MEMORY REPAIR PROTOCOL

Science Backed Method For The Treatment And Prevention of Alzheimer's And Dementia

MARTIN REILLY

Welcome to the Memory Repair Protocol!

By reading this book, you've taken the first step toward improving your memory naturally and healthily. Through science – based advice, you will learn how to retain and optimize your memory, at any age.

Are you ready?

You are about to start a life-changing journey that will activate your brain, focus your thoughts and lower your risk for degenerative brain disorders.

That's right, your decision to fine tune your memory – the healthy way- will do much more than give you an advantage at work. It will also help you maintain brain health well into old age.

I've already done the first step for you.

I've reviewed and considered countless research studies, looking for practical answers to the problem many of us are facing today. I knew we needed a way to slow the progress of degenerative brain disorders like Alzheimer's and Dementia.

My goal was finding the easiest path for maintaining memory, and I share what I have learned in my research in this book.

But, it's all about you.

While I am excited about the information I've learned, ultimately the success of this system is up to you.

I'll provide the tools, but in the end, it's you that will supply the action. Through your hard work and heart, you can rewire your brain to its optimal level.

It's something worth being excited about.

Most people are not willing to make the necessary changes in their lives to reach their goals. By seeking this information and choosing this book, you are already on your way to extending the function of your brain.

Think about it: you aren't wishing for better memory, you are working toward better memory. You have taken charge, and for that, I salute you.

Where do you want to go from here? The keys are literally in your hands.

In 21 days, your brain will not be the same.

What about your loved ones? Have they begun slipping away to Alzheimer's and Dementia?

Now is the time to slow or reverse their symptoms, with science-based natural therapies. You don't have to lose them. This book can show you how.

Remember those moments the two of you have shared? It doesn't have to end. Support your loved ones' brain health with proven natural solutions.

You can do this – and this book is here to help.

To get the most from the Memory Repair Protocol, start by reading the introduction. It provides background into why many doctors misinterpret declining brain health as aging.

The truth is, you do not have to suffer from poor memory, focus or concentration – regardless of your age.

The first chapter is an in-depth overview of brain degeneration. It explains how the brain ages, and why our memory and attention might start to fade.

The next two chapters discuss Alzheimer's and Dementia in-depth. If you are not currently suffering from these conditions, and are only looking to optimize your brain function, you can skim them.

However, keep in mind that the Memory Repair Protocol can also reduce your risk of developing these conditions, so understanding what they are and how they form is highly useful.

In chapter four, I explain why current Alzheimer's and Dementia treatments do not work. There's also an appendix of FDA-approved Alzheimer's medications for you to reference at the end.

It's not just a book on Alzheimer's and Dementia, however.

Chapter five reveals the natural foods you can use to heal the brain, reduce brain degeneration and improve memory and concentration.

I then discuss simple lifestyle changes that can be made at any age to regenerate the brain and improve function. When combined, these two things alone can stop the progressions of Alzheimer's and possibly reverse it.

And don't forget to check out the Memory Repair Protocol in Part II.

While it may look like a diet, it's not. It's a protocol for healthy living that is specifically designed to improve brain function at any age. I've included all of the recipes as well.

You can heal your brain, and the protocol will show you how.

Today is the first day of a new you, part of a larger process to overall health.
Through the science -based natural therapies that I will teach you in this book, you will learn how to enjoy optimal brain function well into your senior years.

As you begin the Memory Repair Protocol, remember, this is about you. Making mistakes is okay. It's okay to go off of the plan occasionally. You are ultimately in control of your recovery.

If you are doing this on behalf of a loved one, don't lose hope. It takes time for the brain to heal, but it can heal.

Everyone deserves the chance to enjoy healthy brain function at any age. Don't let society let you believe otherwise.

Today we begin healing. Let's get started.

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INTRODUCTION

WHAT IS 'NORMAL' AGING?

For decades, science has attempted to understand and differentiate between 'normal aging' and 'abnormal aging.' Specifically, it wanted to define precisely which changes in brain function are a part of natural healthy aging and which are abnormal pathological changes. Presently, mainstream scientific models view Dementia and Alzheimer's disease as natural side effects of the aging process. In fact, the most common view globally (scientific or not) is that aging - the process of getting older - naturally causes changes in the brain and central nervous system that lead to conditions such as memory loss and dementia.

These perceptions of aging leave our elders (and us) with little to look forward to later in life. It's not that surprising that depression statistics among the elderly are high when most people expect aging to cause irreversible brain damage. They believe this brain damage will initially lead to forgetfulness and erratic, odd behavior. Then, it will inexorably strip your very identity away from you just before you die an idiot's death without any dignity.

There is a general bias towards youth on our planet, with the aged being looked down upon and in some cases neglected for their 'deteriorations'. The emphasis on youth is apparent. Young people appear in most advertisements and are preferred employees because the elderly are not 'capable' after retiring age. At the same time, the retiring age itself is getting younger and younger each generation. Old people in modern '1st world' affluent western countries are increasingly put into 'retirement communities – a sort of humane and sanitized prison for the elderly. The global community has seemingly 'pathologized' aging, and the subject is...'dirty,' 'frightening' and awkward – it is one of the greatest modern taboos of our time.

It appears that there is a fine line when it comes to distinguishing normal age-related changes from pathological changes. With the global view on aging being what it is, there is an urgent need, now more than ever, to challenge long-held assumptions about age and health. We need to make sure that we have the story correct and that our assumptions are not leading us further down an inhumane youth-obsessed future path.

One of the aims of this book is to question the established popular view of aging and the concepts of 'natural' 'inevitable decline.' We also hope to clearly present the full story of aging in terms of the brain, along with the latest scientific findings, so that you, the reader may be better informed overall. This book explains why degeneration is so prevalent and, more importantly, what YOU can do about it.

Over the last decade, an increasing amount of evidence from new research offers hope for our beloved elders. It seems to show that brain degeneration is neither inevitable nor a natural outcome of aging.

So, does this mean that the 'mainstream' scientific view of aging, dementia, and Alzheimer's disease is flawed?

What we know about brain function has only gathered serious momentum in the last three decades, especially around the physical factors affecting old age. In fact, when it comes to brain degeneration, we are still mostly in the dark about its underlying causes. Brain functioning is still not completely understood, and new research is constantly creating revisions or refutations in our old hypotheses and cherished dogma. Currently, any explanation into what's really going on is increasingly difficult to justify.

Recent imaging technologies have opened up the brain to researchers, and for the first time, the brain is able to be studied 'in situ' as a living system. We are able to see more of how it functions and in particular, we are able to see to what extent lifestyle, environmental factors, and nutrition contribute to rapid bodily decline in aging.

The last decade has also brought an explosion of genetic information to the table that challenges our beliefs, attitudes and even definitions of aging. Researchers are now finding scientific evidence that certain factors can enhance brain function and optimize cognitive functions in the youth and aged alike.
What has been clearly demonstrated is that informed diets, lifestyle changes, and environmental factors have an impact on our entire bodies throughout our lives, with the brain being no exception. The point is well illustrated by the fact that neurology students from the 1990s were saying, "chronic lifestyle disease may soon become an archaic relic associated with ignorant assumptions made by twentieth-century scientists."

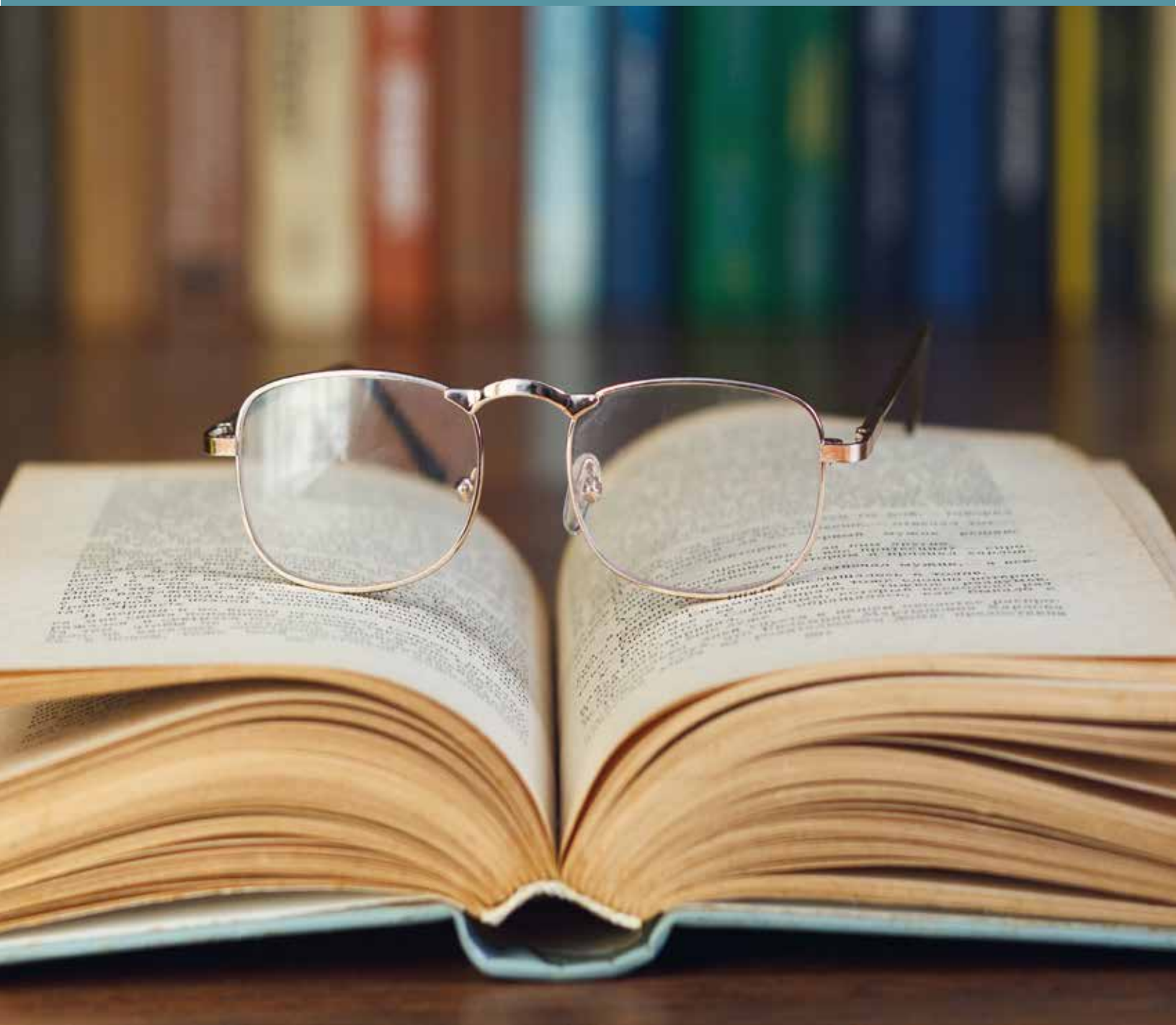
A new understanding is emerging. What we once thought was 'destined,' 'unavoidable' or 'inevitable' may, in fact, be unnatural or even unnecessary. These discoveries are beginning to form a new health paradigm, one that centers on lifestyle and dietary changes for sustained all-around health and longevity - A model that seeks to auspiciously redesign how we relate to and treat the elderly amongst us.

In line with this new paradigm, we've developed a pharmaceutical-free diet and lifestyle plan that is complete with recipes and advice based on the latest scientific research findings. It is created to prevent, halt, or combat degenerating conditions such as Alzheimer's disease or dementia that commonly arise for people as they age.

This book aims to support an affirming and elderly-friendly model by giving you the facts. It will also equip you with powerful lifestyle and dietary tools to combat what seems to be an unnatural deterioration of the brain and its functioning as we age.

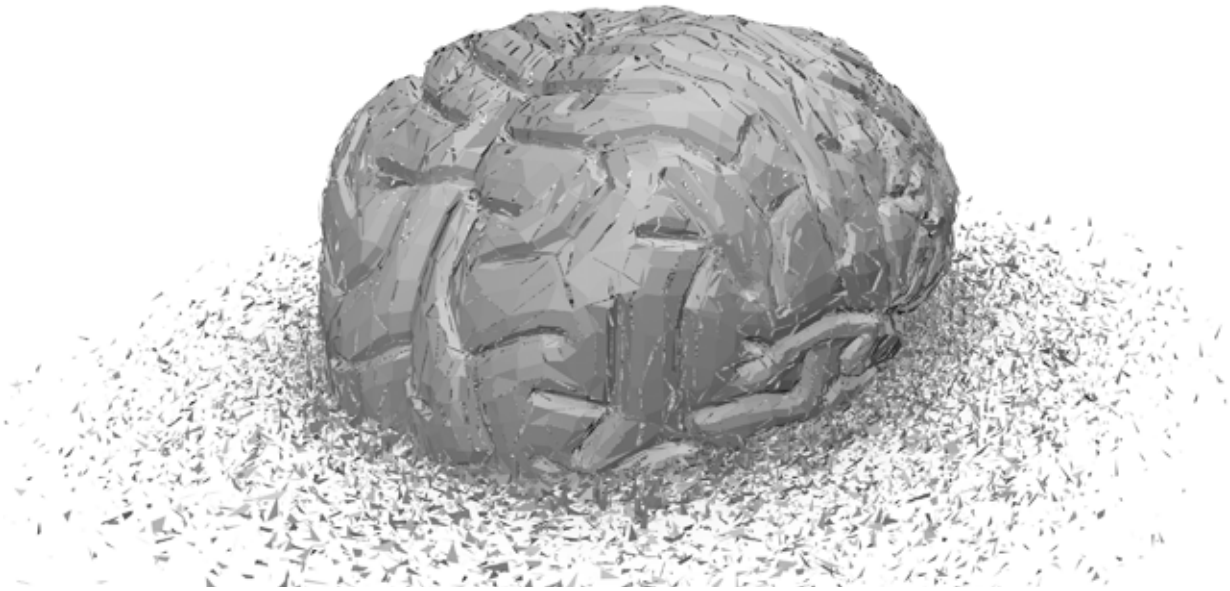
PART 1

THE SCIENCE BEHIND MEMORY LOSS



CHAPTER 1

BRAIN DEGENERATION – WHAT IS IT?



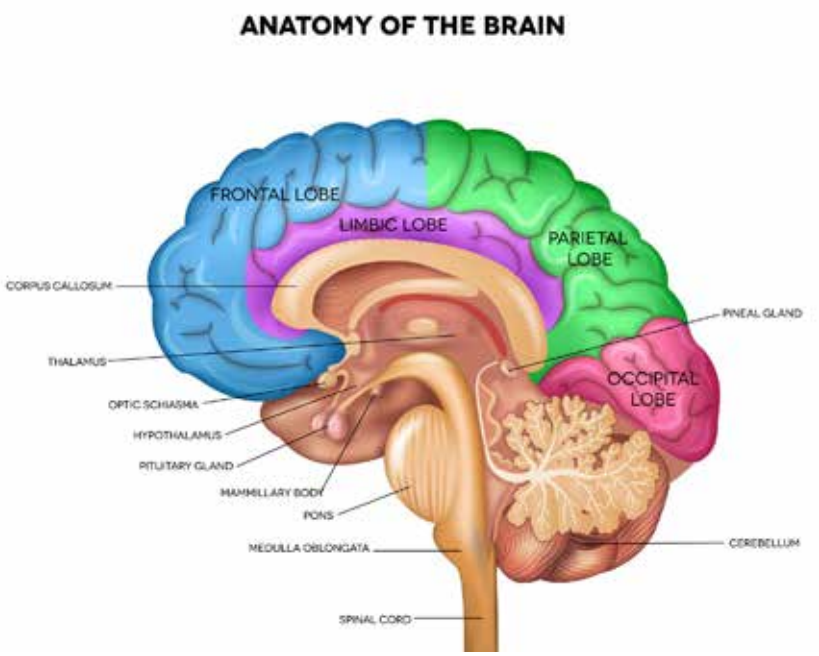
To answer this question, we'll start by looking at the normal factors of aging – in particular, the changes in brain anatomy and function that occur naturally as we get older. Then, we'll discuss neuro-degenerative diseases (diseases that involve the degeneration of nerves – particularly in the brain) with an emphasis on dementia and Alzheimer's disease. Armed with a basic understanding of what is natural aging and what is abnormal disease, we can interpret the latest research and apply it to the prevention and management of degenerative disorders.

To be clear, age is still considered the largest risk factor for brain degeneration. Past research has shown differences, in structure, function and general properties of our brains as we age. These contribute to psychological, mental and physical changes commonly reported by our elderly. These findings are supported by recent research[i] indicating that changes related to gene switches may provide an explanation for what is observed in our brains as we age.

Basic Anatomy of the Brain

Our brains are made up of two basic types of nervous tissue. First, there is gray matter, which consists of nerve cells in the cortex (the outermost layer of the brain – the surface layer). There is also white matter, which consists of fat-rich, myelin-sheathed axons (literally 'arms,' of white nerve cells) that connect nerve cells from the cortex to other areas of the body and brain[ii].

