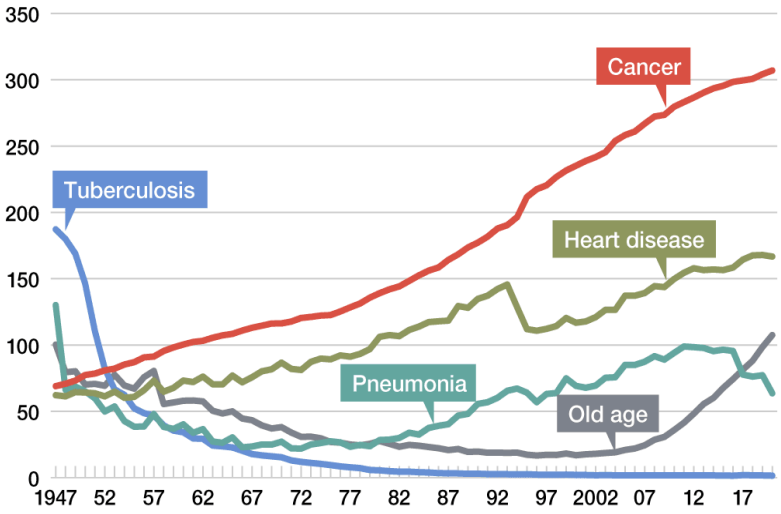
The purpose of this review is to familiarize the reader with the subject of plasmalogens and to assess the personal relevance of this treatment modality. I have attempted to simplify the biochemistry jargon to only what is truly relevant to understand its principles. I have included many citations to substantiate the ideas being represented and allow the reader to investigate the literature further if one so chooses. I also have included sections on various organ systems to be more inclusive for a broader audience. PRT is notably in its infancy, but it is a safe modality with robust supportive data and potentially dramatic benefits.

Adverse internal and external environmental changes cause disease. Therefore, strategic optimization of these internal and external biochemical dysfunctions is the key to promoting health and longevity. Identification and correction of cellular biochemical dysfunction in the pre-disease prodrome stage is the most efficient strategy of disease risk modification. Plasmalogen deficiency is a biochemical dysfunction that we can now detect using a blood test and correct with supplementation. The following pages describe the what, why, and how of plasmalogens.

Plasmalogens Effect on Longevity

As the following graph shows, the death rates of the most common ailments have increased over time. This increase cannot be primarily due to genetic influences since our genome isn't changing that quickly. However, our external environment (micronutrients in our food supply, environmental toxins, less optimized microbiome) and internal environment (pro-inflammatory states, mitochondrial and peroxisomal dysfunction, plasmalogen deficiency) can change rapidly, thus resulting in these changing rates of disease.

Death Rates by Cause



Created by *Nippon.com* based on data from the Ministry of Health, Labor, and Welfare's 2020 overview of monthly vital statistic reports. Numbers are approximate; heart disease does not include hypertension.

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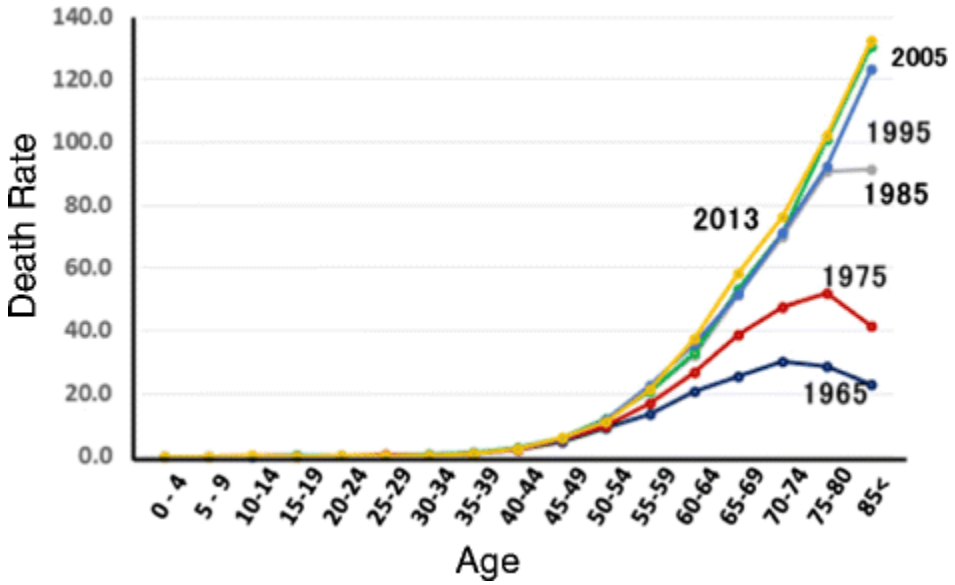
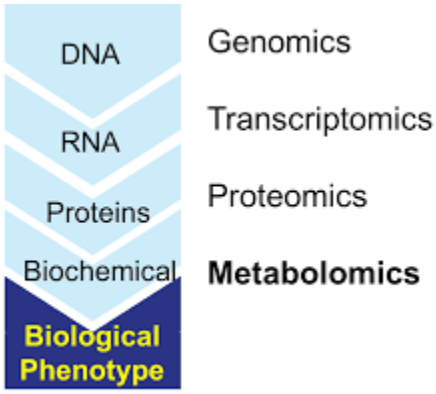
As the graph below shows, death rates are virtually nil until age 50 and then explode at about age 60. This curve segment represents an age-related loss of cellular function (health). This loss of function then ultimately culminates in premature death. Longevity or age reversal strategies attempt to slow or reverse aging at the cellular level to phase-shift or delay these diseases of aging.

The other trend the chart below demonstrates is that the death rate worsens over time, increasing cellular metabolic biochemical dysfunction. Many contributing factors include mTOR mediated aging, mitochondrial dysfunction with resultant dysfunctional cellular energetics, peroxisomal dysfunction with resulting plasmalogen deficiency, progressive insulin resistance, and consequent dyslipidemia, diabetes, and vascular disease. The incidence of metabolic syndrome (diabetes, hyperlipidemia, obesity, hypertension, visceral adiposity) is increasing dramatically and occurs at increasingly younger ages. Metabolic syndrome is due primarily to dietary changes: consumption of processed, low nutrient-dense, low fiber, simple carbohydrate-laden foods like liquid sugary beverages, and fructose (including high fructose corn syrup as an additive in processed foods) in particular, all of which lead to insulin resistance. The hyperinsulinemia, which is 100% anabolic, creates a vicious and worsening cycle of weight gain and comorbidities, shortening lifespans for the first time in US history.

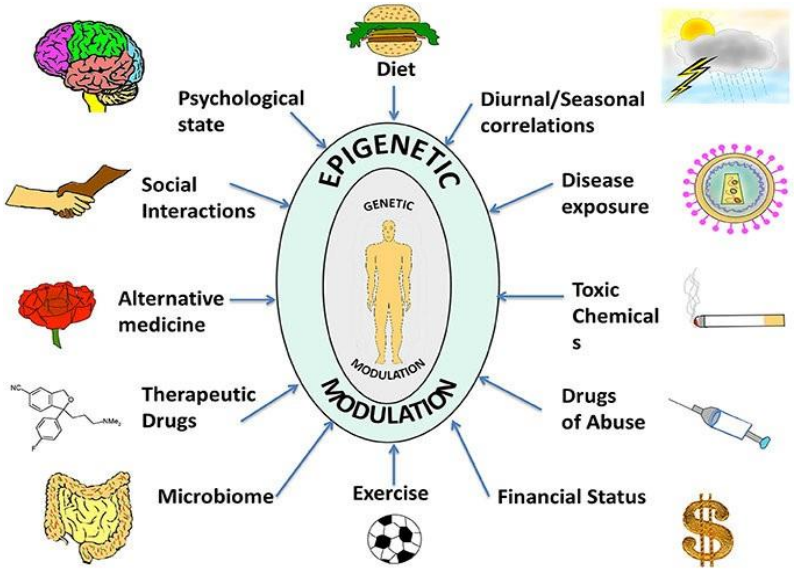


Disease is the eventual pathological manifestation of an uncorrected biochemical dysfunction, with death being the ultimate outcome if left uncorrected. The current medical practice focuses on treating the pathological manifestations of disease, which simply resets the death clock and creates chronic morbidity. Treating symptoms perpetuates illness in an attempt to delay death. Identifying and treating the underlying biochemical, cellular dysfunction in the pre-disease or prodrome stage perpetuates health and prevents disease, whether it be cancer, diabetes, heart disease, or dementia. Longevity or age reversal medicine focuses instead on optimizing cellular metabolism and maintaining a cellular milieu that inhibits disease genesis instead of treating disease states per se after they are allowed to germinate.