The brain also contains chambers - hollows deep inside of it called ventricles. These ventricles are filled with cerebrospinal fluid (CSF). The primary functions of these ventricles are creating and circulating that CSF, to provide nutrients and chemical stability to the brain. They also create buoyancy to protect the brain from impact against the skull.



The brain cortex is the outermost gray layer of the brain that houses the gray matter. The main functions of the cortex are incredibly diverse and varied. What's important to understand is that they are all higher functions such as voluntary movement, planning, speech and language processing, etc.

The hippocampus is involved in memory. Impairment of hippocampal functioning always affects memory, both the recall of established memory and the creation of new memories. Our ability to learn will also suffer if the hippocampus is impaired.

The amygdala is responsible for some aspects of memory, decision making, and emotional reactions. Impairment here can have severe effects on our moods, volatility, and aggressive or impulsive behavior.

What CT & MRI Scans Reveal About Natural Age - Related Structural Changes in The Brain

In the last 2 – 3 decades, advances in scientific technology have made it possible to image the living brain with Computer-aided Tomography (CT) and Magnetic Resonance Imaging (MRI) scans. This lets us rapidly advance our knowledge of how the brain develops and functions. The following changes in brain structure have been seen during aging:

- The brain ventricles expand in size as we age (Ventriculomegaly)[iii] - CT Scan
- The brain decreases in size/volume (MRI)[iv]
- The brain shrinks in different areas at different rates over time. Some studies[v] indicate that brain shrinkage occurs at a rate of 1% per annum as we age.

Neuroplasticity and Aging

Our brains can adapt and respond to our environments by appropriately changing its structure and function. This fantastic ability is called neuro or brain plasticity[vi]. Our ability to learn how to play a musical instrument, for example, relies on the brain's ability to grow neurons into linked interconnected chains called 'neuro-nets.' Neuro-nets are established through the repetitions of daily practice[vii]. This means, the brain actually changes shape as you learn.

Research has found that as we get older, our brain's capacity to be neuroplastic seems to decrease. This is unfortunate because neuroplasticity allows us to learn and grow, as well as lay down new memories[viii]. However, further research has also shown that people in their seventies are still quite capable of learning and developing new neuro-nets when playing a musical instrument. So, age is not the reason for a decline in neuroplasticity.

Researchers have found that 'calcification' interferes with neuroplasticity. Calcification is caused by changes in our body's ability to regulate calcium (calcium regulation). This, (not age), seems to affect neuroplasticity negatively. Scientists have theorized that an inefficiency in the regulation of calcium causes decreased firing of our nerve cells,

which limits the ability of the brain to respond to signals from the environment[ix]. The firing of fewer nerves leads to decreased brain plasticity because the brain would be slow to respond or react to nerve impulses that normally signal to moderate change. In other words, if you can't fire your brain's nerves properly, you will think more sluggishly. There will also be less information conveyed, and less likelihood of a well-connected neuro-net.

The Hippocampus And Aging

Memory involves the hippocampus, a seahorse-shaped fixture that sits in the lower center of the brain. Aside from the functions of the hippocampus mentioned earlier, it is also associated with emotions and the autonomic nervous system (the nervous system responsible for unconscious bodily functions).

cells in the hippocampal region undergo changes as we age. This biochemical alteration is thought to result from enzyme dysfunction, faulty chemical messaging and genetic switches. Such changes lead to memory loss and cognitive decline – the symptoms we seem to observe in our elderly. Importantly, these scientists are now saying that brain degeneration is due to

Some scientists have claimed that nerve

biochemical changes and not actually due to nerve cell death, as was previously believed[x].

"Only a couple of decades ago it was believed that brain cells could not regenerate, but now this absurd idea is no longer taught in most medical schools. Instead, the current consensus is that changes in the structural form and chemical functioning of parts of the brain account for cognitive decline and not cell death, because nerve cells can regenerate."

Thinning of the cortex

In general, studies have shown that from the time we become adults, to when we become senior citizens, there is an overall decrease in the gray matter of the brain. White matter, however, was found to increase until the 40's, after which it declined for the remainder of the individual's lifespan.

An interesting find from neuroscientists is that the left hemisphere of our brains appears more prone to gray matter loss on average than the right. Our left-brain ability to retrieve words and express language seems to diminish with aging - if we are to believe these few studies[xi].

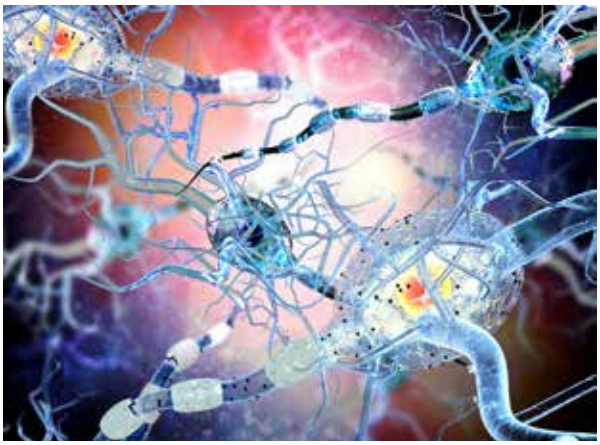
Age-related neuronal morphology (Changes to the shape/functioning of nerve cells)

For decades, mainstream science has promoted the belief that nerve cell death decreases cognitive function with aging; however, this absurd idea is no longer taught in most medical schools because nerve cells can actually regenerate!

Research is now revealing that the decline in cognitive functions seen with aging is more to do with changes in the structure, shape or function of nerve cells.

Neurofibrillary Tangles

Neurofibrillary tangles are pairs of corkscrew shaped filaments (tiny fibers from nerves) that become knotted and lose integrity-causing brain pathology. One of the main differences between normal age-related changes and neurodegenerative diseases is the relatively low occurrence of neurofibrillary tangles in normal aging. Additionally, normal brains do not have specialized structures called 'amyloid plaques' (similar structures to neurofibrillary tangles) while the brains of those suffering



from degenerative disorders have these plaques[xii].

Role of inflammation & oxidative stress

Scientists now believe that the brain is damaged through a natural buildup of excess free radicals over the span of a lifetime. Excess free radicals result in decreased mitochondrial function and damage to brain tissue. The result is increased inflammation (swelling), decreased energy and impaired thinking processes. If the damage caused by free radicals is not relieved by antioxidants, then the development of brain pathology usually follows[xiii].

Specifically, damage by free radicals can cause impaired protein production, lipid (fats - the main component of nerve cells) degradation and DNA mutation in both the cell nucleus mitochondria. DNA mutations and impaired protein production are especially dangerous because they cause a wide range of pathological changes in tissues which then leads to radically (sic) increased rates of aging[xiv].

The theory of 'free radicals and aging' is based on the understanding that damage sustained by DNA causes degenerative brain diseases - genetic changes (mutations) almost always precede rapid, prematurely induced aging. Across both human

and animal studies, results have been remarkably consistent and show in nearly every case that DNA damage accumulates with aging. This understanding is the most popular theory of aging currently favored by modern neuroscientists.

Chemical changes in the body due to aging

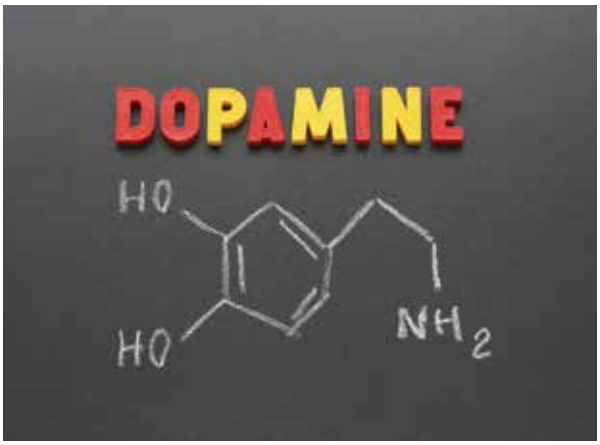
Apart from the physical or structural changes we have seen that accompany normal aging, we also experience a range of biochemical changes as we mature. Nerve cells communicate through chemical molecules called neurotransmitters. Neurotransmitters are found not only in the brain but also in the heart and gastrointestinal tract (GIT).

A neurotransmitter binds to the surface of a cell by fitting into a receptor 'docking' site, specific to each neurotransmitter. If the binding site or the neurotransmitter molecule is altered in any way, then messaging or information cannot be relayed, and nerve function becomes impaired.

Some researchers have identified several changes to neurotransmitters and their binding sites that are thought to occur 'naturally' from aging. These changes occur in at least three major neurotransmitters, 'Dopamine,' 'Serotonin' and 'Glutamate' - each is essential for nerve cell communication in the brain and central nervous system.

Dopamine

There is compelling evidence demonstrating a link between aging and a significant decrease in the synthesis of Dopamine[xv] neurotransmitters and related binding sites. Dopamine losses are seen in areas of the brain known for memory processing, such as the hippocampus and the amygdala[xvi]. This biochemical change is thought to be the underlying reason for degenerative brain disease. It is believed by some that decreased Dopamine causes cognitive rigidity (dogmatism) and physical inflexibility as we age – this would negatively impact our ability to respond dynamically to our



environment[xvii]. Neuroplasticity is also heavily implicated in dopamine production and function.

Serotonin

Many have heard about the importance of Serotonin, which if depleted is associated with depression and impacts wellbeing. There are many different subtypes of serotonin (e.g. 5-HTP), all of which are transported by serotonin transporters. One study showed significantly decreased levels of serotonin production and transportation in elderly populations. [xviii] [xix]

The serotonin system not only affects how good we feel but also impacts the quality of our sleep by regulating melatonin.

Melatonin controls our waking and sleeping cycles[xx]. Scientists have further noted how diminished sleep also depresses our immune function.

Serotonin is also known to moderate our perception of pain – less serotonin means a lower tolerance to pain in our bodies. Think back to a time where you were sleep-deprived, remember those 'aches and pains'? We now know serotonin impacted our threshold for that pain!

Glutamate

Another important neurotransmitter that dwindles as we age is Glutamate. Research studies have shown lower concentrations in elderly participants than that measured in younger participants[xxi].

These differences in Glutamate levels were observed in the motor cortex (part of cortex responsible for voluntary movement) as well as sites associated with degenerative brain disorders[xxii]. Some researchers have suggested that declining levels of glutamate associated with aging may well be a useful predictor or indicator of certain brain disorders[xxiii].

Neuropsychological changes seen during aging

Neuropsychological changes are changes in a person's personal experiences, awareness, cognitive reasoning faculties and outward behavior.

These are those characteristic changes associated with 'being old.'

Orientation – self-awareness in the context of our environment [xxiv]

Usually, medical science evaluates the integrity of a person's mental orientation by checking if they know who they are (identity)

Scientific discourse on aging is now changing, and scientists are claiming that mild disorientation may be natural

their age, where they are physically (place) and the time or date of the neuropsychological examination.

Disorientation is one of the most common symptoms associated with brain dysfunction[xxxv] and testing for 'orientation' is included in all medical neuro-evaluations.

in a healthy aging population[xxvi]. Although mainstream science has endorsed this view, more recent research[xxvii] has shown that loss of orientation may not be a normal process of aging, with 92% of elderly subjects having perfectly normal orientation[xxviii]. This is great news because if we are all going to lose our orientation as a function of aging, then who'd look forward to growing old?

Attention

Our attention relates to our ability to selectively focus on one task at a time while blocking out awareness of unrelated tasks or information[xxix]. Without our selective attention, we would be unable to focus on anything. Many elderly people report that they are not able to focus their attention as well as they did in their youth.

Current studies show limited differences between the young and old when performing two tasks simultaneously. Other research studies indicate that elderly have trouble retrieving or accessing information when their attention is split between two tasks. However, there was no significant difference between test scores of the elderly compared to younger study participants[xxx].

Additionally, the test scores remained similar between the young and old for focused attention on tasks performed over a sustained period of time[xxxi].

In fact, one study featured participants in their seventies who remained stable in their ability to focus for long periods of time .

Again, modern science is showing that decline in attentional ability is not part of the normal suite of aging conditions! Perhaps impaired hearing and vision impact attention testing, because subjects are simply unable to hear the questions; or perhaps the subjects were unable to identify written instructions[xxxii]. These factors may leave the elderly feeling like they have lost their ability to concentrate as effectively as when they were younger. The truth is, they concentrate just fine or even better than their youthful counterparts. Healthy aging is not the negative we have been led to believe!

Changes in memory

It's currently thought that mild cognitive impairment (MCI) is a natural result of aging. MCI is a term used to describe mild memory loss and occasional trouble with accessing words. It is synonymous with another term, Age-related memory impairment (AMI). AMI/ MCI are both considered to be normal phenomena amongst the biomedical community.

People with MCI may have difficulty finding items and may forget to arrive at prescheduled appointments. MCI increases the risk of developing Alzheimer's disease (AD) by 40% and is possibly a bridge between healthy brain function and degenerative brain disorders. One study reported a 55% AD incidence within 4.5 years of MCI diagnosis[xxxiii]. Equally interesting is that not everyone with MCI develops AD.

The process of being able to make new memories becomes more challenging as we age.

Many studies have concluded that this ability declines naturally with age-related changes. Although science has identified many types of different memory functions, only episodic memory, and working memory appear to be affected by natural aging.

Episodic memory relates to remembering the context or source associated with a memory – for example – knowing that you were given a gift, but not able to remember who gave it, or why or how it arrived in your possession. Episodic memory is networked throughout the frontal cortex of the brain (executive functions involving reasoning tasks), and the temporal and parietal lobes. It forms a vast interconnected lattice in each of us that enables specific aspects of our memory to function.

Working memory is the ability to place an event or facts into short-term storage (like a buffer) until the data can be used or assimilated into our long-term memory. Apparently working memory is also reduced as we age, per some scientists; however, others have questioned this interpretation. They claim that there is an environmental explanation for the decline. Working memory is used far less in the elderly as compared to young people, who spend hours learning facts and are consistently using and reinforcing these neural networks.

In general, the neuro-nets that are kept active tend to remain active and thus stay healthy. A well-known saying amongst neuroscientists is 'Nerves that fire together, wire together.' The pathways we do not use tend to degenerate over time. It seems that we must exercise our brains to keep them healthy after all. It's the same with our real muscles, which can serve us if we exercise them regularly throughout our lives.
It's easy to protect memory function as we age if brain exercise is introduced early in our lives and naturally integrated as part of our lifestyle throughout adulthood. Even when the elderly are introduced to exercise for the first time, scientists have noticed that the hippocampus increases in size with improved memory function.

Changes in language

The main change that naturally happens as we age is that it becomes harder to retrieve words[xxxiv]. Everyone has had the experience of trying to remember someone's name or a place, and the information is just not there. 'Oh, it's on the tip of my tongue...!' is a regular part of aging.

Genetic changes

The most interesting research into how our bodies and brains function is in the relatively new field of science called epigenetics. Epigenetics centers on the effects of environmental impact on our genes. Each biomedical discipline is involved in separate epigenetic research, according to their area of specialty.

Nutrigenomics is a typical example of epigenetic research that focuses on the effects of nutrition on our genome. While information from epigenetic research is growing, a single topic does not give a clear picture of aging. Those looking to stay up-to-date often require an understanding and meta-analysis of a broad spectrum of biomedical categories.

Aging is a biomedical phenomenon that spans almost every medical field, and it seems that our current understanding is constantly being overturned by new research that invalidates previous medical models. We are living in exciting times, and when it comes to aging research - we are at the dawn of unfolding a new model for healthy aging.

What is already excitingly clear is that our past comprehension of aging is outdated by current epigenetic research. For the first time ever, we can imagine that we may soon be able to claim that aging may not even exist! At least not in the way we see it or have previously defined it.

The effects of aging vary among people and are due to both individual genetics, coupled with environmental factors. So, what do modern neuroscientists say about normal neuro-aging?

They show that there are changes in gene expression during aging, in addition to a decline in brain function. The current perspective is that accumulated free radical damage results in switching certain genes off somewhere after 40 years and promotes other genes to become active.

The genes that are typically switched off (regulated) are:

- Genes that regulate calcium signaling
- Genes that regulate receptors involved in learning
- Genes that regulate neuronal plasticity – our ability to respond to change

Other genes that are switched on (unregulated) are:

- Genes associated with stress responses that regulate DNA repair
- Genes that promote antioxidant defenses within the body

Delaying the pathological effects of aging

We will explore these areas in more detail in later in this book, but it may be worth a quick, sneak preview. Now that we have covered the observable, measurable factors that change with age, we can perhaps begin to appreciate the measures one might take to counteract some of the more damaging effects of aging.