The process by which DNA is copied to RNA is called transcription. The process by which RNA is used to produce proteins is called translation. Our genome acts as a digital copy of our phenotypic potential in the form of DNA. It doesn't change as we age, so cloned animals are born young, not old. Our environment, however, has a significant impact on how the genome is translated through a process called epigenetics. Our lifestyle choices affect our cellular metabolism, which ultimately slows or accelerates the aging process by affecting which genes turn on or off.



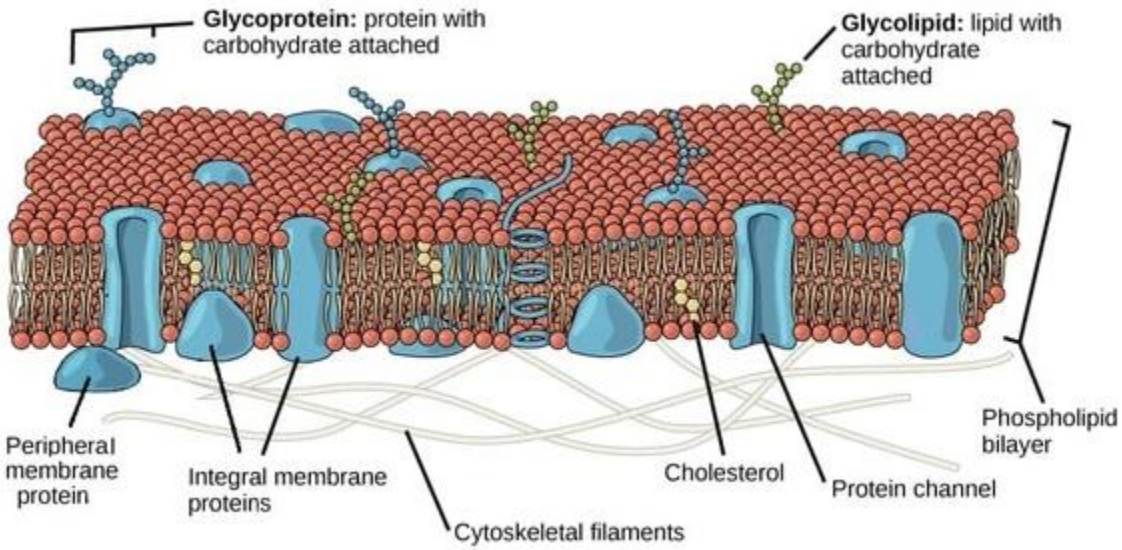
In addition, many environmental factors have an impact on which genes get turned on or off:



Epigenetic influences including but not limited to oxidative stress, diet, and exercise impact cellular energetics via mitochondrial and peroxisomal metabolism resulting in, for example, plasmalogen deficiency. In addition, epigenetic influences the turning on or off of so-called longevity genes, which in turn impact disease prodrome states.

The human body is comprised of trillions of individual cells. Each of these cells is compartmentalized into subcellular organelles. A biologic membrane separates all cells and subcellular organelles. All biologic membranes share a common physical structure - a phospholipid bilayer. All nutrients, waste materials, and metabolites pass between cells and organelles through these membranes, primarily via transport proteins embedded in the membrane. Most proteins are embedded in a membrane, and their function is affected by membrane composition.

The following cartoon depicts membrane anatomy:



All mammalian cell membranes contain a type of phospholipid called plasmalogens. The plasmalogens constitute about 20% of the phospholipids that populate mammalian cell membranes. In humans, levels rise until age 40, then progressively decrease after that, notably 40% lower by age 70. There are large percentages (90%) of plasmalogens in the membranes of certain parts of the brain cell components, including myelin, synaptic vesicles, nuclei, and endoplasmic reticulum. In addition to their structural role in membranes, plasmalogens are critical for membrane fusion activity essential for cell-to-cell communication, especially in the brain. Plasmalogens also play a significant anti-oxidant role, though they are irreversibly consumed in the process instead of other anti-oxidants that can be recharged to be used again. The half-life of plasmalogens is estimated to be 10-30 minutes in the neurons and 10-30 days in the myelin, and thus a continuous supply is required to maintain normal cellular function. Since plasmalogens have their highest concentrations in brain tissue, it is not surprising to see low levels correlated with many brain disorders either as a primary or secondary contributing pathologic mechanism of injury.

The term plasmalogen was coined in 1924. The only primary purely plasmalogen deficiency state identified to date is a genetic plasmalogen deficiency syndrome called Rhizomelic Chondrodysplasia Punctata (RCDP) seen in 1:100,000 births with a mortality of 50 % by age six and nearly 100 % fatal by teenage years. This condition documents the essential role of plasmalogens in organ development.

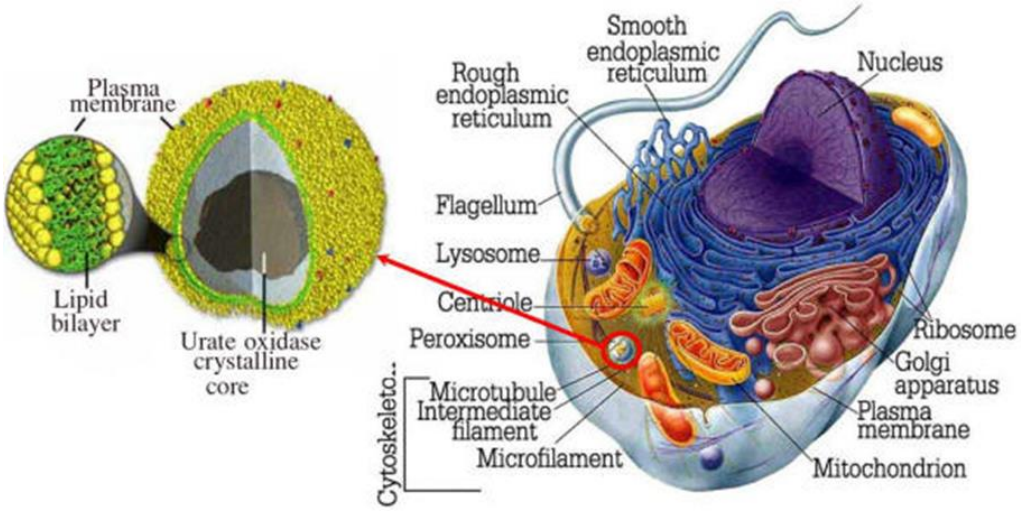
Plasmalogens are produced in a subcellular organelle called the peroxisome. Peroxisomal Biogenesis Disorder-Zellweger spectrum disorder (PBD-ZSD) is a group of phenotypic spectrum disorders reflecting various intensities of peroxisomal dysfunction on a genetic basis, including but not limited to plasmalogen deficiency.

Peroxisomal activity decreases with age. Therefore, in the absence of a genetic disorder, plasmalogens levels start to decline at age 40, accelerated by oxidative stress. Reduced brain plasmalogens levels induce microglia activation, a mediator of neuroinflammation (doi: 10.1016/j.neulet.2017.06.050 and doi: 3390/biomedicines9020216). This can be seen in Parkinson's Disease (PD) (doi: 3390/biomedicines9020216), Multiple Sclerosis (MS) (doi: 1016/j.neulet.2017.06.051) and atherosclerosis/Coronary artery disease(CAD) (doi: 10.1016/j.atherosclerosis.2016.01.003).

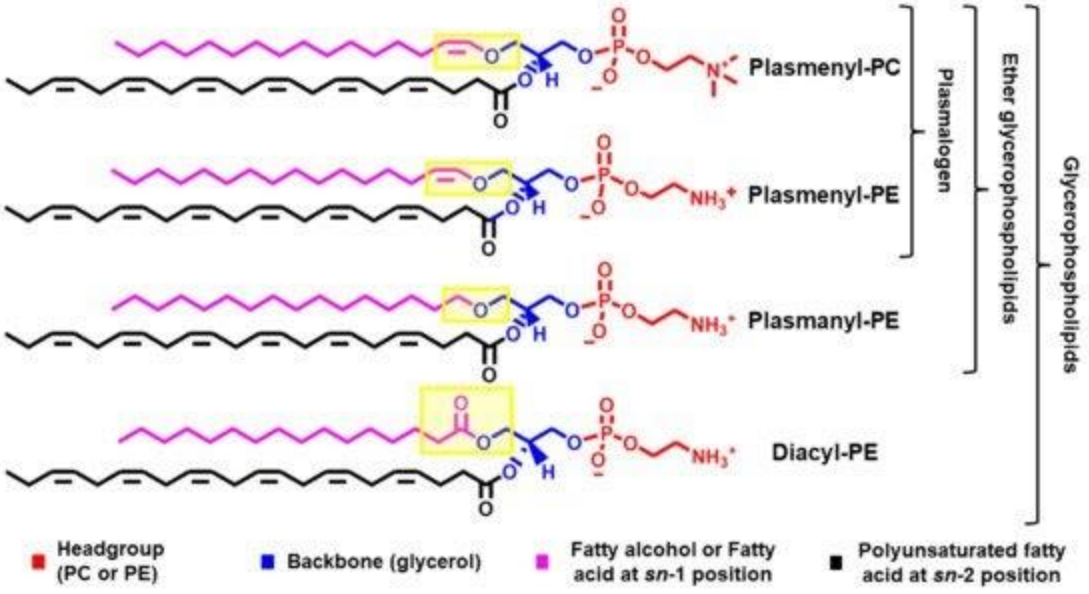
Microglial activation results in lipid peroxidation at the cellular membrane, a process inhibited by plasmalogens. Less microglial activation means less neuroinflammation, the common denominator for brain disease. Less neuroinflammation means less glutamate excitotoxicity, the mediator of neuroinflammation. Neuroinflammation, if unchecked, ultimately results in neuronal cell death, manifests clinically as Alzheimer's disease, Parkinson's disease, Multiple Sclerosis, ALS, etc. These are examples of secondary plasmalogen deficiency states, documenting the role of plasmalogens in maintaining tissue homeostasis.

Phospholipid plasmalogens are a surrogate marker of oxidative stress. Low levels are associated with higher neurodegeneration, as seen with dementia, higher cardiovascular morbidity, mortality, and cancer promotion. Lower plasmalogen levels imply a higher oxidative stress load.

Cell anatomy, including peroxisomes, are depicted below:



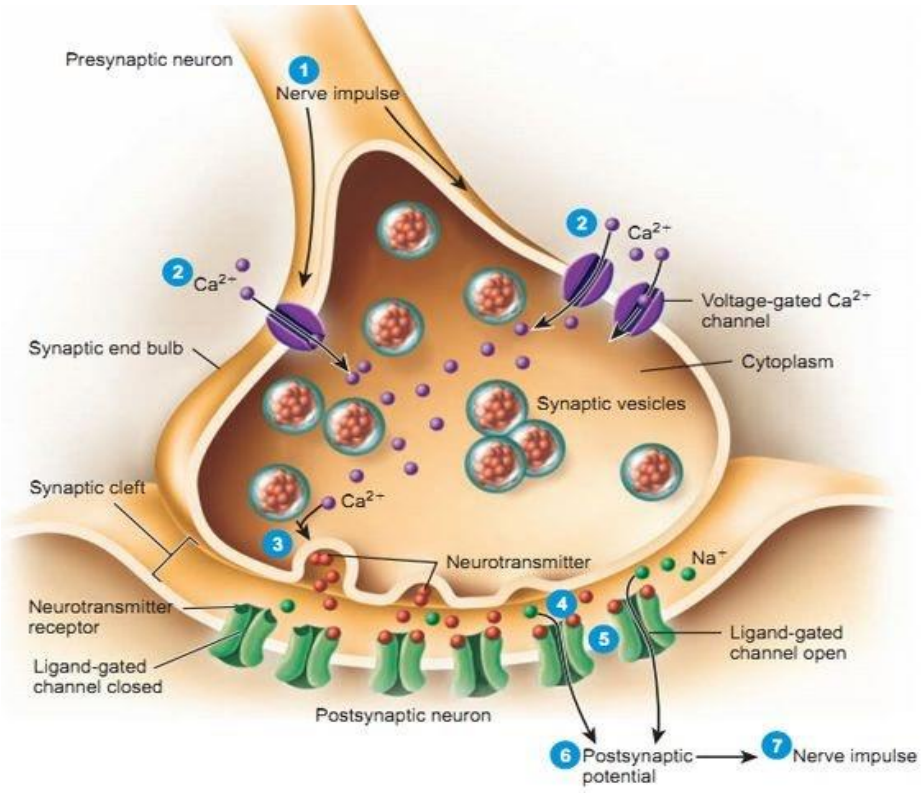
The molecular differences between plasmalogens and other membrane phospholipids appear minute to the non-biochemist, but the biological effects are enormous. Plasmalogens are a class of glycerophospholipids that have a fatty acid with a vinyl-ether bond at the sn-1 site, and a polyunsaturated fatty acid (PUFA) at the sn-2 site of the glycerol backbone as depicted in the following diagram:



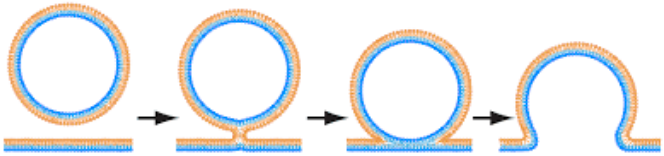
Plasmalogens in Dementia and the Neurochemistry of Cognition

Cognitive test scores within six months of death are directly correlated to decreased choline levels in the brain (Perry et al. 1978). This study confirms decreased cholinergic function as the primary cause of impaired cognition. Membrane fusion is required for neurotransmitter release (including choline) at the synapse between neurons (Glaser & Gross 1995). Membrane fusion, in turn, is dependent upon plasmalogen content in the membrane. Decreased brain plasmalogen levels have a strong association with reduced cognition.

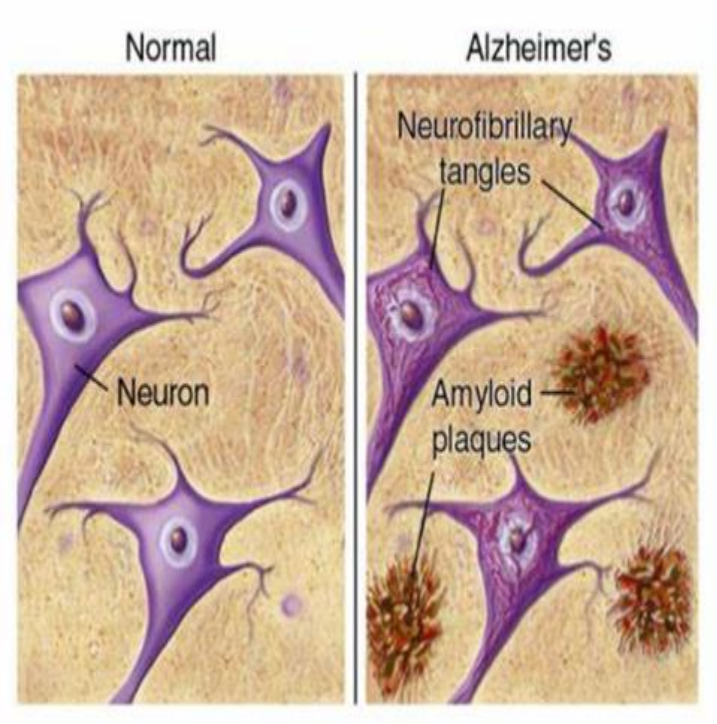
Cartoon of the synaptic cleft and mechanism of release of neurotransmitters including choline in the brain:



The cartoon depicts morphologic changes when a vesicle containing a neurotransmitter such as choline merges with the cell membrane to release it's content such as choline. This membrane fusion event requires adequate levels of plasmalogens to function optimally:

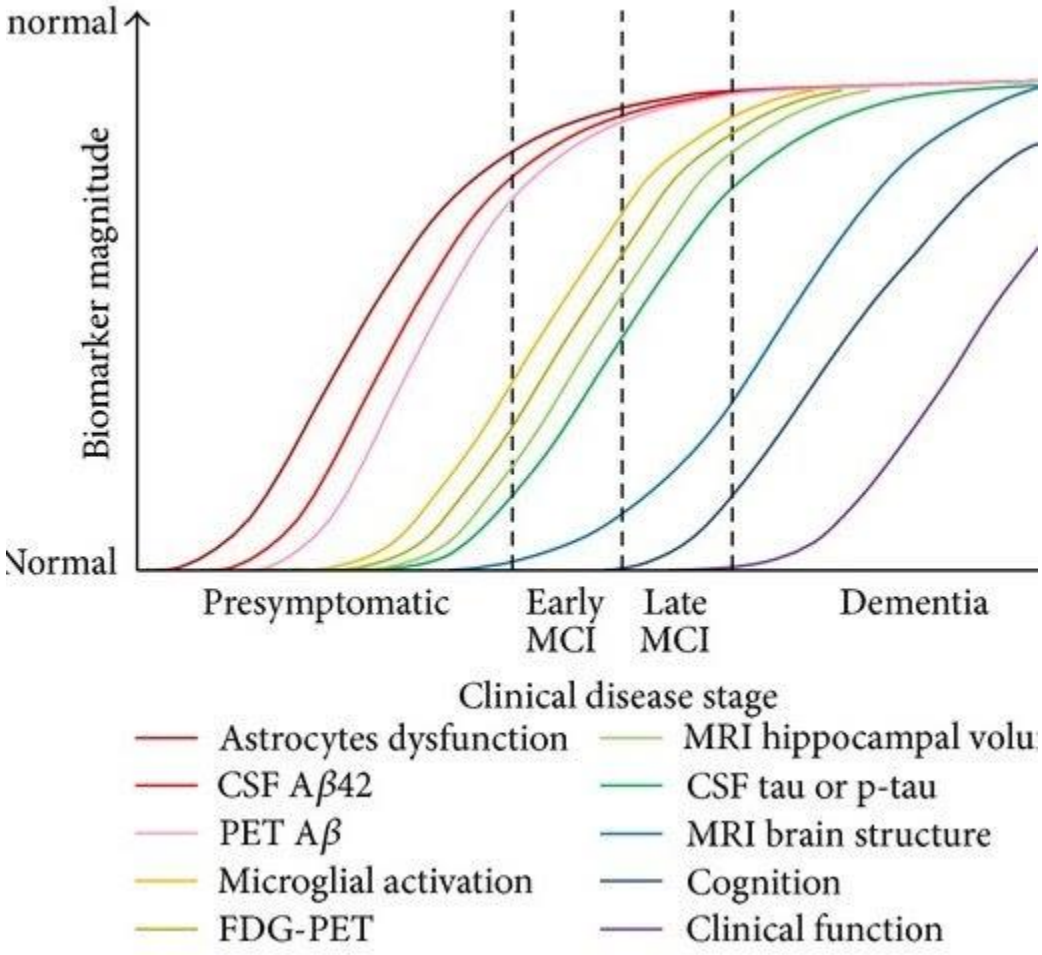


Different forms of neuropathology are used to categorize dementia "types." By age 95, 80% of people have extracellular amyloid plaques accumulating between neurons that contain AB42 protein, a hallmark of Alzheimer's Disease (AD) type pathology. These "Senile" plaques have correlated with brain choline levels (Perry et al., 1981). In the elderly, these amyloid plaques and associated intracellular neurofibrillary tangles containing tau protein appear together (Braak & Braak 1997). By age 80, everyone has neurofibrillary tangles in their brains (Braak & Braak 1997). Interestingly, 55% of cognitively normal people meet the neuropathological criteria for AD at the time of death (Bennett et al., 2005).

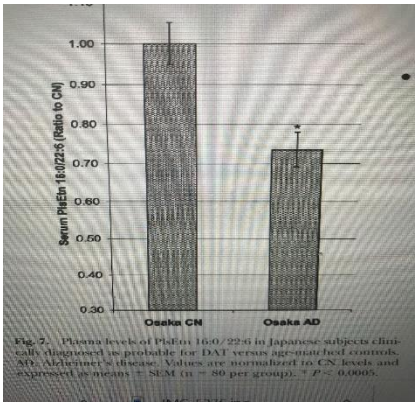


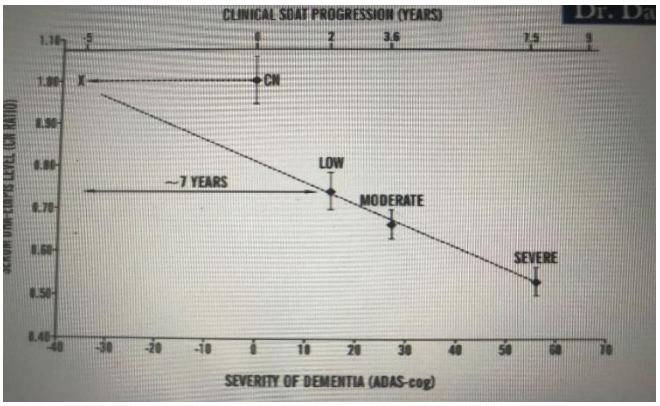
Therefore, neuropathology is not a biomarker for brain function nor the cause of reduced brain health. Cellular biochemical systems maintain a pathology-free brain, but the brain often becomes imbalanced with age. Reduced brain function (dementia), neurodegeneration, and the accumulation of neuropathology are all caused by the same biochemical dysfunction occurring before death.

The following graph depicts that the biochemical dysfunction significantly precedes even the earliest sign of cognitive dysfunction, i.e., minimal cognitive impairment (MCI). The clinical diagnosis of dementia is the last to appear. Astrocyte dysfunction (in brown) and microglial activation (in yellow) are both activated by plasmalogen deficiency. Clinicians evaluate patients when they become symptomatic in the MCI or dementia stage. By then, there is already brain atrophy due to neuronal loss and significant accumulation of amyloid plaques and neurofibrillary tangles. The prodromal biochemical cellular dysfunction has already been ongoing for years by this time.



In 2005, Dr. Goodenowe's non-targeted metabolomics technology discovered several biomarkers that were decreased in the blood of persons with dementia, which he subsequently identified as plasmalogens. Dementia patients have been found to have low blood plasmalogen levels. (Goodenowe et al J Lipid Res 2007;48:2485-2498). Plasmalogens deficiencies are more severe in white matter (40% reduced) vs grey matter (10% reduced in mild AD and 30 % in severe AD) (J Neurochem (2001) 77,1168-1180) and choline plasmalogens found to be 73% reduced in the prefrontal cortex of 10 patients with AD (J Alzheimers Dis 24(3):507-517 doi: 10.3233/JAD-2011-101608) and reduction in ethanolamine plasmalogens in the mid temporal cortex in AD (Res 698:1995: 223-226 doi: 10.1016/0006-8993(95)00931-F)





Since this initial discovery, pre-mortem blood plasmalogens levels have been documented to correlate with post-mortem brain plasmalogens levels, and both blood and brain plasmalogens have been documented to correlate with both cognition (MMSE) and Braak neuropathology severity (Wood et al 2011). Furthermore, the rate of dementia in persons with high plasmalogens was found to be 5 times lower than in persons with low plasmalogens (6% vs. 31%). In addition, low levels of plasmalogens exacerbate APOE4 mediated higher risk for developing dementia, and high levels negate this genetic APOE4 risk (Goodenowe and Senanayake Brain Sci 2019;9:92 doi:10.3390/brainsci9040092).

DHA-plasmalogen elevation have been found to dose dependently reduced levels of AB1-42 (the protein found in AD plaques) (Wood et al 2011). Brain membrane cholesterol is directly correlated with dementia risk (Cutler et al 2004). Plasmalogen deficiency increases membrane cholesterol levels. Increasing membrane plasmalogen levels increases membrane cholesterol efflux and thus lowers membrane cholesterol levels (Mankidy et al 2010) and thus lowers risk of developing dementia. Low brain plasmalogen levels are associated with decreased cognition.