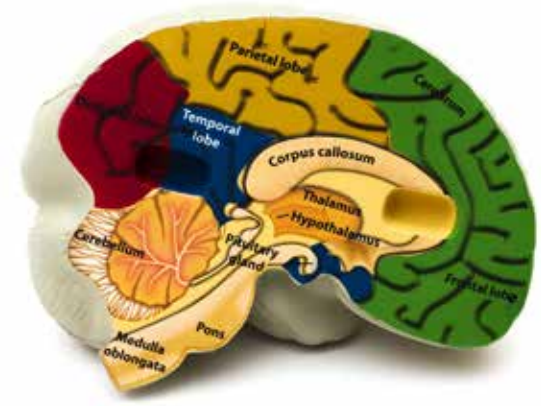
Aging is natural and inevitable with the process beginning from birth. We now suspect that severe symptoms are not synonymous with aging and can be halted and possibly even prevented through nutrition and lifestyle factors. We also believe it might be possible to minimize the natural non-pathological effects of aging.

In both cases, simply following the lifestyle behaviors below has shown dramatic and significant results:

- Education[xxxv] that results in informed, healthy choices[xxxvi]
- Physical exercise[xxxvii]
- Engaging in ongoing mental activities[xxxviii] such as crossword puzzles and reading
- Learning new information such as developing a new hobby or activity
- Managing stress responses
- Keeping social and friendship networks active[xxxix]
- Ensuring a healthy nutritious diet rich in therapeutic antioxidants[xl]

Hypothalamus inflammation and GnRH

One study has shown evidence linking inflammation of the hypothalamus with overall increase in aging. The research documented a strong connection between activation of NF-κB, a protein complex involved in DNA transcription, inflammatory cascades, and cell survival. Activation of NF-κB alters Gonadotropin-releasing hormone (GnRH) that exhibits anti-aging properties when injected in areas outside of the hypothalamus.



Conversely, aging factors were induced when researchers injected the hormone directly into the hypothalamus. While this initial study suggests a new way to tackle aging, more research needs to be conducted before these findings can be used to develop a novel approach to anti-aging[xli].

Brain exercise affects both the young and old. For the young, if introduced early it can form a constructive habit that can be instilled throughout adulthood. For the elderly, especially those that suffer from Alzheimer's or other disorders that affect the memory it is therapeutic. When the brain is introduced to exercise, the hippocampus region can regain its size and improve memory.

Causes of memory disturbance

There are also many different physical and psychological factors that can affect the way our memory functions[xlii]:

- Anxiety
- Stress
- Depression
- Infection
- Thyroid imbalance
- Dehydration
- Nutritional deficiencies such as insufficient Magnesium, Zinc, Vitamin B6 & B12, and folate
- Alcoholism
- Medication
- Substance abuse
- Lack of exercise / sedentary lifestyle

CHAPTER 2

BRAIN DISORDERS - A MODERN PANDEMIC?

According to the National Institute of Health ADEAR Center, Alzheimer's disease is a progressive and irreversible brain disorder that currently affects over 5 million US citizens. It typically affects those in their 60's and is characterized by the slow destruction of memory and the ability to think effectively, ultimately leading to an inability to perform even the simplest of daily tasks.

Dementia

Dementia is the loss of rational thinking, memory, and reasoning that prevents a person being able to live normally. In its most severe form patients are entirely dependent on others to survive. Alzheimer's Disease accounts for approximately 80% of all reported dementia cases and is identified as the 6th leading cause of death in the US, but experts assert that it is the third leading cause of death in the elderly population along with cancer and heart disease.

Dementia is classified according to the area of the brain affected, for example vascular

dementia (changes in arteries or veins). People commonly develop more than one type of dementia.

Alzheimer's Disease

In 1906 Dr. Alois Alzheimer discovered unusual changes in brain tissue when conducting a autopsy on a patient who had suffered from a strange form of mental illness. Her symptoms had included memory deficit, language abnormalities and strange behavior - characteristic of what we define as Alzheimer's Disease today. The autopsy identified abnormal clumps of brain tissue, today known as amyloid plaques and bunches of entangled fibers typically known as Neurofibrillary or Tau tangles in our current biomedical models. It's fascinating that at the time Dr. Alzheimer treated his patient, this mental illness was not considered common. This implies that something has changed in the last century to elevate this disease to almost pandemic proportions. In fact, we are seeing an increase in Alzheimer's (AD) cases globally, making us question what could create these differences in the elderly between 1906 and a century later.

These days we know that in addition to the plaques and tangles in AD, we also find nerve cell death or brain atrophy (degeneration). This interferes with the ability of neurons to communicate with the body in terms of sending and receiving information. These changes represent the underlying basis of Alzheimer's and understandably give rise to the collection of symptoms (making it a syndrome) that we then diagnose as Alzheimer's. What do these changes look like?

Cross section of a healthy brain compared to brain tissue atrophy in Alzheimer's disease

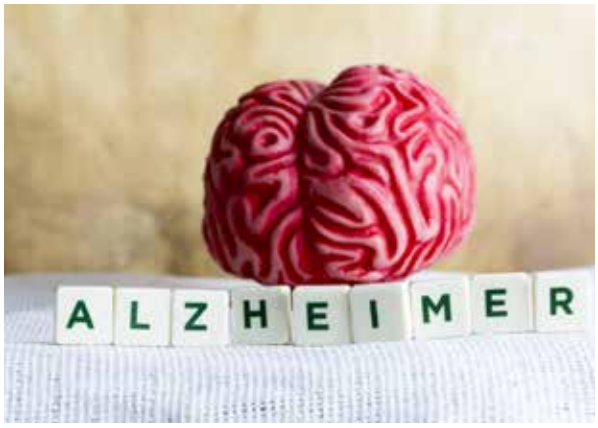
Progression of Alzheimer's disease

Most Alzheimer's experts believe that the onset of AD begins almost a decade before a person is aware of memory and thinking abnormalities. During this symptom-free period, the brain makes abnormal proteins that create amyloid plaques. Healthy nerve cells start to function less efficiently until they completely lose their connectedness to other neurons and then die off.

Although these changes are widespread throughout the brain, most initial damage is sustained in the hippocampi area (primarily involved in memory retrieval and storage) of the brain. As the damage spreads through other parts of the brain, we begin to see accelerated neuron death and the brain starts to atrophy. This is when most people are diagnosed with this awful disorder. In the late stages of Alzheimer's, the brain has shrunk dramatically with vast areas of damaged or lost brain tissue.

Preclinical Alzheimer's disease

MCI (Mild Cognitive Impairment) with associated memory symptoms is often the first diagnosis in a progression that leads to Alzheimer's Disease. MCI memory problems are mild and do not interfere with normal everyday functions like they do in Alzheimer's. Some people experience difficulties with their sense of smell and also report problems with movement. People with MCI may reverse these symptoms back to normal levels of function, although many will end up with Alzheimer's.



Initial symptoms signaling the onset of Alzheimer's vary. Some people have more difficulties with impaired judgment, faulty reasoning or difficulty finding words compared with others who have more trouble with memory function.

It is often difficult to diagnose AD early since it's not really a disease, but rather a syndrome (a collection of observed symptoms) that often overlap with changes due to natural aging.

Mild Alzheimer's disease

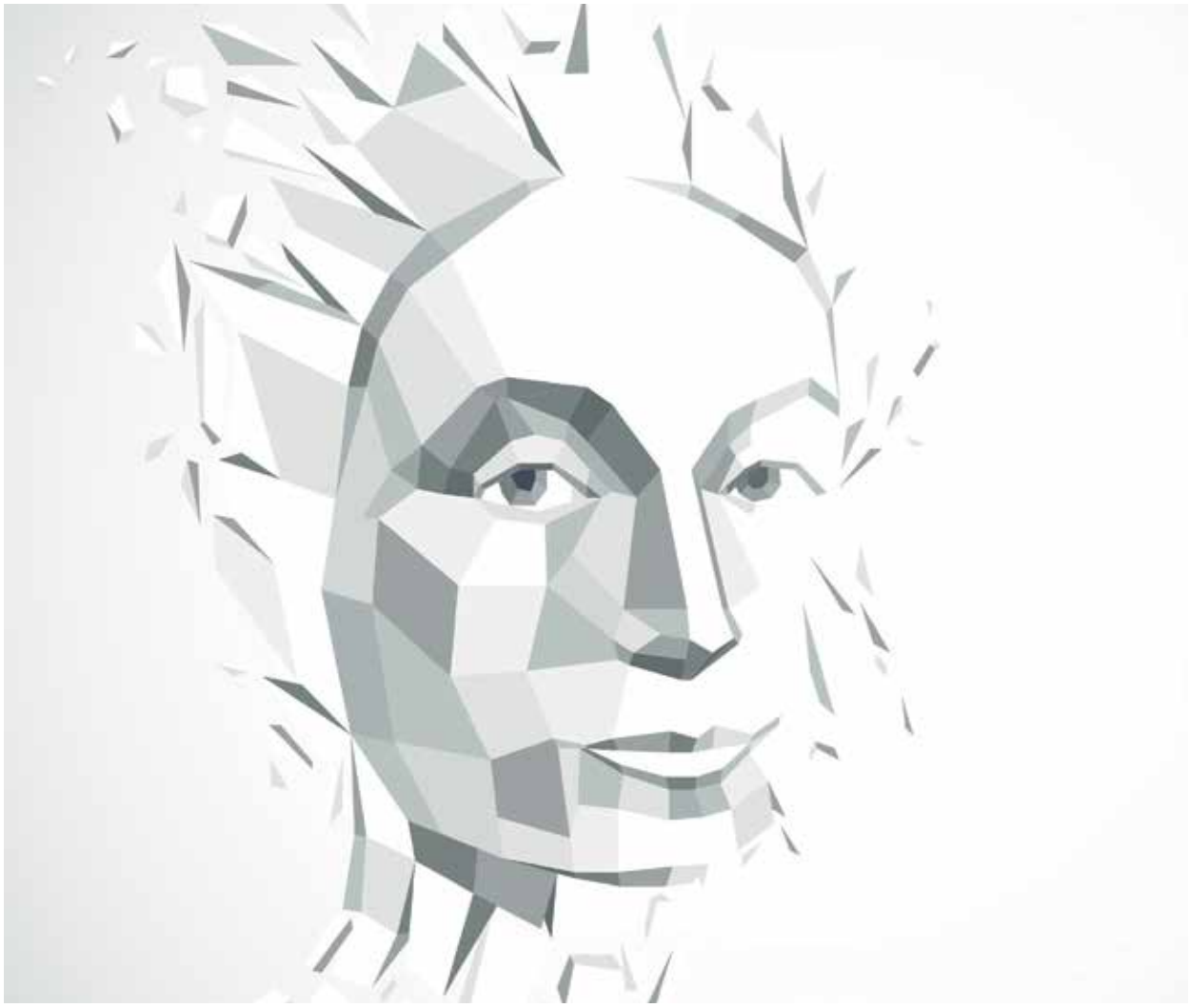
At the onset of AD, people may experience forgetfulness, memory loss and difficulty with certain mental tasks. At this stage, they may be diagnosed with mild onset AD if they experience some or all of the following:

- Getting lost or 'wandering'
- Repetitively asking the same questions
- Taking longer completing daily tasks
- Personality or behavior changes, such as feeling suspicious of others

Moderate Alzheimer's disease

This level of brain damage impairs areas of the brain that regulate language, reasoning, sensory processing and conscious thought. People at this stage typically suffer from:

- Increased memory loss
- Confusion
- Unable to lay down new memories
- Difficulty learning a new task
- Problems multi-tasking e.g. difficulty getting dressed
- Difficulty adapting to new situations
- Some people can experience psychosis in the form of hallucinations, delusions or paranoia
- May act impulsively (as compared with past history)



Severe Alzheimer's disease

Severe AD is identified when nerve cells have lost functionality due to brain atrophy and the widespread development of plaques and tangles in brain tissue.

At this point, the disease has progressed so that the often-bedridden patient can no longer even communicate and is completely dependent on others for survival. The body will eventually shut down and die at this stage.

CHAPTER 3

THE REAL CAUSES – ENVIRONMENTAL, LIFESTYLE, AND GENETIC FACTORS

Alzheimer's is a slow degenerative brain disease that is an extension of normal age-related changes due to inflammation, increased free radical production, faulty protein conduction and mitochondrial dysfunction. These elements are initiated by genetics, lifestyle factors, and our environments.

Mild Alzheimer's disease

At the onset of AD, people may experience forgetfulness, memory loss and difficulty with certain mental tasks. At this stage, they may be diagnosed with mild onset AD if they experience some or all of the following:

- Getting lost or 'wandering'
- Repetitively asking the same questions
- Taking longer completing daily tasks
- Personality or behavior changes, such as feeling suspicious of others

Genetics [xliii]

Alzheimer's typically comes in two categories - late onset AD, (95% of which are diagnosed in their sixties), and early onset AD (those diagnosed between the ages of 30 and 60 years) which make up the remaining 5%. Late onset AD is generally related to genetic markers linked to the apo-lipo-protein E (APOE).

Early onset AD mostly results from inheriting the FAD or Familial Alzheimer's disease gene. An extra copy of chromosome 21 found in Down's syndrome also promotes AD because the gene confers harmful amyloid proteins which may form into plaques.

Epigenetic research is also contributing a deeper understanding of the genetic profile of this disease.

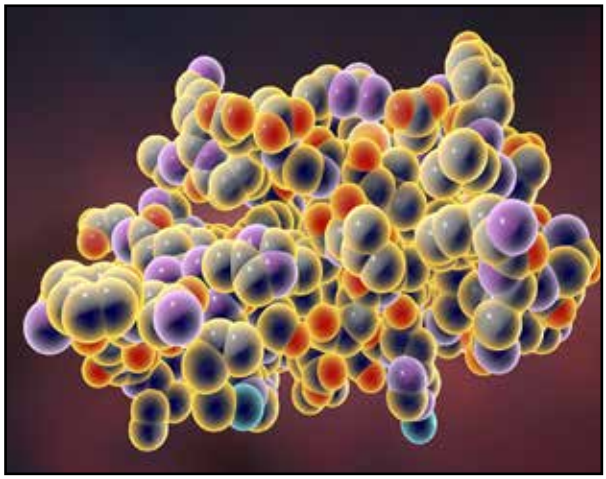
It is showing how lifestyle and environmental factors affect gene regulation and revealing the mechanisms underlying or contributing to brain degeneration.

Health, Environmental and Lifestyle Factors

Current research is proving exciting when it comes to Alzheimer's. The latest research indicates that there are multiple factors involved in the progression of AD. The studies reveal there is a way to halt Alzheimer's and its progression completely.

There has been much interest in the role vascular diseases such as stroke, heart disease and hypertension play in cognitive functional decline. There has also been a strong correlation that diabetes and metabolic syndrome play a role in dementia and the enhanced damage seen through more severe aging factors. These chronic lifestyle diseases have all increased in frequency over the last few decades and are now seemingly epidemic levels in multiple countries across the globe.

If you think about it, it does not seem that surprising that Alzheimer's is yet another



disorder that stems from lifestyle factors. In just a while, we will show you how changing our lifestyles can lower the risk for severe brain degenerative disease (along with other chronic lifestyle diseases). The end of these terrible pathological problems which appear to be endemic to our modern lives may finally and conclusively be in sight.

Risk Factors for Alzheimer's disease

The following factors are associated with an increased probability of developing Alzheimer's disease[xliv]. The list continues to expand with increased research. Many of these factors can be addressed through lifestyle and environmental changes[xlv]. Eventually, we hope that with advances in biomedicine and epigenetics, we will be able to alter our genes and switch them on and off immediately. For now, we have the arsenal to stave off dementia and Alzheimer's Disease through nutrition and lifestyle, allowing us to live long, healthy and meaningful lives.

Risk Factors That Are Associated With AD

- Increased Age
- Family history of Alzheimer's disease
- Carrying the ApoE4 gene
- Some bacterial infections
- Cardiovascular disease (and risk factors) – this includes diabetes, atherosclerosis, high blood pressure and high cholesterol[xlvi] and strokes[xlvii] as they are associated with an increased deposition of amyloid beta
- History of head trauma
- High homocysteine levels
- Nutrient deficiencies
- Abdominal obesity (a high waist-to-hip ratio)

Theories around Alzheimer's Disease

There are plenty of theories around Alzheimer's, and all of them contribute to an overall understanding of the symptoms of this catastrophic degenerative process. Here are some summaries of the theories behind AD.

Senile Plaques

We previously discussed amyloid plaque clumping as a prominent feature of Alzheimer's Disease. These protein fragments build up initially in key areas of the brain, such as the hippocampus. The amyloid plaques contribute to oxidative damage associated with increased free radical formation.

Increased free radical formation often causes a phenomenon called "excitotoxicity."

Excitotoxicity can be thought of as a receptor site 'burning out' from overstimulation – AKA, a 'fried' receptor. Naturally, fried receptors and elevated free radical counts exacerbate inflammation which leads to increased cell death and an increase in neurofibrillary tangles/plaques[xlviii]. Individual therapies aimed specifically at decreasing these plaques have been disappointing[xlix], but combining therapies through nutritional approaches is now producing unbelievably good results.