Human Plasmalogen Replacement Therapy Studies

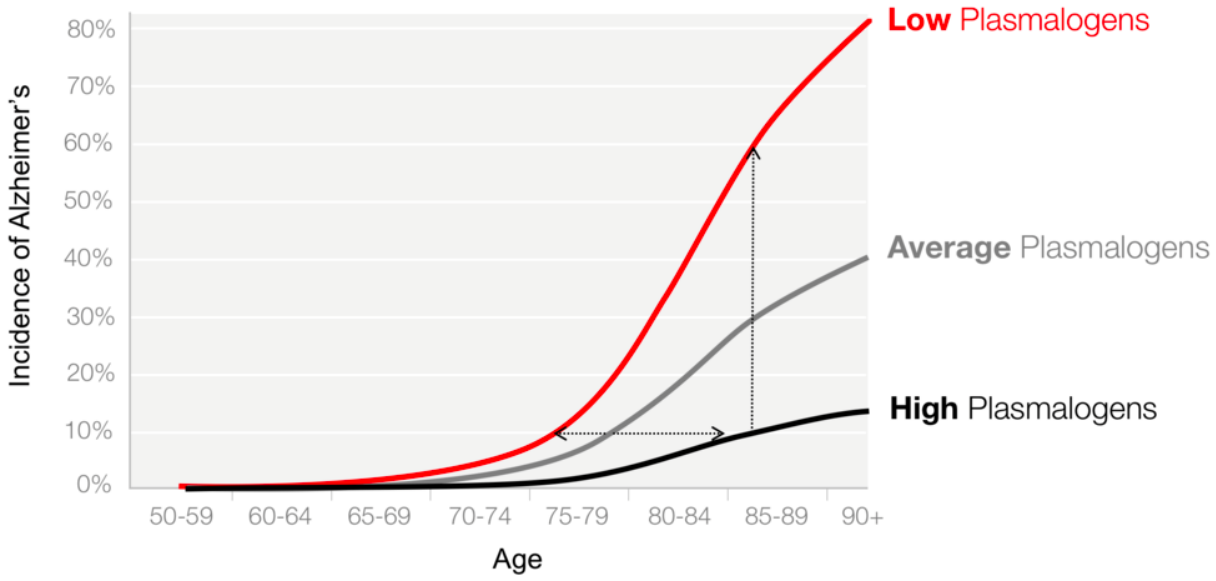
The first large human clinical study with 1659 subjects demonstrated low serum ethanolamine plasmalogens are associated with AD diagnosis, cognitive impairments, and levels of CSF tau. (Kling et al. DOI:10.1002/alz12110). A subsequent human study of 1112 subjects over age 60 showing that ether lipids and plasmalogens correlate with AD and AD risk factors (Lim et al J Alz Dis 76;2020:303-315 doi 10;3233/jad-191304). 2 studies of 696 controls with 268 AD patients in one, and 210 controls and 178 with AD in the other evaluated the peripheral lipidomes (detectable lipids in the blood). Out of all the lipids tested, only plasmalogens correlated with AD. In a study of 328 patients treated with 1 mg daily plasmalogens over 24 weeks, there was clinical improvement in mild AD symptoms but not in MCI patients (Ebiomed. 2017;17:199-205 doi: 10.1016/j.ebiom.2017.02.012). In another study, cognitive improvements were seen in 24 weeks in 178 patients with MCI, 98 patients with mild AD, and 10 patients with PD, as well as in 12 weeks in 57 patients with moderate AD, and 18 patients with severe AD (doi: 10.1007/978-3-030-60204-8_14).

Humans with high brain levels of plasmalogens have low brain levels of amyloid. Humans with APOE-4 genotype (23% of the US population have Apo E3/E4 and 2% have Apo E4/E4) have higher than normal brain amyloid levels. Humans with the APOE-4 genotype and high brain plasmalogens do **NOT** have higher than normal brain amyloid levels. In other words, adequate levels of brain plasmalogens **negate** the APOE4 genetic predisposition to AD (Goodenowe & Senanayake 2019). APOE E4 is associated with reduce cholesterol membrane efflux (Michikawa 2000). Low plasmalogen levels reduce cholesterol efflux (Mandel et al 2013). Plasmalogen supplementation restores cholesterol efflux and lowers membrane cholesterol levels (Mankidy 2010). Therefore, APOE e4 genotype is associated with amyloid – not cognition as its effect can be negated by adequate levels of brain plasmalogens.

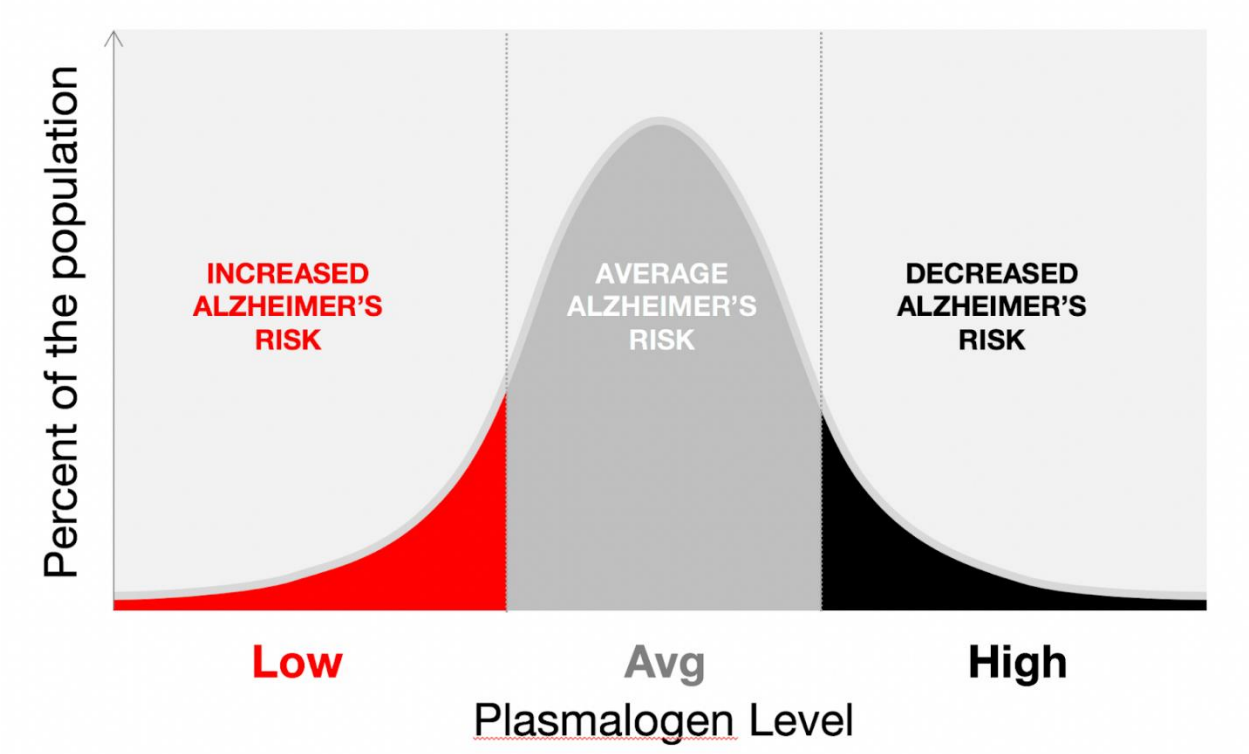
Therefore, in summary:

1. **Multiple international human biomarker studies independently confirm that low blood plasmalogen levels are associated with an increased risk of dementia.**
2. **Low blood plasmalogens predict both prevalence and incidence of dementia.**
3. **Blood plasmalogen levels correlate with brain plasmalogen levels.**
4. **High blood plasmalogens neutralize the APOE-4 genotype expression.**
5. **The association between plasmalogens and cognition is independent of traditional brain AD pathology.**
6. **Increasing membrane plasmalogens in laboratory cell culture experiments dose-dependently decreases the formation of beta-amyloid.**
7. **Humans with high brain levels of plasmalogens have low brain levels of amyloid.**
8. **Humans with the APOE e4 genotype have higher than normal brain amyloid levels.**
9. **Humans with APOE e4 genotype and high brain plasmalogens do NOT have higher than normal brain amyloid levels.**
10. **APOE e4 genotype is associated with amyloid – not cognition.**

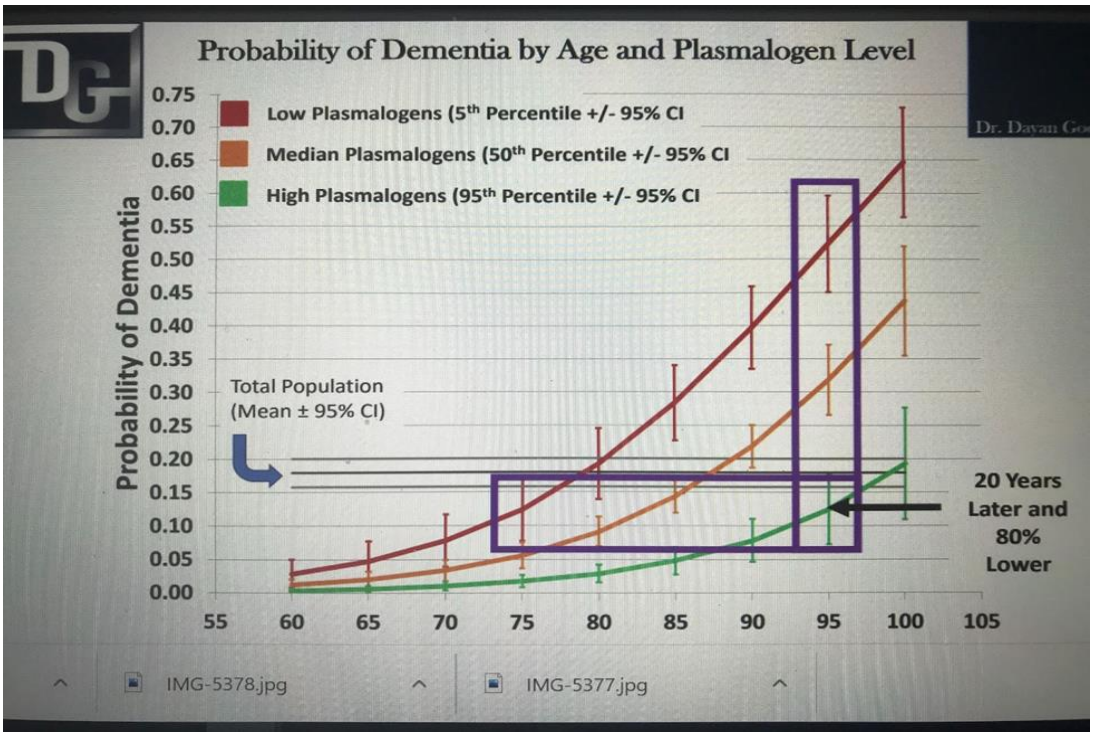
The following graphs depict the relationship between Alzheimer's disease and plasmalogen serum levels as assessed in the prodrome scan blood test. As confirmed in human studies, the serum plasmalogen levels correlates directly with the brain plasmalogen levels.



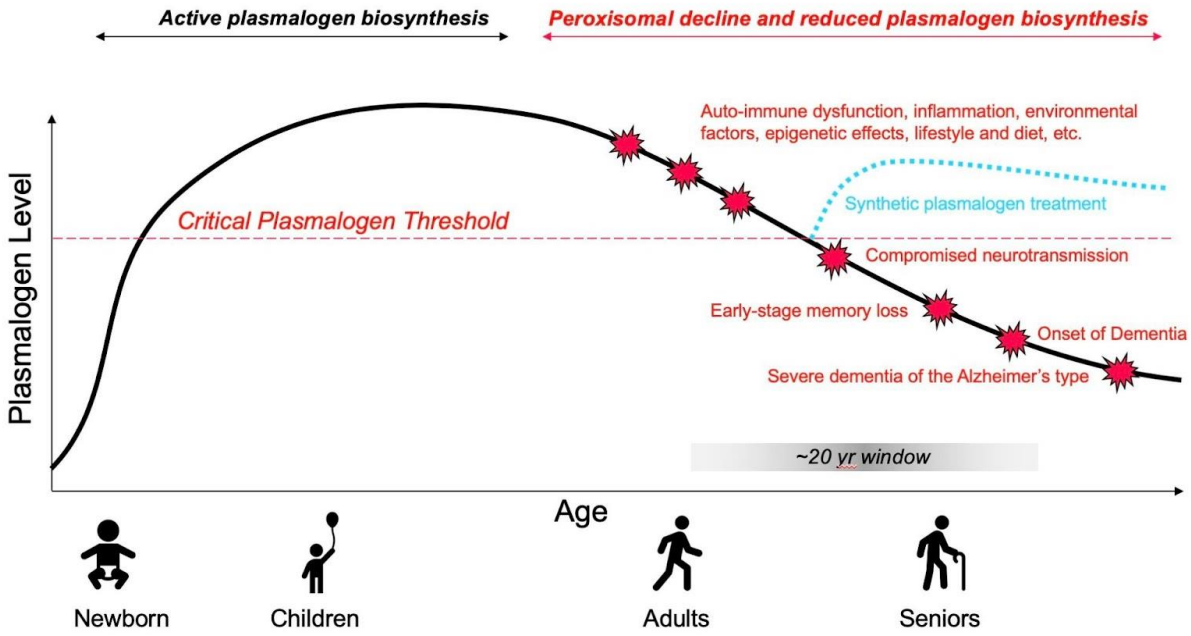
Or depicted differently:



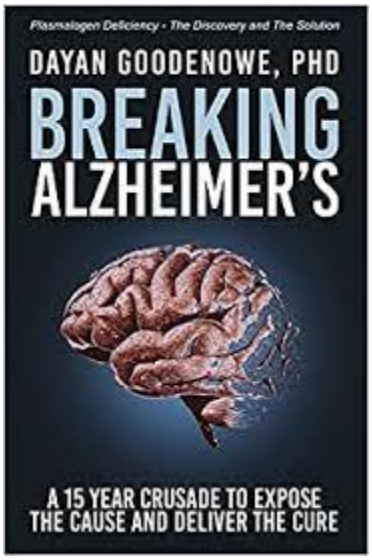
The following graph illustrates that the highest plasmalogen level group has an 80% less dementia risk at age 95 compared to the lowest plasmalogen level group which equates to the same relative risk for a 20 year younger 75 year old in the low plasmalogen group.



The following graph depicts the intended purpose and effect of plasmalogen augmentation:



Dr. Goodenowe's book on this subject is available:



Plasmalogens in Parkinson's Disease

Parkinson's Disease (PD) is a chronic neurodegenerative disease that affects dopaminergic neurons in the substantial nigra in the brainstem. Cardinal symptoms include resting tremor, slowness to move (bradykinesia) stiffness (cogwheel rigidity) on exam, and shuffling gait. It is treated with a dopamine precursor called L-DOPA which enhances the functioning of the remaining dopamine neurons to compensate for the degenerated neurons. Over time, as the remaining neurons continue to degenerate, L-DOPA starts to loss it's effectiveness. In addition, long term L-DOPA therapy creates side effects including drug induced dyskinesias or involuntary abnormal choreaform movements.

The cause of most cases of PD is unknown but MPTP neurotoxin exposure is one experimental model used to study PD. Blood plasmalogen levels are notably low in persons with PD. Plasmalogen supplementation prevents (Brain Res. 2017 Nov 1;1674:70-76. Doi 10.1016/j.brainres.2017.08.120.) and prevents and rescued dopaminergic neurodegeneration in MPTP treated mice (Brain Res 2019 Dec 15;1725:146460. Doi: 10.1016/j.brainres.2019.146460.). In addition, Plasmalogen supplementation reduces and prevents L-DOPA induced dyskinesias in PD monkeys (Behav Brain Res 2018 Jan 30;337:183-185. Doi: 10.1016/j.bbr.2017.09.023) after only 2 days of treatment (Gregoire et al. Beh Brain Res 286(2015)328-337). 200 mg/kg of oral plasmalogens will increase serum and brain plasmologens by 200 % in rabbits (Wood et al Lipids in Health and Disease 2011,10:227. http://www.lipidworld.com/content/10/1/227

Neuroinflammation and associated oxidative stress has been determined to be a component of PD. (Transl Neurodeg 2015;4:p.19. doi:10.1186/s40035-015-0042-0). Plasmalogens are thought to protect cells from oxidative stress. (Curr Med Chem 2009:16(16):2021-2041 doi:2174/092986709788682164) and (Free Rad Bio Med 2015;84:296-310 doi:10.1016/j.freeradbiomed.2015.03.012) Low plasmalogens levels are indicative of oxidative stress which irreversably consumes plasmalogens to conteract the stress and associated superoxide radicals. (Clin Chem Lab Med 2009;47(7):894-897 doi: 10.1515/cccm.2009.205)

A human clinical study with 14 patients, 7 with deep brain stimulators (DBS) using 1 mg daily of plasmalogens for 24 weeks, showed improvement in nonmotor symptoms of PD along with normalization of blood plasmalogen levels. (Doi: 10.1155/2020/2671070). Recent study with 10 PD patients showed improvement in nonmotor PD symptoms (doi: 10.1155/2020/2671070)