Neurofibrillary Tangles

We have discussed these tangles before, but what are they exactly? Nerve cells have a skeleton made up of microtubules that are held in place by proteins called tau. In Alzheimer's disease, the microtubules disintegrate leaving a sticky mess of tau proteins that collect together in bunches called neurofibrillary tangles or NFTs.[l] They function similarly to amyloid beta plaques in that they also cause inflammation and cell death. Once again, these disturbances can be avoided through proper diet and lifestyle choices.

Acetylcholine deficit

We have seen how all the other neurotransmitter profiles are altered by Alzheimer's. Acetylcholine deficiency - another key neurotransmitter was once thought to be the main cause of AD. Therapy tackling acetylcholine imbalance alone has not given the hoped-for results, even though this neurotransmitter is vital for cognitive processing[li].

Although clinical trials prove that acetylcholine supplementation decreases symptoms, it does not prevent the problem. Currently, scientists see this neurotransmitter deficit as being a sign of brain degeneration and not as a cause of AD[lii].

Oxidative Stress

Oxidative stress is a process in which volatile molecules called free radicals, cause damage to cells. Free radicals are normal by-products of metabolism, but when mitochondria become overburdened with pollutants and toxins then free radical production increases dramatically. This process results in dysfunctional mitochondria.

Oxidative stress is majorly involved in the damage caused by amyloid beta deposits, which in turn generate greater free radical production[liii]. Oxidative stress is involved when neurons are damaged, due to free iron collecting on the surfaces of nearby cells called microglia. Microglia cells support and repair neurons, so damaging them directly lead to increased cell death. Iron, therefore, increases free radical formation and generates oxidative stress with a whole host of concomitant problems[liv].

Inflammation

The inflammatory process is crucial to the development of AD. When high levels of amyloid beta accumulate in the brain, it stimulates an immune response. This results in an inflammatory cascade that ends up damaging brain cells[lv].

Part of this response is promoted by tumor necrosis factor-alpha (TNF-α), a proinflammatory cytokine found in high levels in Alzheimer's patients.

Mitochondrial Dysfunction

Mitochondria are our body's energy manufacturers within our cells. They produce energy in the form of adenosine triphosphate (ATP). ATP is the energy currency used to finance our cellular needs. Mitochondrial dysfunction is a hallmark of most lifestyle and age-related diseases. A recent discovery has shown that ApoE4, a gene variant linked to Alzheimer's disease, plays a significant role in disrupting mitochondrial function[lvi].

This confirms that mitochondria limit the toxicity of amyloid proteins[lvii].

Mitochondrial dysfunction and oxidative stress work together in forming a nasty cycle that ultimately leads to neuronal death and large portions of the brain undergoing atrophy.

Excitotoxicity

Glutamate is the most common 'excitatory' neurotransmitter that needs to be in balance for normal brain function. Over firing of glutamate pathways in the brain can be toxic to neurons, causing a phenomenon known as 'excitotoxicity.' Excitotoxicity is promoted by amyloid beta deposits, neurofibrillary tangles, mitochondrial dysfunction and oxidative stress.

Excessive activation of N-methyl-D-aspartate (NMDA) causes glutamate excitotoxicity of the receptors. This is the basis of a theory behind the FDA approved drug Memantine

(Namenda®) that blocks the NMDA receptor and is prescribed for the treatment of moderate to severe Alzheimer's disease[lviii].

Decline of Sex Hormones

As we age, we experience a decline in estrogen in women and testosterone in men. Evidence is accumulating that hormonal losses may also be implicated in Alzheimer's disease. Although it is still to be proved, scientists have noticed that sex hormones seem to help protect the brain from developing Alzheimer's disease[lix]. Future research will help us to understand this connection better.

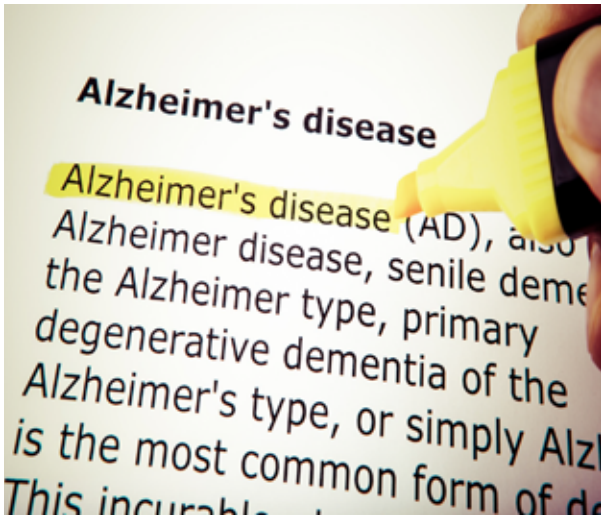
Infections

According to some researchers, chronic infection (either viral or bacterial), may contribute to the development of Alzheimer's disease. This theory is not well known by most of the medical community; however, the research to support this hypothesis is steadily growing. For example, the Spirochetes bacteria has been found in approximately 90% of Alzheimer's patients while being absent from control groups[lx]. Spirochetes and other parasites in the brain increase inflammation and the formation of amyloid beta and neurofibrillary tangles.

Other studies have found that amyloid beta is possibly an adaptive response to infection. These findings have largely been ignored by mainstream medical bioscience. The leading view was that organisms could not enter the brain through the blood-brain barrier, so they discounted this type of research. Now with these new discoveries, some scientists believe that combating infections quickly may delay or even prevent catastrophic degenerative brain changes[lxi].

Diagnosis of Alzheimer's disease

Alzheimer's disease can only truly be diagnosed after death when it's possible to perform an autopsy. Prior to that, doctors generally diagnose by excluding other causes for the presenting symptoms. This is usually done by taking a detailed case history, medical pathology tests and brain scans[lxii].



Other diseases that need to be ruled out before an AD diagnosis can be made include:

- Parkinson's disease
- Stroke
- Tumours(s)
- Sleep deprivation or prolonged insomnia
- Medication side-effects
- Infection
- Dementia unrelated to Alzheimer's

Other Diseases That Cause Dementia[lxiii]

Dementia is diagnosed when two or more mental functions become impaired, such as memory loss with the inability to retrieve appropriate words.

Treatment is basically the same as that used for Alzheimer's disease. Some of the other diseases that cause dementia are briefly listed below[lxiv].

Vascular dementia:

This type of dementia occurs when blood vessels in the brain narrow and lose elasticity, disrupting blood flow through the brain. It is often seen in those who've experienced a stroke.

Symptoms are similar to Alzheimer's but happen suddenly as opposed to the slow progressive decline observed in AD. Treatment is aimed at reducing the main risk factors of smoking, diabetes, and hypertension.

Lewy body dementia:

This type of dementia is another progressive brain disorder caused by a buildup of protein fragments called Lewy bodies. Symptoms involve visual hallucinations, a decline in alertness and attention span,

and motor problems such as rigidity or muscular tension similar to Parkinson's disease. Treatment attempts to alleviate symptoms rather than cure them.

Parkinson's disease with dementia:

Parkinson's disease occurs when we lose the ability to make dopamine in brain cells. The disease presents with tremors in the hands, arms, legs, jaw and face. Other signs include body stiffness with slow movement,

accompanied by impaired balance and coordination. Memory loss can also occur in advanced stages. The pharmaceutical Exelon (rivastigmine) is often prescribed for the treatment of Parkinson's disease.

Front-temporal dementia:

This type of dementia occurs when the frontal and anterior temporal lobes of the brain shrink in size. Symptoms range from impulsiveness to listless behavior, and people may express socially inappropriate

behavior, as well as a slow loss of language functions. No known treatment works, so therapy is prescribed for symptom relief such as anti-depressants. Behavior modification is sometimes also attempted, but is rarely helpful.

Huntington's disease:

Huntington's disease is an inherited brain disorder. Symptoms include mental and emotional disturbance with the loss of memory and uncontrolled movements. Early symptoms reported are mood swings, depression, problems learning something new and forgetting information. Treating symptoms to control volatile emotions is the only type of therapy indicated for these patients.

Creutzfeldt-Jakob disease (CJD):

This disease has been in the news on and off over the last three decades. A rare form of it is called "Mad Cow Disease". It is caused by prion proteins causing radical and quick changes in the brain ending with death.

Prions are abnormally shaped proteins resulting from pathological agents that are taken in through eating cows, birds, and other contaminated animal products[lxv].

These abnormal protein fragments cause rapid degeneration of brain tissue and are always fatal. In the early stages, people may experience failing memory, behavioral changes, lack of coordination and visual disturbances. Mental impairment becomes rapidly more severe as the illness progresses. There is no known cure and drugs may be prescribed to deal with symptoms as they arise.

Dementia Risk Factor Matrix [lxvi]

Very Likely

Advanced age, family history (Alzheimer's, Parkinson's), apolipoprotein E-4, Down's syndrome, head trauma (10x risk w/ApoE4) depression, reduced blood flow, stroke, estrogen imbalance, poor word fluency

Likely

Emotional stress, toxic damage, alcohol abuse, nutrient deficiencies, transmitter deficits, metabolic deficits, under activity, lower educational level, occupational electromagnetic exposure

Possible

Aluminum exposure, latent viruses, sugar consumption, olfactory deficit, coronary artery disease

CHAPTER 4

BIG PHARMA FAILS TO PROVIDE EFFECTIVE SOLUTIONS [lxvii]

The following is a brief list of the medications authorized by the FDA for the symptomatic treatment of Alzheimer's (including their most reported side effects)[lxviii]:

Mild to moderate Alzheimer's Disease

The below medications are cholinesterase inhibitors that attempt to prevent the breakdown of acetylcholine, the neurotransmitter involved in communicating between nerve cells and other cells:

- Donepezil (Aricept®)
- Rivastigmine (Exelon®)
- Galantamine (Razadyne®)
- Tacrine (Cognex®)

(Side Effects: Gastro Intestinal discomfort including nausea, vomiting, and diarrhea)

Moderate to Severe Alzheimer's Disease

Memantine (Namenda®) is used to block glutamate. Its side effects are dizziness, headaches, constipation, and confusion.

The side effects are difficult to distinguish from the common symptoms associated with AD.

Behavioral symptoms associated with Alzheimer's

such as restlessness, sleeplessness, depression and irritability can all be treated with conventional pharmaceuticals used for these imbalances. Each come with their own effects and often further exacerbate AD decline.

These medications work by manipulating neurotransmitters involved in signaling information between different nerve cells. They do not cure or help reverse or prevent underlying pathology and do nothing to restore the body to homeostasis.

As with most other pharmaceuticals prescribed for chronic lifestyle diseases, once you begin, you are on medication for the remainder of your life. This is because a pharmaceutical substitute for a neurotransmitter that the body usually assembles on its own causes our body to down-regulate the production of them. We become chemically addicted to our medication in order to continue living.

Chemical dependency not only causes our bodies to stop producing its own supply of any given neurotransmitter but will also affect the number of receptor sites on the cell surface, so that there are fewer sites for neurotransmitters to dock.

Eventually the neurotransmitter binding sites close inwards. This leaves extra space on the cell surface to be used for other functions, but we would have lost our ability to regulate our bodies without chemical dependency.

One can see that these treatments fail to treat the underlying causes and in the process, actually make matters far worse. The conclusion that pharmaceuticals make things worse can be drawn even before we look at the side effects of taking large unnatural quantities of synthetic, isolated chemicals into our bodies. Their very intended mechanisms of action are part of the problems they create.

Although there may be an initial improvement of cognitive performance when taking these drugs, there is certainly much evidence that they only work for a limited period of time before tolerance to their effects develops. In some cases, they do not help patients at all[lxix].

CHAPTER 5

NATURAL SCIENTIFIC SOLUTIONS

Foods that Can Reduce Your Risk of Alzheimer's disease

Extra Virgin Olive Oil [lxx]

Many people have heard about the benefits of extra virgin olive oil, and many have already benefited from including it into their dietary lifestyle. What you may not know is that olive oil has been shown to stop amyloid beta plaque from forming in the brain.

People who live in the Mediterranean eat a diet rich in olive products with very low incidence of cardiovascular and Alzheimer's diseases. It's also a part of a Paleo diet. Olive oil contains a special phytochemical called oleocanthal. Researchers believe that oleocanthal protects nerve cells by promoting enzymes that effectively remove amyloid fragments from the brain. This process is observed when olive oil is taken twice daily. Olive oil is best taken in a cold pressed form as heat destroys its healing benefits.

Choose locally sourced olive oil, if available; commercial brands are often contaminated with other products[lxxi].

Coconut Oil [lxxii]

Coconut oil can completely reverse Alzheimer's. It's able to provide the brain with ketones to use as an alternative energy source for brain cells. This is excellent news for those who suffer from debilitating diseases like Alzheimer's or have diseases where mitochondrial deficiency is involved.

In Alzheimer's, where systems are breaking down, energy production is limited, and glucose metabolism is difficult, ketones provide the perfect brain power solution. Those with AD have brain cells that can't effectively use glucose from carbohydrates.

Glucose is the main source of energy for the brain, and neurons can die without it. Coconut oil provides the brain with an alternative source of fuel called ketones. This unique oil is comprised of medium-chain triglycerides (MCT) which can be converted into ketones by the liver.

Some scientific data suggests that Alzheimer's can be reversed during this energy conversion process. In one highly publicized incident, a physician named Mary Newport gave coconut oil to her husband Steve, who was suffering from Alzheimer's. While the drugs for Alzheimer's disease seemed to make no difference to his condition, he immediately started improving after consuming coconut oil.

MOST BERRIES

Acai Berry

Studies have shown that South American Acai Berry pulp has powerful anti-inflammatory effects. Inflammation is one of the leading underlying results of many conditions associated with aging. It's also been revealed that acai berry extract inhibits the growth and reproduction of leukemia cells[lxxiii].

Aronia Berry

Found predominantly in North America, the Aronia Berry comes in purple, red and black. It has some of the most potent anti-oxidant results published worldwide, with one of the highest ORAC values known to man[lxxiv]. It can reduce severe inflammation[lxxv] and studies have proven it to reduce colon cancer in rats[lxxvi]. In other studies, it helped reduce diabetes too[lxxvii].

Blackcurrant

Very rich in Vitamin C content[lxxviii],blackcurrants were able to increase research participant's ability to adapt their eyes to darkness as well as reduce the symptoms of having tired eyes[lxxix]. They have shown to protect low-density lipoprotein (LDL) against oxidative stress, which also provides cardiovascular protection benefits[lxxx] and improves circulation[lxxxi]. Rodents that were fed blackcurrant juice experienced increased longevity[lxxxii] with improved blood flow[lxxxiii].This berry even has anti-bacterial properties[lxxxiv] and is also known to alleviate the effects of urinary tract infections[lxxxv].

Blackberry

One of the most tested of the berries, blackberries are known anti-carcinogens. They literally cause self-destructing cancer cells in those that originate in the throat, breasts, colon and prostate[lxxxvi]. Also antibacterial[lxxxvii], this berry is high in anthocyanin (aka C3G), which prevents free radical damage from UV rays[lxxxviii] and protects the liver[lxxxix], as well as blood lipids from lipid peroxidation[xc]. Blackberries also reduce inflammation[xci] and protect blood vessels and supply[xcii].

Blueberry

As well as being a potent source of antioxidants, blueberries are rich in omega-3 alpha-linolenic acid, which is beneficial for brain health[xciii].Blueberries protect the aorta[xciv], as well as prevent the brain from deteriorating or losing memory and functionality[xcv]. Much like blackberries, blueberries induce the self-destruction of cancer cells[90].

Cherry

Proven to be effective in treating arthritis, gout, pain caused by inflammation[xcvi], diabetes and heart disease, cherries are also full of anthocyanins with anti-oxidant properties. Tart cherry powder reduced the amount of cholesterol, glucose, triglycerides and insulin in the blood of rodents[xcvii]. It also reduced cholesterol particularly well in the liver and optimizes insulin production in the pancreas[xcviii]. It also an anti-carcinogen[xcix].

Cranberry

Famous for treating bladder, kidney, and urinary infections[c], cranberries are full of beneficial compounds for the body. It disallows E.Coli bacteria from attaching to urinary tract and bladder walls, allowing them to be removed from our bodies with ease[ci]. Cranberries are also useful in preventing cancers[cii] and reduce ulcers[ciii]. They protect our cardiovascular systems indirectly by creating stable blood pressure, restricting platelet accumulation and reducing inflammation[civ].

Elderberry

An age-old remedy for flu or the common cold, science has recently confirmed this berry as being effective. Elderberry soothes flu-like symptoms and shortens the time one is ill as well[cv]. Studies revealed Elderberry anthocyanins help to protect against free radical damage especially in blood vessels, which is indicative of cardiovascular protective properties[cvi].

Grape or Grape Seed

Grapes are rich in polyphenols, anthocyanins, and antioxidants. The health benefits of grapes were perhaps highlighted by red wine, which retains the berry's health benefits[cvii]. Grapes have compounds that help to protect against oxidative stress[cviii], protect heart cells[cix] and increase the blood flow to the brain, which is highly beneficial for anyone suffering from a brain degenerate disorder.