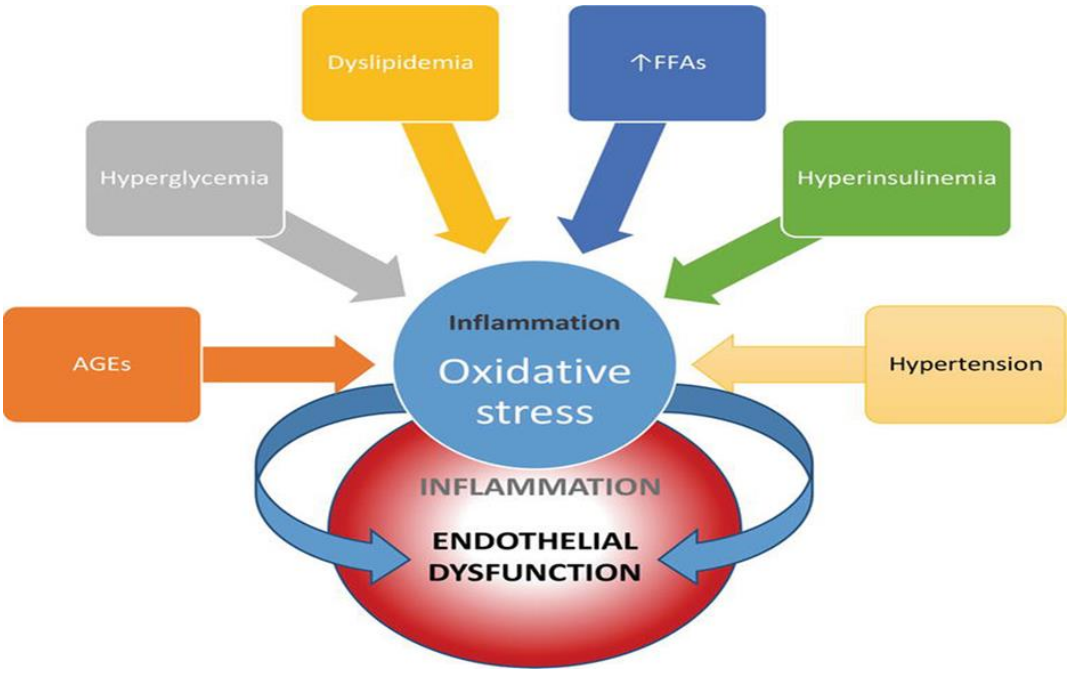
Plasmalogens in MS

One experimental model for MS is Cuprizone, a copper chelator that targets many metalloenzymes such as ceruloplasmin, impairs the activity of the copper dependent cytochrome oxidase, decreases oxidative phosphorylation and produces degenerative changes in oligodendrocytes resulting in demyelination, the halmark feature of MS. DHA plasmalogens prevent demyelination when used prophylactically in the mouse cuprizone model and induced remyelination when given post cuprizone exposure (Wood et al. 2011). A phospholipidomic signature has been discovered in MS patients. (doi.org/10.1016/j.abb.2020.108672

Plasmalogens in Cardiovascular disease (CVD)

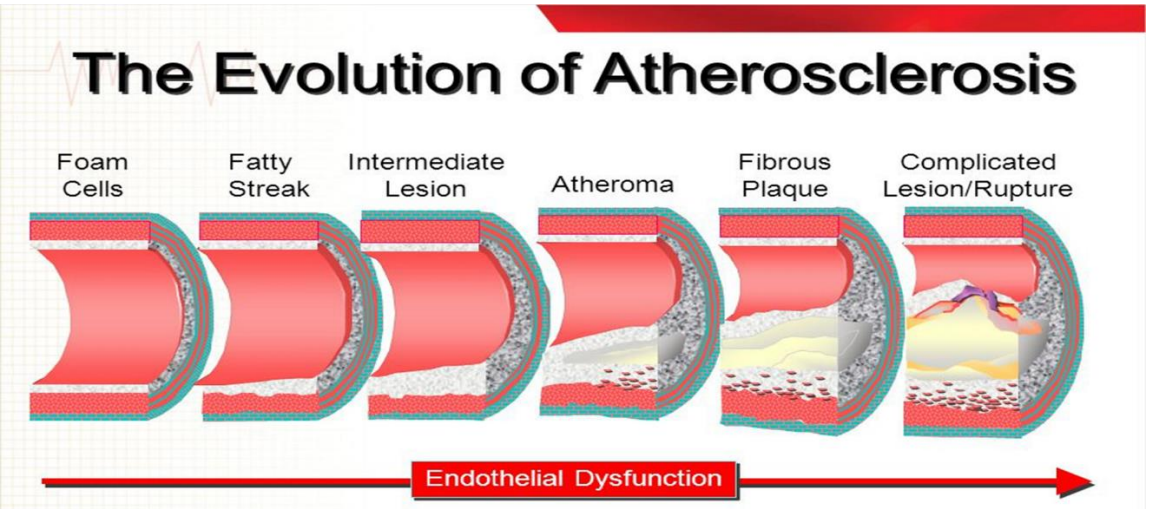
Inflammation is the key component of both the initiation and the propagation of the atherosclerotic pathologic cascade. Impaired intracellular energy, and lipid metabolism and transport, are the key underlying aspects of inflammation. There are many paths that lead to the same outcome, but the solutions are convergent. Reduced blood flow and reduced membrane function lead to varying levels of hypoxia. Hypoxia is pro-inflammatory and cytotoxic. Neuroprotection is more a function of targeting and supporting cellular maintenance functions than blocking neurotoxic mechanisms. These are the cycles that need to be reversed if present. Maintaining healthy membrane function including adequate plasmalogen levels is preventative of atherosclerosis.

Numerous pro-inflammatory factors induce oxidative stress resulting in endothelial dysfunction as depicted in the following diagram, including insulin resistance and associated hyperinsulinemia, hyperglycemia, hyperlipidemia, advanced glycation end-products (AGEs) and hypertension.

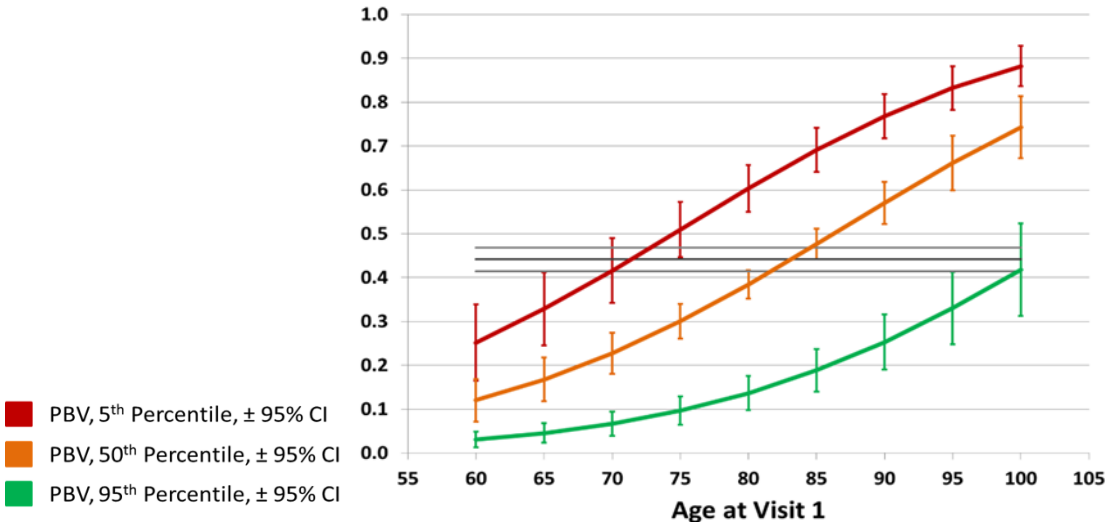


Endothelial dysfunction initiates the atherosclerotic cascade in which LDL enters the arterial wall, gets oxidized, then absorbed by activated macrophages creating foam cells that, over time, create arterial plaques, as illustrated in the following cartoon. Low endothelial nitric oxide levels and low plasmalogen levels accelerate atherosclerotic changes.

This process results in the development of arteriosclerotic plaque, narrowing of the vascular lumen, and ultimately vascular events such as strokes, heart attacks, and peripheral arterial disease.



The following graph depicts the probability of dying in 5.3 years relative to the percentile of plasmalogen levels:

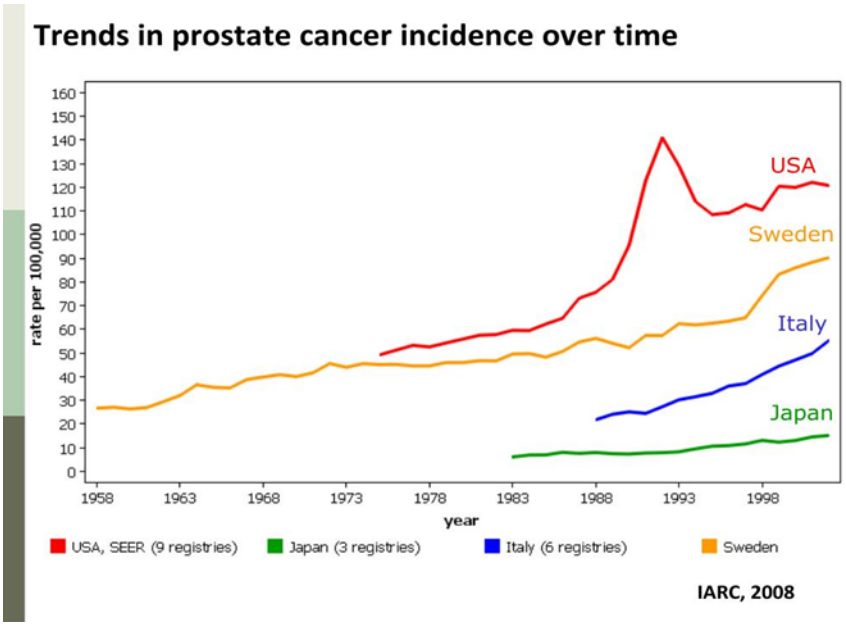


The 95th percentile group of plasmalogens has the same mortality risk as does the fifth percentile group 30 years younger and 50% lower than age-matched controls.