Grapes also help in protecting neurons, potentially preventing strokes![cx] Other benefits of grapes include the reduction of cholesterol[cxi], inflammation[cxii] and cancer growth.

Pomegranate

Probably having the highest number of antioxidants, pomegranates have proven to be one of the most effective fruits on the planet at protecting against oxidative stress in our bodies. Pomegranates reduce blood pressure[cxiv], arterial plaque[cxv] and a few forms of cancer (prostate[cxvi], breasts[cxvii], lung[cxviii], and colon[cxix]). This fruit also repairs skin and results in more youthful skin[cxx].

Strawberry

Another fantastic antioxidant and anti-carcinogen[cxxi], strawberries are known for being a source of minerals and vitamins. In an experiment conducted on artificially-aged rats, strawberries successfully decreased the age-induced deficit in memory and brain function[cxxii]. Another study proved they prevent excess blood clots, by alleviating blood pressure and reducing the risk of blood heart attacks and strokes[cxxiii].

Leafy Greens and Cruciferous Vegetables

Vegetables such as the leafy greens spinach and kale should be consumed every day for their abundant source of antioxidants and fiber - especially for the elderly.

Research revealed that after eating these, senior citizens did better when tested on memory or verbal ability. In a breakthrough study, 13,000 nurses were observed in their 60's, and 10 years later, in their 70's. The nurses who ate five portions of cruciferous vegetables each week had higher results than those who only ate them twice weekly.

Rosemary [cxxiv]

This herb has been associated with memory and remembrance for centuries. It's used in a large variety of cooking and is readily available at supermarkets. You can purchase it in its dried form, inside capsules or as Rosemary tinctures. This herb is a brilliant option for treating Alzheimer's symptoms, as its main benefits include enhancing memory as well as improving circulation. It does this using powerful phytochemicals which work to prevent and aid in the treatment of AD by complementing acetylcholine.

Acetylcholine is a neurotransmitter that is crucial for accessing and storing memories. Acetylcholine levels are often low in Alzheimer's patients, resulting in nerves that can't transmit impulses to one another. Rosemary achieves what most pharmaceutical drugs attempt to do, it manages to correct acetylcholine. It's also all-natural and has no harmful side-effects, unlike pharmaceuticals. Water mint is another herb that holds similar properties to rosemary in that it can also prevent acetylcholine from falling apart.

Lemon Balm

Also known as Melissa Officinalis, Lemon Balm falls under the mint family tree. It's best known for its ability to restore and relax nerves. Lemon Balm is widely recommended by natural practitioners as a treatment for Alzheimer's. It has a very similar effect in the brain as rosemary and water mint, acting positively upon acetylcholine.

Studies have proven that lemon balm can increase cognitive function and memory ability almost instantaneously. You can purchase lemon balm dried, as tea, in capsules, as oil, or as an extract.

Huperziaserrata – Huperzine A

This plant contains the phytochemical Huperzine A, which can block the receptor for NMDA. This helps to prevent or reduce the excitotoxic effects of glutamate. Equally impressive is Huperzine A's ability to block the enzyme acetylcholinesterase that degrades the acetylcholine.

This neurotransmitter is directly involved in the pathways involved in memory and cognitive functions[cxxv].

The actions of Huperzine A are similar to Donepezil[cxxvi] and Galantamine drugs but with better bioavailability, longer duration, and therefore, greater efficacy than the pharmaceutical drugs prescribed for Alzheimer's[cxxvii]. It also has fewer side-effects than the pharmaceutical counterparts. Research has demonstrated that cognitive performance increased with only 300-500 mcg of Huperzine A taken daily[cxxviii].

Panax Ginseng

This amazing plant has an ancient reputation as a powerful traditional Chinese therapeutic that alleviates fatigue, increases concentration and boosts the immune system. Ginseng decreased the death rate of nerve cells in Alzheimer's cases. Recently, scientists have shown that this plant species contains ginsenosides, molecules that are structurally like steroids, which exert profound effects on memory[cxxix].

In one study, memory effects were enhanced for a six-hour period, on 400mg dosage. Unfortunately, the effects wear off within three months of taking the last dose[cxxx]. These results show promise for symptomatic relief from Alzheimer's by using Ginseng.

Ashwagandha

Ashwagandha (Withaniasomnifera, winter cherryor Indian ginseng) has been used since ancient times in India to treat imbalances associated with aging[cxxxi]. This plant has extraordinary anti-aging properties. Not only was it able to reverse the accumulation of amyloid beta deposits[cxxxii], but scientists have also confirmed that it can completely reconstruct networks of damaged neurons and regenerate nerve cells[cxxxiii].

Amazingly this botanical is also able to boost acetylcholine levels and provides a safe method of treating Alzheimer's without harmful side effects[cxxxiv]. These actions are produced by the anolides compound, an action-packed antioxidant superstar[cxxxv].

Ginkgo biloba

Ginkgo biloba is an ancient anti-aging botanical superstar. It is a traditional medicinal herb that is used to enhance memory and mental function. Modern science has recently confirmed this herb as a potent antioxidant with remarkable healing benefits. It is a useful anti-inflammatory that reduces blood clotting[cxxxvi] and modulates neurotransmission[cxxxvii]

A most exciting discovery revealed that ginkgo blocks amyloid-beta production in the brain[cxxxviii]. Another study has demonstrated Gingko's ability to prevent beta amyloid cell death[cxxxix], while multiple studies have shown Gingko to have positive benefits on mental performance without negative side effects[cxl].

Another factor that contributes to Gingko's anti-aging success is that it increases blood flow to the brain, which also improves memory and mental processes.

As with many brain supporting nutrients, Gingko has shown more powerful results when used as part of a combined therapy with vitamins.

Curcumin

Curcumin is being hailed as a phytochemical that out-performs pharmaceutical anti-inflammatories without any damaging side effects[cxli]. It is found in the turmeric plant Curcuma longa.

Multiple studies indicate that curcumin's neuro-protective healing properties, at doses between 1 – 4g daily, are a perfect way to combat and prevent Alzheimer's disease. Its impressive array of actions includes:

- inhibition of amyloid beta
- clearance of existing amyloid beta
- anti-inflammatory effects
- Potent antioxidant ability,
- delayed degradation of neurons[cxlii]

- Decrease oxidative damage from free radicals[cxlv]
- Modulates inflammatory cytokine levels in brain cells

- Potent metal chelator - binding copper, cadmium, lead and iron[cxliii]
- Decrease cognitive dysfunction
- Reduce neural synaptic damage
- Reduce amyloid plaque deposits[cxliv]
- Reduces nuclear factor-kappaB, a factor that regulates many genes involved in the cytokine inflammatory cascade[cxlvii]

Time to stock up on aromatic spices and create new taste sensations! The good news is, you will enjoy these spices well into your older years.

Coffee and Caffeine

Coffee has recently been in the news after findings that regular consumption reduces the risk of developing Alzheimer's or Parkinson's diseases[cxlviii]. Regular coffee drinkers can decrease amyloid beta deposits by preventing the enzyme that is involved from laying deposits. Another anti-aging perk for established coffee drinkers is that coffee has been proven to improve short-term (working) memory[cxlix].

Coffee beans also contain another powerful antioxidant polyphenol called Chlorogenic acid. This phytochemical decreases hypertension, inflammation, blood platelet build-up and reduces the risk for type II diabetes[cl]. Polyphenols are largely destroyed through roasting, so Chlorogenic acid amounts differ according to how long the bean is roasted. There is even a patented method that increases the amount of polyphenols compared to standard procedures[cli].

Green Tea – Let's go green

This tea is an epic miracle worker. The active components responsible for green tea's powerful benefits are the flavonoid class of polyphenols known as catechins. These potent antioxidant phytochemicals can bind to metals (chelator) with powerful anti-inflammatory action[cliii]. The flavonoids found in green tea, especially epigallocatechin gallate (EGCG), are known to reduce amyloid beta deposits in the brain.

Amazingly, scientists discovered a whole host of remarkable healing properties of green tea that include:

- Suppression of mental dysfunction resulting from amyloid beta damage[cliii]
- Decreased neurotoxicity associated with amyloid beta damage[cliv]
- Powerful antioxidant
- Effective anti-inflammatory
- Reduction of Alzheimer rates[clv]
- Possible modification of nerve cell signalling

- Possible regulation of genes responsible for cell lifespan
- Possible role in regulating the mitochondria – the powerhouse of energy production in each cell
- Potent metal chelator
- Reduces amyloid beta protein[clvi]
- EGCG appears to block plaque from forming in the brain
- Contains L-Theanine, which decreases anxiety in multiple studies

Cinnamon

This is a spice that most people are familiar with and use in many recipes and food products, but what if it's medicine as well? In a 2016 study, mice were fed small quantities of cinnamon to assess if it could affect the hippocampus and turn slow-learning mice into fast learning ones[clvii].

The metabolite sodium benzoate (NaB) compound, which is found in cinnamon, has previously shown to support neuro-plasticity molecules. The results showed that cinnamon is a very powerful hippocampus stimulant and improves spatial memory too, making the slow mice learn much faster.

In the case of humans, it should help to improve cognitive and spatial memory as well.

Dark Chocolate – an awesome superfood

Finally, fabulous news for chocolate lovers everywhere - We can protect our brain and improve our lives by eating dark chocolate regularly. Plus, when made Paleo-friendly with coconut oil and natural sugars, it has even more health benefits.

Dark chocolate / cocoa can:

- Increase our happiness factor by increasing endorphins[clviii]. Endorphins bind to opiate receptors in our brain

giving us euphoria. They help balance stress and reduce pain.

- Help alleviate depression while boosting positivity and increasing happy mood states. This is because chocolate contains tryptophan which is a precursor to serotonin.
- Add to happiness with its bliss molecule, anandamide that simulates THC (tetrahydrocannabinol), the active compound in cannabis[clix].
- Act as a mild aphrodisiac due to the combination of phenylethylamine[clx] and theobromine[clxi].
- Boost memory attention span, problem-solving and response times mostly because of increased blood circulation to the brain[clxii]. The cocoa flavonoid polyphenols are behind these potent brain-boosting properties[clxiii].
- Protect our brains from free radical damage. Cocoa and dark chocolate contain high levels of antioxidants, so much that researchers claim it is worthy to be classified as a 'superfood.'
- Contain flavonoids that improve memory and learning in the hippocampus[clxiv].
- Contain moderate amounts of caffeine which is known to stimulate mental alertness, memory, improved mood and increase in energy[clxv].
- Possibly alleviate cravings for sweet, savory and junk food[clxvi].
- Possibly reduce Alzheimer's risk and dementia[clxvii].
- Inhibit the stress hormone cortisol[clxviii]. Chocolate contains magnesium, which is an essential mineral that is vital to healthy brain function. It is reported to improve memory, help to increase focus, promote deep sleep, and enhance our mood[clxix].
- Decrease insulin resistance. This is helpful in Alzheimer's because insulin dysfunction is associated with AD[clxx].
- Act as a prebiotic in the gut by increasing levels of healthy bacteria, Lactobacilli and Bifidobacteria[clxxi].
- Provide neuroprotection and increases neuroplasticity.

Eating dark chocolate regularly is a smart option[clxxii].

SUPPLEMENTS

Alpha Lipoic Acid (ALA)

This powerful antioxidant can decrease inflammation[clxxiii] and improve acetylcholine concentration[clxxiv]. An additional (and exciting) function of alpha lipoic acid is its ability to detoxify the brain from heavy metals and toxins. This potent metal chelator can bypass the blood-brain barrier, unlike most other chelators that restrict their work to the gastro-intestinal tract. Initial research has shown promising results.

These included stabilized cognitive functions[clxxv] in addition to halting disease progression[clxxvi] in AD. This was achieved by individuals who took a daily dose of 600mg alpha lipoic acid for two years.

Acetyl-L-Carnitine (ALC)

Acetyl-L-Carnitine (ALC) is another exciting antioxidant that protects and repairs brain damage[clxxvii]. It significantly boosts acetylcholine levels and promotes healthy mitochondrial function, giving protection against the destructive effects of the amyloid beta protein[clxxviii].

Combining alpha lipoic acid with Acetyl-L-Carnitine, produced synergistic effects in an interesting study that reversed mitochondrial decay in aged subjects[clxxix]. Other studies have shown that ALC could decrease high Homocysteine levels. Homocysteine is considered to be a useful biomarker of serious disease progression.

High Homocysteine levels are associated with[clxxx]:

- Blood-brain barrier deterioration and loss of integrity
- High amyloid beta concentration in brain tissue
- Increased neuro-fibrillary tangles
- Cognitive dysfunction

ALC is a remarkable nutrient, because apart from lowering Homocysteine, it is also able to prevent damage from amyloid beta proteins. It does this by interfering with amyloid beta protein metabolism - without any harmful side effects[clxxxi]. ALC is metabolic cofactor and energizer that enhances mental functioning as we age[clxxxii].

Vitamin D – the "sunshine vitamin."

There is ample evidence to show that people all over our planet are deficient in Vitamin D. Older members of our population and those with Alzheimer's disease are most at risk for low levels of vitamin D. Considering the abundance of receptors for Vitamin D in the brain[clxxxiii], it is not surprising that this hormone has been noticed by scientists, because of the powerful effects it has on

Vitamin D appears to regulate calcium levels in the brain – this affects whether a nerve will fire. Calcium is needed for neurons to conduct an electrochemical signal. Other actions performed by Vitamin D are related to promoting nerve growth, giving it a well-deserved reputation as being neurotrophic (promotes new brain tissue growth).

nerve cells.

Vitamin D also supports the brain to detoxify harmful substances, with research revealing that there is a 30% increase in amyloid beta clearance out of brain tissue when taking Vitamin D supplementation[clxxxiv]. It is an impressive antioxidant, with potent anti-inflammatory effects capable of clearing beta amyloid plaque deposits out of brain tissue.

Our modern lifestyles, along with environmental factors, have affected the

amount of time that people spend in direct sunlight. Reducing our exposure to the sun has gained increasing popularity amid fears of ozone depletion and increased cancer risk from solar radiation. Vitamin D, however, is manufactured freely when we are exposed to sunlight. Sunblock may, in fact, be costing us more than we bargained for! They may protect skin from harmful radiation, but rob us of our ability to produce Vitamin D in our bodies and may contribute to the widespread deficiency of vitamin D. This is scary when we read research that Vitamin D deficiency is strongly associated with mental impairment! In one study, subjects who took a regular high dosage Vitamin D benefitted by a whopping 75% reduction in developing Alzheimer's[clxxxv].

According to some experts, it's important to expose 75% of your body to sunlight for 20 minutes daily to benefit from natural Vitamin D production. If this is not possible in your current lifestyle, then Vitamin D needs to be added as part of your daily supplemental program until you can revamp your lifestyle and diet for optimal vitamin D levels.

Vitamin D is a fresh reminder that we don't have to suffer debilitating effects of brain degeneration – all we need to do is change our lifestyles to experience wholesome natural health with our mental capacities fully intact.

Vitamins C and E

An increase in the deterioration of lipids from free radical damage is associated with Vitamin E deficiency in Alzheimer's patients[clxxxvi]. Combining the well-known antioxidants, vitamins C and E into a supplementation program, resulted in decreased oxidative damage in participants with Alzheimer's[clxxxvii] disease and decreased AD rates in general[clxxxviii]. The healing potential of these vitamins is improved when taken together, showing enhanced synergistic effects with combination [clxxxix]therapy. These results were achieved with moderate dosages of 400 IU Vitamin E and 500 mg Vitamin C taken daily.

Another interesting finding is that Vitamin E was associated with a 26 % increase in lifespan amongst Alzheimer's sufferers. Vitamin E is found in foods such as peanuts, green leafy vegetables, kiwi fruit, tomatoes and some seed and nut oils, like flaxseed oil and peanuts. Sunflower seeds also contain Vitamin E[cxc].

Vitamin C can be found in rosehips, strawberries, potato skins and all citric fruits.

Docosahexaenoic acid (DHA)

Docosahexaenoic acid (DHA) is an omega-3 fatty acid that is found most abundantly in fish[cxci]. Amazingly DHA contributes 30 – 50 % of the total fatty acid content of our brains[cxcii]. Once again this is yet another super nutrient that can decrease amyloid beta production[cxciii]. It also increases phosphatidylserine levels[cxciv]. Studies indicate that Omega-3 fatty acids block neurofibrillary tangle formation[cxcv].

To benefit from foods rich in omega-3 fatty acids, be sure to regularly eat organic walnuts, flax, and hemp seeds.

Deep sea fish such as Alaskan salmon, mackerel and sardines are also potent sources of these oils. Avoid farmed fish as these are usually grain fed. Another thing to keep in mind is that many of our oceans are full of toxic commercial products that have contaminated our seafood supply. This is why many health practitioners recommend that fish oils, such as cod liver oil, be taken in supplemental form[cxcvi]. Fish is the best solution if your sea water is not contaminated!

Vinpocetine

Vinpocetine is found in periwinkle plant and has been discovered to protect nerve cells, as well as increasing blood circulation to the brain. Other studies have shown that it also protects against excitotoxicity.

It has a reputable history in Eastern Europe, as a well-tolerated treatment for memory ailments. One study showed that taking 10 mg of this botanical three times daily is especially good at decreasing symptoms associated with vascular dementia[cxcvii].

Pyrroloquinoline quinone (PQQ)

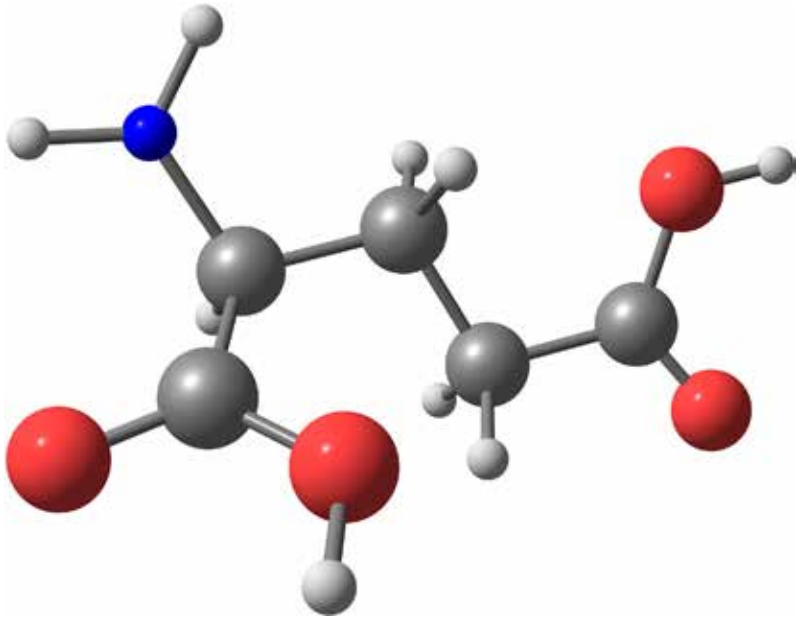
Pyrroloquinoline quinone (PQQ) is an incredibly potent health nutrient that stimulates the growth of new mitochondria in aging cells. It also can protect and repair mitochondria[cxcix].

Scientists believe that it may prevent Alzheimer's disease because apart from its mitochondrial healing effects it is also able to protect against amyloid-beta deposits[cc]. PQQ supplementation with 20 mg per day caused significant cognitive improvement in patients. This effect was amplified[cci] when PQQ was taken together with 300mg of CoQ10.

Phosphatidylserine

Phosphatidylserine (PS) is a natural constituent of our cell membranes. Research has found that supplementation with PS dramatically improves memory functions in those with memory impairment[ccii].

Recent research[cciii] has demonstrated that supplementing PS with omega-3 fatty acids, especially DHA, gives optimal results due to combined synergistic effects.



Vinpocetine

Resveratrol is a phytochemical superstar due to its amazing healing properties. This potent stilbene polyphenol is what imparts the color and aroma to grapes and red wine. It is found in some of the highest concentrations in boiled peanuts, blueberries, and Japanese knotweed. This incredible nutrient soared to global prominence after scientists discovered it had phenomenal anti-aging benefits.

Resveratrol has been shown to[cciv]:

- Reduce amyloid beta levels
- Reduce neurotoxicity
- Decrease cell death
- Protect the hippocampus from degenerating
- Prevents learning impairment
- Improves coordination and balance
- Neutralize free radical damage and protects nerve cells

One study demonstrated a lower rate of dementia and Alzheimer's disease associated with a regular intake of a glass of red wine[ccv]. Red wine polyphenol antioxidants have also been reported as halting the degenerative process of AD[ccvi].

Grape Seed Extract

Grape seed extract is a well-known natural therapy, used traditionally to treat cardiovascular disease due to its high procyanidin antioxidant profile. Studies[ccvii] have shown it to be highly protective against amyloid beta damage. Grape Seed extract can slow the progression of Alzheimer's disease, in addition to providing a vast array of extra health benefits.

Magnesium

Magnesium is an essential mineral needed as a co-factor for many enzyme functions. It's also necessary for NMDA glutamate receptors to work during memory processing[ccviii].Magnesium is particularly important for long-term memory.

It's well documented that a magnesium deficiency can cause mental dysfunction[ccix]. Studies have found that imbalances in serum magnesium levels cause cognitive impairment[ccx].

B Vitamins

Alzheimer's disease (AD) and mild cognitive impairment (MCI) are both linked to low levels of Vitamin B6, B12, and folate[ccxi] and increased homocysteine levels[ccxii].

Vitamin B12:
Low B12 levels are associated increased cognitive deterioration[ccxiii] and twice the risk of developing Alzheimer's disease within three years[ccxiv].

Vitamin B6:
Low levels are associated with an increase in brain lesions in patients with Alzheimer's disease[ccxv].

Folate:
Folate is essential to make DNA[ccxvi]. Low levels are strongly related to cognitive impairment[ccxvii].

Niacin

Increased levels of daily dietary niacin (vitamin B3) reduces mental decline by 70 %.

Niacin has been reported as being protective against Alzheimer's disease.

Coenzyme Q10 (Co-Q10)

Co-Q10 is a cofactor used by many chemical reactions in our cells. It is only found in organ meats and in some fish – sardines are a good source. It appears to be a vital antioxidant for protecting our brains from aging and AD.

It has shown increased benefits when combined with other nutrients known to prevent brain degeneration[ccxviii].

CoQ10 beneficial properties include:

- Negating the effects of mitochondrial dysfunction
- Decreasing excessive production of amyloid beta[ccxix]
- Potentially prevents amyloid deposits[ccxx]
- Improving memory, attention span and behavior in AD patients[ccxxi]
- Improving energy production by reducing oxidative stress
- Improving immune function[ccxxii]

N-acetyl cysteine

N-acetyl cysteine (NAC) is a precursor to glutathione - a super potent antioxidant. Many studies have associated neurodegenerative diseases with glutathione deficiency[ccxxiii].

Glutathione levels are boosted by taking NAC, and this protects against oxidation, particularly in the brain. In another study, oxidative damage was alleviated with improved mental performance when taken regularly[ccxxiv].

Blueberry Extract

Berry important news! Blueberries have recently astonished scientists when they discovered they contain polyphenols that reverse cognitive and motor dysfunction[ccxxv]. In addition, it stimulates new brain cell growth and improves neuroplasticity or the ability to adapt to change. These results were seen in the hippocampus, which is the site where memory is processed and largely affected by Alzheimer's[ccxxvi]. Blueberries were rated as the top antioxidant in terms of their potent ability to neutralize free radicals[ccxxvii].

Most of us love these berries, so it's not difficult to protect ourselves by taking a handful daily. It's important to eat a variety of berries, as they are all potent antioxidants. Eating a variety in combination causes an increase in many health benefits.

Luteolin

Luteolin is another polyphenol found in fruits and vegetables, such as green peppers, carrots, and celery. It is an effective protector against Alzheimer's. Initial studies have reported a significant decrease in amyloid beta levels and a reduction in neurofibrillary tangles. These are merely a few reasons to increase these nourishing colorful foods into your daily diet.

Wild Green Oat Extract

Wild green oat extract is derived from Avena sativa L.,a powerful and natural MOA-B (mono-amine-oxidase beta inhibitor)[ccxxviii]. This property causes increased Dopamine levels in the brain and decreases oxidative stress in nerve cells, providing a remarkable nutrient to combat dementia naturally[ccxxix]

This is an exceptional property considering the fact that Alzheimer's patients have approximately three times the amount of MOA-B activity as compared to healthy folk[ccxxx]. Other benefits that MOA-B inhibitors offer is a reduction of amyloid beta, improved mental function, and enhanced memory, making this extract a potent anti-Alzheimer's supplement[ccxxxi]. Consult a natural medical physician for proper dosing.

Nicotinamide Riboside

Nicotinamide riboside is an active source of vitamin B3 that is used to moderate cellular metabolism and DNA transcription[ccxxxii] . This substance can alter energy levels, protect DNA from harmful changes, offer stress resistance, increase survival and defer harmful factors that contribute to pathological aging[ccxxxiii]. Research studies are heaping praises on this amazing vitamin compound for the incredible array of potent anti-aging benefits it confers[ccxxxiv].

Bacopamonnieri – (Brahmi) [ccxxxv]

This herb has been used for centuries by Ayurvedic practitioners to treat memory problems, hypothyroidism, fatigue, anxiety, and stress[ccxxxvi]. Two decades ago, scientists discovered that it also has powerful anti-cancer properties. In addition, this herb has been shown to increase nerve cell communication and decrease amyloid plaques. Both actions support optimal brain health. 300mg daily is considered a safe dose[ccxxxvii].

CHAPTER 6

NEW HORIZONS – RE-INVENTING OURSELVES

The scientific research we have reviewed in this book provide compelling evidence for making healthy nutritional choices that will benefit and protect us. Equally as important are lifestyle strategies that we now know provide a way to ensure a healthy, quality-filled life. Here are some of the main lifestyle factors research shows are critical to supporting optimal brain health.

Exercise – a powerful reformer

If you do not exercise much, you may be resistant to the idea of it – that is, until you read about its powerful anti-aging effects! Some of the benefits include decreasing cardiovascular disease risk and reducing stress. A new understanding is that all types of exercise create new connections in the brain. This is exciting because science now suggests that exercise is correlated with increased nerve cell growth in the exact area that regulates memory – the hippocampus. Even gentle exercise, such as walking, increases neuronal growth in the hippocampus[ccxxxviii].

Exercise improves learning and memory skills. Most importantly, exercise generates the formation of mitochondria, which enhances energy production in our cells. Using our muscles improves blood flow throughout our body. It helps the immune system by clearing the lymphatic vessels and clears toxins from our system. Additionally, it is associated with improved moods and well-being[ccxxxix].

To benefit from exercise, experts recommend a routine of at least 20 - 30 minutes for five days a week.

Learn something new - activate your curiosity

Our brains enjoy novelty, and now we know that while we are learning, we are naturally protecting ourselves from disease by keeping the brain more active. 'Neurons that fire together are wired together' is the catch phrase coined by neuroscientists. What they are saying is: when we use different pathways, we are increasing neuroplasticity, ensuring that our mental functions are robust as we age.

Medical experts recommend activities such as learning a new instrument, or taking up a new hobby. Even practicing crosswords or Sudoku will produce the desired effect. The key is to find new areas that interest you. People report feeling more alive when they are learning something new that they enjoy! Make it a goal to learn something new daily – reactivate your curiosity!

Avoid toxic chemicals as much as possible

It's not possible to avoid all toxins in our lives; but to protect ourselves from developing Alzheimer's, we need to at least attempt to avoid pesticides and harmful chemicals. Many products used in agricultural practices have not only been linked to massive bee deaths but also to brain cell death. Increased exposure to toxins resulted in a whopping 53% increased risk of developing AD.

Many toxins affect mitochondrial function by increasing free radical formation and oxidative stress. Finding ways to detox from contaminants and avoiding products that are known to be toxic, is at the very least a lifesaving and sensible strategy to adopt for healthy living.

Avoid Lead and Aluminium

These metals are both severely toxic for our bodies and our brains. Both have been linked to Alzheimer's and lead has been connected to Parkinson's disease. It has long been established that lead has serious negative effects on children's brains. Lead has also been associated with degenerative brain processes as people age.

PCBs (Polychlorinated Biphenyls) [ccxl]

This substance, although having been banned in the 70s, is found throughout our modern environments and contributes negatively to Alzheimer's Disease.

PCBs are not easily broken down and cluster in parts of the brain. Even low-level exposure is enough to cause serious damage, according to some scientists.

Remove Chronic Stress and Anxiety from Your Life

Don't worry, be happy! Stress is directly linked to creating Alzheimer's. A study carried out on mice revealed that highly stressed mice had terrible memory. These stressed mice also had more beta-amyloid proteins found inside their brains[ccxli].

A 2010 study showed that middle-aged people with high levels of stress are more likely to develop AD than those with normal stress levels. They were also 65% more likely to develop Dementia. This 35-year study was carried out on Swedish women and later published in the Brain Science Journal.

Depression can raise your risk of Alzheimer's disease [ccxlii]

Depression has a significant effect on our bodies and the structure of our brains, physically altering the structure in a negative way. This structural change leaves us with a 50% increased risk of developing Alzheimer's. The deformation occurs because depression prevents blood from entering the brain. Over time, this decreased blood flow leads to further AD symptoms - until it's full blown.

Try your best to be as happy as possible and enjoy life as much as you can! It's not always necessary to stress or feel depressed.

Activities like meditating, socializing and exercise can help you reduce depression and anxiety. Eating nutritiously also has a role to play in fighting depression, as you need the right sources of nutrients to make happy chemicals!

New Horizons – Meditation is the art of re-inventing ourselves

Meditation is an excellent health-booster that has been revered for thousands of years to still the mind and hone mental clarity. Medically, it has an impressive repertoire of being able to lower blood pressure, stress and pain caused by a variety of conditions.

The Alzheimer's Research and Prevention Foundation (ARPF) completed a study with the University of Pennsylvania on how meditation affects memory loss. In this study, a group of individuals performed

KirtanKriya (a Kundalini Yoga traditional meditation) for 12mins a day for eight weeks. After the eight weeks, they observed increased brain activity in areas that dominate memory.

The participants had dramatically improved cognitive and memory function, as well as selective attention and focus. It also reduced anxiety, depression, cortisol and increased their immune functions![ccxliii]

Avoid Processed Foods and Alcohol

No matter how old you are, your body needs a good quality organic whole food diet, rich in nutrients from fruits, vegetables, nuts, and seeds to keep healthy. However, all food is not healthy for your brain.

Studies have linked fast food to the development of Alzheimer's. They revealed how fast food deforms the brain and creates those changes in chemicals that are associated with the development of brain disease. While most people understand the risks related to fast food if you are diabetic, many do not realize that patients with AD also have a problem with insulin much like diabetics. Foods that help regulate insulin benefit both diabetics and those with Alzheimer's. A diet that is low in sugar and has no refined/processed foods is ideal.

Fast food has an unfathomably high level of nitrates, additives, and preservatives which are all linked to a more rapid development of AD. We are also exposed to nitrates via rubber, latex, fertilizers and pesticides. Fast food additives such as aspartame and neurotoxins like MSG are devastating to our brain health and especially devastating for those with Alzheimer's. Alcohol is also highly not recommended for those looking to prevent Alzheimer's or dementia, as it already is known to cause memory loss and shrink the brain after prolonged drinking.

The Mediterranean Diet

People living in the Mediterranean have caught the attention of scientists due to multiple studies indicating that the inhabitants of this region experience less prevalence of Alzheimer's disease as well as a marked reduction in cardiovascular disease. In fact, their diets had significantly lower statistics for most dementias and brain degenerative diseases[ccxliv] including Parkinson's and MCI (Mild Cognitive Impairment).

In some studies, AD risk was decreased when people partook in a diet that was rich in fruits, vegetables, fish, nuts, and legumes and had lower intakes of meats, high-fat dairy, and sweets[ccxlv]. The Mediterranean diet not only reduced the risk of AD but also slowed the rate of progression from pre-dementia to brain degenerative disease states.

In one study, people who adopted the Mediterranean diet most closely, showed a 28% decrease in developing mental impairment disorders compared to those that did not adhere strictly to the diet[ccxlvi]. This study also showed that participants who were diagnosed with MCI at the onset of the study demonstrated a 48% risk reduction in developing AD after staying on the Mediterranean diet for 4 years[ccxlvii].

Yet, other studies have shown that Alzheimer's patients that strictly observed the Mediterranean diet lived longer lives – increasing their lifespan by an average of 3.9 years. Those that followed the diet moderately lived an average of 1.3 years more[ccxlviii].

The Mediterranean diet is made up of healthy foods such as fish, vegetable oils, non-starchy vegetables, low glycemic index fruits, and red wine, all of which have been independently documented as being potential factors for combating dementias and AD[ccxlix].

The Ketogenic Diet

There has been much interest in the neuroprotective effects of the Ketogenic diet. The diet involves following a strict protocol of consuming foods high in fats combined with moderate protein intake and limited carbohydrates. The diet causes the body to switch from the normal metabolic process of burning glucose to an alternate route of burning ketones as a source of fuel. This process may have beneficial effects on the brain. We generate ketones when the body breaks down fat, and once we trick our bodies to produce ketones, they can be used to supply energy.[ccl]

The initial research appears promising, with the ketogenic diet producing a decrease in amyloid beta levels after a 6-week period one in animal study. More research is needed, however, as ketogenic diets are also associated with some adverse side effects[ccli]. Side effects may include increased cholesterol, kidney stones, and possible gastro-esophageal reflux[cclii].

The Caloric Restriction Diet

Some research has shown that restricting caloric intake increases longevity and confers protective properties against developing degenerative brain disorders[ccliii]. One study showed that an increase in in caloric intake was correlated with double the risk of developing MCI – Mild Cognitive Impairment. The association was caloric dependent with low caloric intake showing the lowest incidence of cognitive dysfunction in the aged population studied[ccliv].

The Paleo Diet

A diet rich in vegetables, fruits, nuts & seeds, olives, fish & seafood, lean grass fed meats, eggs, and legumes that also limits dairy products, cereals, added salt, processed foods, refined fats and sugar has significant health benefits. The Paleo Diet includes foods that are highly anti-inflammatory, anti-oxidant and protective for brain health.

Natural scientific solutions

After reviewing the research around diet and brain degeneration, one thing is clear – diet remains the largest contributor to our aging naturally. It also offers significant protection to grow old without neurodegenerative disease crippling our worlds and burdening our loved ones with taking care of us when we are no longer capable.

As we now know, consistently maintaining a healthy lifestyle is a priority for brain health, so we need to find a diet that is both functional and effective. We need a diet that is both easy to follow and enjoyable.

We have looked at many of the foods that offer dramatic anti-aging effects, and if we increase these sources in our nutritional lifestyle, then we will achieve our healthy goals.

Theoretically, we should need nothing else other than our diets and healthy lifestyle factors to succeed at creating a new paradigm of growing old, one that is creative, active and productive with enhanced energy and vital functions intact. The research is compelling – we don't have to let ourselves suffer...

Dietary considerations for optimizing memory and preventing brain degeneration

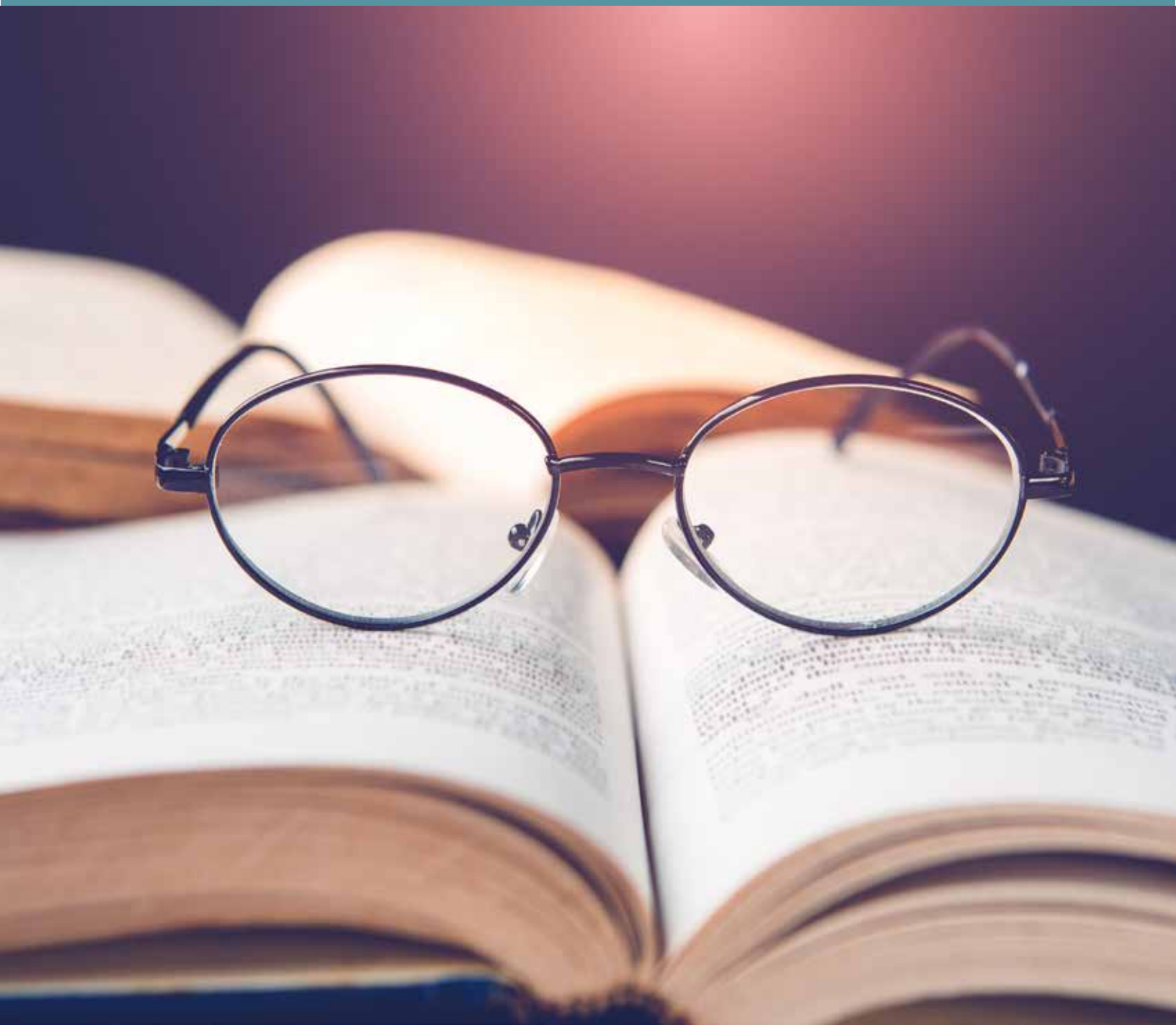
These recommendations are the basis for the Memory Magnetizer Diet

- Eat a diet rich in polyphenol antioxidants
- Eat foods rich in choline
- Eat healthy fats that decrease inflammatory pathways in the body
- Avoid saturated and trans fatty acids, especially hydrogenated fats
- Balance omega 3 to omega 6 fatty acids ratio. Reduce animal fats and increase vegetable and nut oils.
- Avoid simple sugars and increase complex carbohydrates that have low glycemic index properties to keep a stable blood sugar balance
- Avoid refined, processed foods – choose whole foods wherever possible and increase raw food content – consider adding smoothies to your diet
- Avoid commercial sweeteners that contain aspartame
- Eat herbs and spices that are known to chelate metals
- Avoid colorants and additives such as MSG, which is known to clog efficient mitochondrial function
- Avoid genetically modified foods – eat natural, organically grown foods
- Avoid animal products where animals are grain fed – choose free range products
- Try to eat locally produced foods – not only can you support your community, but you can also ensure healthy agricultural practices
- Read food labels – it's important to know what you are taking into your body
- Eat several smaller meals throughout the day to keep blood sugar levels stable and increase energy efficiency
- Drink plenty of fresh spring or filtered water – at least 6 – 8 glasses daily
- Avoid drinking fruit juice unless the pulp has been added back to the juice. Berry juices contain the highest amount of brain enhancing polyphenols. Include a glass of red wine at least 5 days of the week and enjoy organic filtered coffee in the mornings or indulge in a mug of cocoa
- Don't forget to include green tea
- Try to avoid soy products that are commercially produced unless they have been fermented properly- they are addictive and impact negatively on working memory

- Combine a variety of different colored foods into your diet on a daily basis
- Increase seeds and nut consumption

PART 2

MEMORY REPAIR PROTOCOL 21 DAY DIET PROTOCOL



Day 1	21 Day Diet Plan
First thing	Water with a dash of lemon - hot or cold
Breakfast	Turmeric smoothie , Filter Coffee
Mid am	Handful mixed nuts and seeds , Green Tea Dark Chocolate - 1 block
Lunch	Grilled Fish with Berry Sauce , steamed vegetables Grapefruit
Mid afternoon	Handful mixed berries
Dinner	Tuna & Carrot Salad , Avocado and carob mousse Glass red wine
hour before bed	1 Fruit - banana , Hot cocoa and cinnamon drink
Notes:	Take 1 Vitamin E soft gel with a main meal (400 IU) Take 1 Omega-3 tablet with a main meal

Day 2 — 21 Day Diet Plan

First thing	Water with a dash of lemon - hot or cold
Breakfast	Blueberry protein pancakes , Filter coffee
Mid am	Handful mixed nuts & seeds , Green Tea Dark Chocolate - 1 block
Lunch	Chicken Stew , Green salad , Papaya with lemon All Red Smoothie
Mid afternoon	Handful mixed nuts
Dinner	Chopped fresh vegetables , Homemade almond buttter Glass red wine
hour before bed	1 Fruit - banana , Hot cocoa and cinnamon drink
Notes:	Take 1 Vitamin E soft gel with a main meal (400 IU) Take 1 Omega-3 tablet with a main meal

Day 3	21 Day Diet Plan
First thing	Water with a dash of lemon - hot or cold
Breakfast	Green superfood smoothie , Filter coffee
Mid am	Handful mixed nuts and sunflower seeds Green Tea , Dark Chocolate - 1 block
Lunch	Carrot & coriander soup
Mid afternoon	
Dinner	Chicken stew , Red grape and pommegranate juice
hour before bed	1 Fruit - banana , Hot cocoa and cinnamon drink
Notes:	Take 1 Vitamin E soft gel with a main meal (400 IU) Take 1 Omega-3 tablet with a main meal

Day 4 — 21 Day Diet Plan

First thing	Water with a dash of lemon - hot or cold
Breakfast	Pineapple & coconut smoothie , Filter Coffee
Mid am	1 Fruit- apple ,Handful mixed nuts and sunflower seeds
Lunch	Chicken curry salad
Mid afternoon	
Dinner	Raw sushi , Glass red wine or grape juice Dark Chocolate - 1 block
hour before bed	1 Fruit - banana , Hot cocoa and cinnamon drink
Notes:	Take 1 Vitamin E soft gel with a main meal (400 IU) Take 1 Omega-3 tablet with a main meal

Day 5	21 Day Diet Plan
First thing	Water with a dash of lemon - hot or cold
Breakfast	Blueberry Protein Pancakes , Filter coffee Glass water
Mid am	Celery sticks with homemade almond butter Green Tea , Dark Chocolate - 1 block
Lunch	Savory berry sauce & salmon , Turmeric smoothie
Mid afternoon	Handful of mixed nuts
Dinner	Raw Sushi ,Apple & cinnamon crumble Glass red wine or grape juice
hour before bed	1 Fruit - banana ,Hot cocoa and cinnamon drink or herb tea
Notes:	Take 1 Vitamin E soft gel with a main meal (400 IU) Take 1 Omega-3 tablet with a main meal

Day 6 — 21 Day Diet Plan

First thing	Water with a dash of lemon - hot or cold
Breakfast	2 Boiled Eggs , Low-carb cracker , Filter coffee
Mid am	Turmeric smoothie Handful mixed nuts and sunflower seeds
Lunch	Sweet potato salad , Coffee or tea , Water
Mid afternoon	
Dinner	Carrot & coriander soup , 1 low-carb cracker Olive tapenade Glass red wine or grape juice
hour before bed	1 Fruit - banana ,Hot cocoa and cinnamon drink or herb tea
Notes:	Take 1 Vitamin E soft gel with a main meal (400 IU) Take 1 Omega-3 tablet with a main meal

Day 7	21 Day Diet Plan
First thing	Water with a dash of lemon - hot or cold
Breakfast	2 poached eggs and 1 low-carb cracker Filter Coffee ,Water
Mid am	Handful mixed nuts and sunflower seeds
Lunch	Carrot & coriander soup ,Green / herb tea , Water
Mid afternoon	Handful mixed nuts and sunflower seeds Green Tea , Dark Chocolate - 1 block
Dinner	Chicken Stew , Cauliflower rice Red grape and pomegranate juice
hour before bed	1 Fruit - banana ,Hot cocoa and cinnamon drink or herb tea
Notes:	Take 1 Vitamin E soft gel with a main meal (400 IU) Take 1 Omega-3 tablet with a main meal

Day 8 — 21 Day Diet Plan

First thing	Water with a dash of lemon - hot or cold
Breakfast	All Red Smoothie , Filter Coffee , Water
Mid am	Handful mixed nuts and sunflower seeds
Lunch	Fruit salad , Green / herb tea , Water
Mid afternoon	Dark chocolate - 1 block , Green Tea
Dinner	Garlic & Rosemary streak ,Glass red wine or grape juice Apple & Cinnamon Crumble
hour before bed	1 Fruit - banana ,Hot cocoa and cinnamon drink or herb tea
Notes:	Take 1 Vitamin E soft gel with a main meal (400 IU) Take 1 Omega-3 tablet with a main meal

Day 9	21 Day Diet Plan
First thing	Water with a dash of lemon - hot or cold
Breakfast	Turmeric smoothie , Filter coffee , Water
Mid am	2 low-carb crackers with homemade almond butter Green Tea , Dark Chocolate - 1 block
Lunch	Sweet potato salad ,Handful mixed nuts and sunflower seeds
Mid afternoon	Fruit and seeds , Dark Chocolate- 1 block
Dinner	Raw Sushi Apple & cinnamon crumble Glass red wine or grape juice
hour before bed	1 Fruit - banana
Notes:	Take 1 Vitamin E soft gel with a main meal (400 IU) Take 1 Omega-3 tablet with a main meal

Day 10 — 21 Day Diet Plan

First thing	Water with a dash of lemon - hot or cold
Breakfast	2 boiled eggs & sundried tomato tahini & low carb cracker Filter coffee
Mid am	Get up and Go smoothie Handful mixed nuts and sunflower seeds
Lunch	Creamy Asparagus Soup , 1 apple , Coffee or tea , Water
Mid afternoon	Turmeric smoothie , Dark Chocolate - 1 block Grapefruit
Dinner	Turmeric chicken & 'rice ' , Avocado and carob mousse Glass red wine or grape juice
hour before bed	1 Fruit - banana
Notes:	Take 1 Vitamin E soft gel with a main meal (400 IU) Take 1 Omega-3 tablet with a main meal

Day 11 — 21 Day Diet Plan

First thing	Water with a dash of lemon - hot or cold
Breakfast	Green superfood smoothie , Filter coffee , Water
Mid am	Superfood power balls
Lunch	Tuna & carrot salad , Carrot and veggie juice , Water
Mid afternoon	Handful mixed nuts and sunflower seeds , Green Tea Dark Chocolate - 1 block
Dinner	Chicken Stew & cauliflower rice , Fresh fruit and nuts Red grape and pomegranate juice
hour before bed	1 Fruit - banana
Notes:	Take 1 Vitamin E soft gel with a main meal (400 IU) Take 1 Omega-3 tablet with a main meal

Day 12 — 21 Day Diet Plan

First thing	Water with a dash of lemon - hot or cold
Breakfast	Pineapple coconut smoothie , Filter coffee , Water
Mid am	Handful mixed nuts and sunflower seeds
Lunch	Curry chicken salad , Apple , Green / herb tea ,Water
Mid afternoon	Chocolate berry smoothie Nuts and seeds
Dinner	Savory berry sauce and grilled salmon Glass red wine or grape juice , Fruit salad with coconut cream
hour before bed	1 Fruit - banana
Notes:	Take 1 Vitamin E soft gel with a main meal (400 IU) Take 1 Omega-3 tablet with a main meal

Day 13	21 Day Diet Plan
First thing	Water with a dash of lemon - hot or cold
Breakfast	2 low-carb crackers with almond butter Filter organic coffee , Water
Mid am	Superfood power balls , Green Tea , Dark Chocolate - 1 block
Lunch	Creamy asparagus soup , Grapefruit , Papaya with lemon
Mid afternoon	Handful mixed nuts and sunflower seeds
Dinner	Turmeric Chicken & cauliflower rice , Apple Glass red wine or grape juice
hour before bed	1 Fruit - banana , Hot cocoa and cinnamon drink or herb tea
Notes:	Take 1 Vitamin E soft gel with a main meal (400 IU) Take 1 Omega-3 tablet with a main meal

Day 14 — 21 Day Diet Plan

First thing	Water with a dash of lemon - hot or cold
Breakfast	Chocolate berry smoothie , Filter organic coffee , Water
Mid am	Green Tea ,Handful mixed nuts and sunflower seeds
Lunch	Creamy asparagus soup , Grapefruit , Coffee or tea , Water
Mid afternoon	All Red smoothie , Dark Chocolate - 1 block
Dinner	Cauliflower tabouli ,Avocado and carob mousse Glass red wine or grape juice
hour before bed	1 Fruit - banana , Hot cocoa and cinnamon drink or herb tea
Notes:	Take 1 Vitamin E soft gel with a main meal (400 IU) Take 1 Omega-3 tablet with a main meal

Day 15	21 Day Diet Plan
First thing	Water with a dash of lemon - hot or cold
Breakfast	Turmeric smoothie , Filter Coffee , Water
Mid am	Handful mixed nuts
Lunch	Italian Turkey Salad , Water
Mid afternoon	Handful mixed nuts and seeds , Green Tea Dark Chocolate - 1 block
Dinner	Chicken stew and cauliflower rice , Fresh fruit and nuts Red grape and pommegranate juice
hour before bed	1 Fruit - banana , Hot cocoa and cinnamon drink or herb tea
Notes:	Take 1 Vitamin E soft gel with a main meal (400 IU) Take 1 Omega-3 tablet with a main meal

Day 16 — 21 Day Diet Plan

First thing	Water with a dash of lemon - hot or cold
Breakfast	Blueberry protein pancakes , Filter Coffee , Water
Mid am	Turmeric smoothie
Lunch	Cauliflower tabouli , Apple , Green / herb tea , Water
Mid afternoon	Handful mixed nuts
Dinner	Turmeric chicken and cauliflower rice Glass red wine or grape juice , Avocado & carob mouse
hour before bed	1 Fruit - banana , Hot cocoa and cinnamon drink or herb tea
Notes:	Take 1 Vitamin E soft gel with a main meal (400 IU) Take 1 Omega-3 tablet with a main meal

Day 17	21 Day Diet Plan
First thing	Water with a dash of lemon - hot or cold
Breakfast	Grapefruit , 2 low-carb crackers and almond butter , Filter coffee
Mid am	Handful mixed nuts and seeds , Green Tea Dark Chocolate - 1 block
Lunch	Cauliflower tabouli , Steamed vegetables
Mid afternoon	Handful mixed berries
Dinner	Savory berry sauce and grilled salmon , Avocado and carob mousse , Glass red wine
hour before bed	1 Fruit - banana Hot cocoa and cinnamon drink
Notes:	Take 1 Vitamin E soft gel with a main meal (400 IU) Take 1 Omega-3 tablet with a main meal

Day 18 — 21 Day Diet Plan

First thing	Water with a dash of lemon - hot or cold
Breakfast	Papaya with lemon , 2 low-carb crackers & olive tapenade Filter coffee
Mid am	Chocolate berry smoothie ,Handful mixed nuts and sunflower seeds
Lunch	Creamy asparagus soup , Green Salad
Mid afternoon	Handful mixed berries
Dinner	Garlic and rosemary steak , Glass red wine
hour before bed	1 Fruit - banana , Hot cocoa and cinnamon drink
Notes:	Take 1 Vitamin E soft gel with a main meal (400 IU) Take 1 Omega-3 tablet with a main meal

Day 19	21 Day Diet Plan
First thing	Water with a dash of lemon - hot or cold
Breakfast	Get up and Go Smoothie , Filter coffee
Mid am	Handful mixed nuts and sunflower seeds , Green Tea Dark Chocolate - 1 block
Lunch	Curry chicken salad , Apple
Mid afternoon	
Dinner	Chicken Stew and cauliflower rice Red grape and pomegranate juice
hour before bed	1 Fruit - banana , Hot cocoa and cinnamon drink
Notes:	Take 1 Vitamin E soft gel with a main meal (400 IU) Take 1 Omega-3 tablet with a main meal

Day 20 — 21 Day Diet Plan

First thing	Water with a dash of lemon - hot or cold
Breakfast	Green superfood smoothie ,Filter coffee
Mid am	Handful mixed nuts and sunflower seeds
Lunch	Italian turkey salad , Chocolate berry smoothie
Mid afternoon	Handful mixed berries
Dinner	Chicken stew and cauliflower rice , Fruit Salad Glass red wine or grape juice
hour before bed	1 Fruit - banana , Hot cocoa and cinnamon drink
Notes:	Take 1 Vitamin E soft gel with a main meal (400 IU) Take 1 Omega-3 tablet with a main meal

Day 21	21 Day Diet Plan
First thing	Water with a dash of lemon - hot or cold
Breakfast	2 poached eggs and 1 cup spinach
Mid am	Handful mixed nuts and seeds ,Green Tea Dark Chocolate - 1 block
Lunch	Carrot and coriander soup , All red smoothie
Mid afternoon	Superfood power balls
Dinner	Turmeric chicken and cauliflower rice , Avocado and carob mousse Glass red wine
hour before bed	1 Fruit - banana , Hot cocoa and cinnamon drink
Notes:	Take 1 Vitamin E soft gel with a main meal (400 IU) Take 1 Omega-3 tablet with a main meal

RECIPES

Smoothie Recipes

Method

For all smoothies, simply place the ingredients into a blender and you're done. Smoothies are a super energizing and powerful health meal.

Turmeric Smoothie

Ingredients

- 1 cup coconut milk
- 1/2 cup frozen pineapple or mango chunks
- 1 fresh banana
- 1 tablespoon coconut oil
- 1 teaspoon turmeric (can be increased to 1 tsp)
- 1/2 teaspoon cinnamon
- 1/2 teaspoon ginger
- 1 teaspoon chia seeds (optional)
- 1 teaspoon maca (optional)

Berry 'get up and go' Smoothie Recipe

Ingredients

- 1/2 cup frozen pineapple
- 1 medium beetroot
- 1 red apple
- 1/2 cup chopped celery
- 1 cup water

Chocolate Berry Smoothie

Ingredients

- 1 cup frozen Blueberries
- 2 teaspoons cocoa powder
- 1 cup coconut milk
- ¼ teaspoon vanilla essence
- Pinch of cinnamon
- 2 teaspoons honey

Pineapple Coconut Smoothie

Ingredients

- 1 cup frozen pineapple
- ½ small banana, chopped
- ½ cup coconut milk
- ¼ cup orange juice
- 4 to 5 ice cubes
- 2 tablespoons shredded coconut
- 1 teaspoon honey

Hot Cocoa and Cinnamon Drink

Ingredients

- 2 tablespoons unsweetened cocoa powder
- 1 tablespoon chopped dark chocolate
- ½ teaspoon powdered stevia
- 1/8 teaspoon ground cinnamon
- Pinch salt
- 1 cup canned coconut milk

Method

Stir together the cocoa powder, dark chocolate, stevia, cinnamon and salt in a mug. Heat the milk in the microwave or on the stove until steaming. Pour the milk into the mug and stir everything together until fully combined. Enjoy the hot cocoa cinnamon drink while hot.

Green Superfood Smoothie

Ingredients

- 1 cup spinach
- 1 cup frozen mixed berries
- ½ cup coconut milk
- ½ cup water
- 2 teaspoons honey
- 1 green apple

All Red Smoothie

Ingredients

- 1 cup chopped red cabbage
- ½ red bell pepper
- 6 strawberries
- ½ cup raspberries
- ½ cup cranberries
- 1 cup water
- 1 tablespoon honey
- 1/4 cup ice cubes

SALAD

Tuna and Carrot Salad

Ingredients

- 3 cups grated carrots
- 1 can tuna, drianed
- ½ cup raisins
- ½ diced red onion
- 1 cup finely chopped parsley
- Salt and pepper to taste

Tahini Dressing

- ¼ cup fresh lemon juice
- ¼ cup tahini
- 1 clove garlic – crushed
- 2 tablespoons extra virgin olive oil
- 1 teaspoon honey
- 1 tsp cumin
- 1/8 teaspoon cayenne pepper
- ½ teaspoon salt
- 2 tablespoons finely chopped parsley
- Water to thin mixture if needed

Method

Whisk together all the dressing ingredients until smooth and adjust seasoning as required. If the mixture is too thick add water until the desired consistency is reached.

Toss all the salad ingredients together in a bowl with the dressing. Leftovers can be stored in the fridge for up to 5 days

Italian Turkey Salad

Ingredients

- 1 head kale
- 1 ½ cups cherry tomatoes – sliced in half
- ½ diced red onion
- 1/3 cup diced kalamata olives
- 1 finely chopped English cucumber
- 1 finely chopped parsley
- ¼ teaspoon crushed red pepper flakes
- Salt and pepper to taste
- ½ cup cooked turkey breast, chopped

Lemon Dressing

- 2 tablespoons fresh lemon juice
- ½ teaspoon cumin
- 1 clove garlic – crushed
- ½ teaspoon salt
- ¼ teaspoon extra virgin olive oil

Method

For the dressing, combine lemon juice, cumin, garlic and salt in a bowl. Drizzle in olive oil while whisking together. Season according to taste and then set it aside.

Add the remaining salad ingredients to a bowl and toss well. Drizzle the dressing evenly over the salad and season with salt and pepper. This recipe serves 4.

Homemade Almond Butter

Ingredients

- 3 cups whole almonds, raw
- 1 tsp coconut oil

Method

Pour almonds and oil into a good quality food processor. Pulse until the almonds turn to a pasty consistency.

Store in the refrigerator for 3 weeks, in an airtight jar

Sun-dried tomato tahini

Ingredients

- ¼ cup fresh lemon juice
- ½ cup tahini
- 1 clove garlic – crushed
- ¾ cup sun-dried tomatoes in oil that has been drained
- ¾ cup filtered water
- 1 tablespoon olive oil
- 1 tsp cayenne pepper
- Salt and ground black pepper to taste

Method

Place all ingredients into a blender with the water to create a smooth consistency. After tasting adjust seasoning to suit your palate and enjoy. The tahini can be stored in the fridge for 2 days in an airtight container.

Green Olive Tapenade

Ingredients

- 2 cups green olives, pitted and chopped
- ¼ cup almonds
- ¼ cup olive oil
- 2 crushed garlic cloves
- 1 tablespoon freshly chopped rosemary leaves

Method

Put all ingredients into a blender and blend until smooth. Place in an airtight container and store for up to one week in the fridge. This can be made with Kalamata olives and a little chilli added for a variation.

Chicken Stew

Ingredients

- 2 tablespoons olive oil
- 1 medium chopped red onion
- 1 crushed clove of garlic
- 2 teaspoons dried oregano
- 1 large can crushed Italian tomatoes
- 1 cup chicken broth
- 2 bay leaves
- Pepper to taste
- 1 cooked chicken - skinned and cut into bite sized pieces
- ½ cup Kalamata olives – chopped
- 1 tablespoon freshly squeezed lemon juice
- 1 tablespoon flaked almonds

Method

Heat olive oil and cook onion until softened for about 5 minutes. After adding garlic and oregano, continue to cook for 1 minute. Pour in the remaining ingredients except the chicken. After bringing the ingredients to a boil, cook for another 5 minutes. Finally, add the chicken pieces and simmer for an additional 10 minutes.

Remove the stew from the heat and add olives and lemon juice.

Sweet Potato Salad

Ingredients

- 2 cups romaine lettuce
- 2 medium sweet potatoes potatoes, boiled, diced
- 2 diced red bell peppers
- 1 tablespoon cumin
- Salt & pepper to taste
- Olive oil

Method

Sauté peppers in olive oil for 5 minutes. Place sweet potato, lettuce and peppers in large bowl with salt and pepper. Lastly, add olive oil to complete your delicious salad.

Avocado and Carob Mousse

Ingredients

- 2 Avocados,
- 2 tbs. honey
- 1/3 cup carob powder
- 1 tbs. coconut oil
- ½ tsp vanilla extract

Method

Place avocado flesh into a blender and add remaining ingredients. Blend until silky smooth.

Chill in the refrigerator for at least an hour. Serve with mint leaf garnish. The mousse will last in the refrigerator for up to 2 days.

Carrot and Coriander Soup

Ingredients

- 2 lb carrots, chopped lengthways
- 5 cloves garlic
- 2 handfuls fresh coriander, chopped
- 1 medium sweet potato
- 1 tsp cumin
- 1/2 tsp ginger
- 4 cups vegetable stock
- Salt and pepper to taste.

Method

Bring all ingredients to boil until vegetables are tender. Puree mix until smooth, and season to taste. Recipe serves 5.

Creamy Asparagus Soup

Ingredients

- 4 cups chicken stock
- 2 lb asparagus, ends removed & chopped
- 1 large onion, chopped
- 3 cloves garlic, chopped
- 1 tablespoon olive oil
- Salt and pepper to taste

Method

Sauté the onions and garlic in olive oil, until tender. Add the asparagus and chicken stock. Bring to a boil until tender. When the asparagus has cooked, blend the mix until smooth and serve hot. Season to taste.

Savory Berry Sauce and Grilled Salmon

Ingredients

- 1 tablespoon olive oil
- 1 garlic clove, thinly sliced
- 1/4 teaspoon salt
- 1/4 teaspoon chopped fresh thyme
- 1 cup fresh blueberries
- 1/4 cup water
- 1 tablespoon balsamic vinegar
- 4 salmon steaks / fillets with skin (each about 3/4 inch thick)
- 3 tablespoons thinly sliced fresh mint

Method

Heat olive oil, garlic, salt and thyme. Stir until fragrant, about 30 seconds then add blueberries, water and vinegar. Stir to blend. Mash berries while cooking until sauce thickens, stirring often, for 3 - 4 minutes. Season with freshly ground black pepper and remove from heat.

Brush salmon on both sides with olive oil; sprinkle with salt, thyme and black pepper.

Grill salmon for 4 to 5 minutes per side. Transfer to plates. Stir 2 tablespoons sliced mint into warm blueberry sauce. Spoon the sauce over the salmon and sprinkle with remaining mint. Serve immediately. Recipe serves four.

Cauliflower Tabouli Salad

Ingredients

- 1 head cauliflower
- 2 cloves garlic, finely chopped
- 1 bunch parsley, finely chopped
- 2 tablespoons olive oil
- 2 tablespoons lemon juice
- 1 tablespoon chopped mint
- 1 cup cherry tomato, finely chopped
- Salt and pepper to taste

Method

Process the cauliflower in a food processor until it resembles a rice consistency. Mix processed cauliflower with the remaining ingredients, combining the lemon juice and olive oil with the salad. Serve cold. Recipe serves 4.

Raw Sushi

Yields: 4 rolls of 8 pieces

Ingredients

- 4 Large Nori sheets
- Cauliflower Rice (see below)
- 1 avocado sliced
- 1/3 cucumber sliced
- 4 asparagus spears
- Coconut amino (or organic fermented soy sauce). Wasabi paste
- Pink Ginger

Method

To assemble sushi, lay one sheet of nori and cover with a thin layer of cauliflower “rice” being careful to leave few cm border on just one side of the nori. Then, lengthwise on top of the “rice” lay the avocado and cucumber slices. Place the asparagus spears in a line down the middle. Feel free to combine different ingredients as the mood takes you – be creative!

Carefully pick up the opposite end to the clear border of the rice and begin to roll over the ingredients, tugging tightly as you roll to keep the roll free of gaps in the middle. Continue to roll until hitting the border free of “rice” and squeeze the roll gently all along the length of it, shaping it round. “Glue” the border free of rice to the ready roll. Nori should be a little bit moist because of the cauliflower “rice”. However, if the nori won’t stick, you can moist the nori a bit with your fingers dipped in water.

Then, with a very sharp knife carefully cut the roll in half. Then cut each half in half again and once more cut the four pieces in half resulting in 8 small pieces. Arrange the pieces on a plate and serve with ginger, Wasabi and coconut amino / Braggs amino acids or quality non-GMO soy sauce.

Raw Cauliflower “rice”

Ingredients

- 1/2 head of a cauliflower
- 1 tbsp. raw Tahini
- 1 tsp seasoned apple cider vinegar
- 1/2 tsp pink Himalayan salt or sea salt

Method

Combine all ingredients into a food blender and blend until smooth.

Protein Blueberry Pancakes

Ingredients

- 3 eggs
- 1/4 cup almond milk
- 3 tablespoons coconut oil
- 1 tablespoon honey
- 2 scoops protein powder
- 1/3 cup almond flour
- 1/2 cup blueberries

Method

Whisk together eggs, milk, 2 tbsp coconut oil, honey and protein powder. Fold in almond flour and blueberries.

Melt 1 tbsp of coconut oil on hot skillet and cook pancakes in 1/3 cupfuls until golden on each side.

Serve with a drizzle of honey, and add fresh berries. Recipe serves 2

Curry Chicken Salad

Ingredients

- 2 chicken breasts, cut in strips
- 1 teaspoon curry powder
- 1 teaspoon curcumin
- ½ teaspoon cumin
- 1 tablespoon olive oil
- ½ red onion, diced
- 3 stalks celery
- 2 cups romaine lettuce
- Salt and pepper to taste
- 1 teaspoon lemon juice

Method

Heat olive oil in a skillet. Add onions and sauté for 2 minutes. Add chicken breast and spices. Cook thoroughly.

Remove the chicken from the heat, and combine with the romaine and chopped celery in a bowl.

Serve with a drizzle of lemon juice. Recipe serves 2.

Turmeric Chicken and Cauliflower rice

Ingredients

- 2 chicken breasts, cut in strips
- 2 cups cauliflower rice (see above recipe)
- 1 tablespoon turmeric
- 1 tablespoon cumin
- ½ tablespoon cayenne pepper
- 2 cloves garlic, minced
- 1 tablespoon coconut oil
- 1 can coconut milk
- Salt and pepper to taste
- 1 red bell pepper, sliced

Method

Heat coconut oil on a skillet on medium heat. Add chicken breasts, garlic, spices and salt & pepper. Cook thoroughly.

Add peppers and cook for a further 5 minutes. Mix in coconut slowly, and simmer for a few minutes.

Serve chicken with cauliflower rice. Recipe serves 3.

Garlic & Rosemary Steak

Ingredients

- 2 lean cuts of steak
- 2 sprigs rosemary
- 4