In other words, longevity is directly correlated with plasmalogen levels.

Plasmalogens and cancer

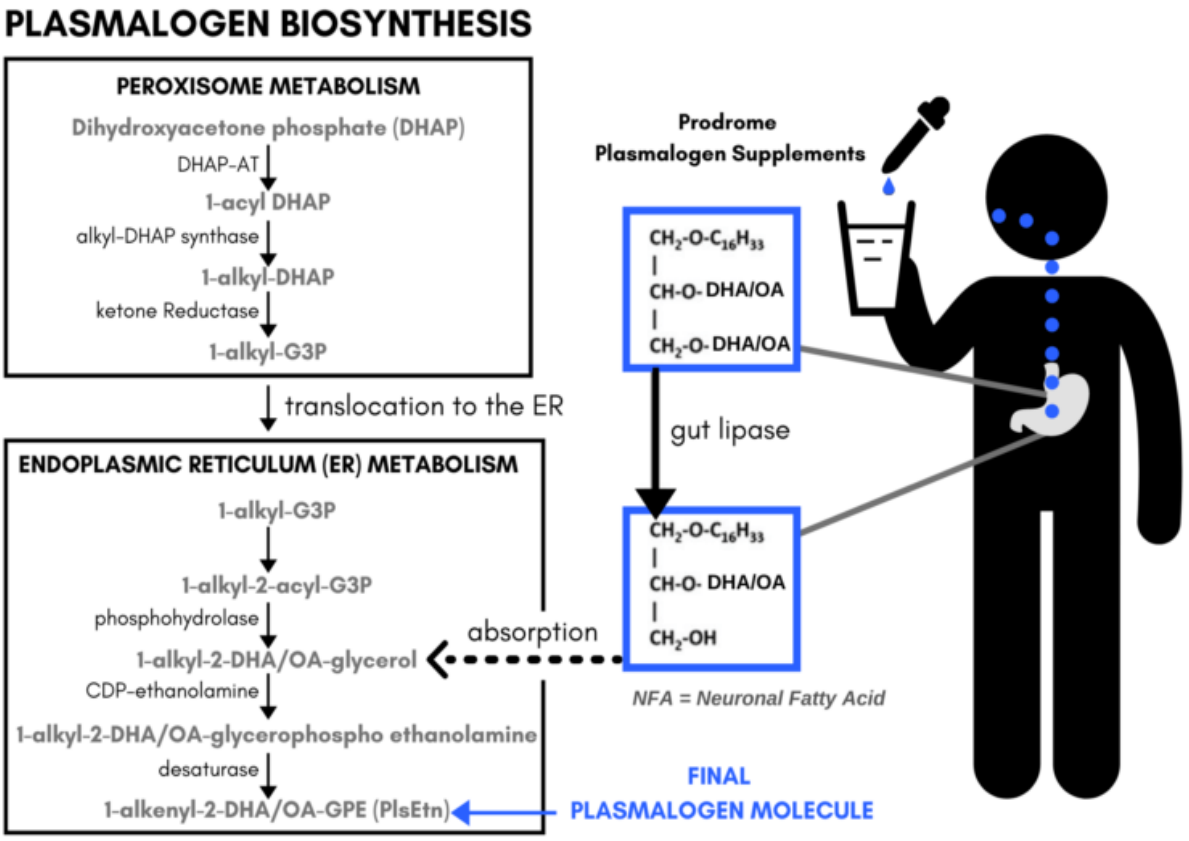
The following graph depicts the increase in incidence of prostate cancer over time. It is rather typical for cancers in general. Low plasmalogen levels have been found in virtually all cancer groups in humans. Treating the cancer whether with surgical excision or chemotherapy or radiation therapy even to the point of being curative for that particular cancer, does not change the plasmalogen deficiency. This deficiency is the prodromal condition that creates a precancerous state and uncorrected will foster development of additional cancers.



Plasmalogen Replacement Therapy (PRT)

Plasmalogens are found naturally in marine and land animals in low concentrations. In addition, bioavailability, when taken orally, is markedly reduced due to cleavage of the sn3 position and resultant deactivation in the acidic medium of the stomach and intestinal mucosa.

The Prodrome Science plasmalogen supplement bypasses the peroxisomal dysfunction that caused the low plasmalogen levels by strategically negating this gastric effect, allowing absorption of a bioactive product. The supplement provides a precursor directly to the cellular endoplasmatic reticulum to complete the plasmalogen synthesis despite persistent peroxisomal dysfunction.



Orally available synthetic plasmalogens have been documented to circumvent bioavailability issues and provide PRT clinical options:

- (Dis Model Mech. 2020 Jan 24;13(1)dmm 042499 doi: 10.1242/dmm.042499 and
- Lipids Health Dis 2011 Dec 5;10:227 doi: 10.1186/1476-511X-10.227).

PRT's safety has been well established (Das AK, Lipids 1992 Jun 27(6):401-5).

Latest review articles published on PRT:

- (Membranes(Basel) 2021 Nov.11(11):838 doi: 10.3390/membranes11110838) and
- (Escriba PV, Prog Lipid Res 2015 Jul;59: 38-53 doi: 10.1016/j.plipres.2015.04.003)

Why would you want to measure your blood plasmalogen levels?

1. To assess overall mortality risk.

2. To assess the modifiable risk of dementia despite genetic predisposition.

3. To assess the modifiable risk of a brain disorder, including neurodegenerative (Alzheimer's disease, Multiple Sclerosis, Amyotrophic Lateral Sclerosis (ALS), Parkinson's Disease).

4. To assess the modifiable risk of cardiovascular disease, including dyslipidemia, atherosclerosis, coronary artery disease, heart disease.

5. To provide a scientifically based, modifiable, quantifiable corrective action plan to optimize the above issues risk-free.

Now that you have a better understanding of the issue, and should you elect to pursue PRT either:

- to proactively promote longevity or minimize the risk of dementia, or
- as an attempt to address an ongoing medical condition such as AD, PD, CVD, MS, etc.,

Follow these steps:

1. To assess your baseline plasmalogen levels, arrange a ProdromeScan blood test directly from Prodrome sciences (Prodrome.com). If you have additional questions, review their FAQ at https://prodrome.com/frequently-asked-questions-%e2%80%8b/
2. Establish an account with them via this link and use the Colorado Ageless Institute practitioner access code CFORTIN25. By using our code, you will receive a 25% discount on the blood test: https://prodrome.com/patient-registration/
3. If not already a Colorado Ageless Institute patient, contact our office at ColoradoAgeless@protonmail.com to arrange a consultation. The ProdromeScan blood test is available for purchase only via certified healthcare professionals. Dr. Fortin will include baseline biomarkers to quantify therapeutic progress serially, interpret ProdromeScan results and engineer a personalized therapeutic strategy.
4. If appropriate for brain conditions, our office can arrange a baseline cognitive assessment and a P300 (brain speed) test.
5. Purchase plasmalogens supplements if recommended. Use discount code CFORTIN25 at checkout to save 25% on your purchase. Click here to shop: https://prodrome.com/store/
6. Reassess clinically in 3 & 6 months. We will schedule your follow-ups with Dr. Fortin.
7. Arrange a follow-up prodrome scan test to confirm the normalization of levels.
8. Arrange a follow-up cognitive assessment and P300 to confirm interval changes.

For this and any other related treatment modality, contact us at ColoradoAgeless@protonmail.com.

Prodrome Scan:

- The scan is a multisystem blood test to assist in achieving and maintaining optimal biochemical health.
- Measures 100 biomarkers organized into specific biosystems known to be associated with optimal health and longevity.
- The scan evaluates biosystem reserve capacity and biosystem function.
- Each assessed biosystem measured is modifiable using targeted lifestyle, nutraceutical, and diet-based strategies
- Allows one to objectively measure, track, and optimize the performance of each targeted intervention strategy.
- Prodrome scan demystifies one's biochemistry, allowing one to leverage this new knowledge to optimize health and longevity in addition to reducing the risk of age-dependent disease.
- Every biosystem identified and described in the Prodrome scan can be optimized by targeted environmental interventions, including diet, lifestyle, and supplements.

REPORT SAMPLE: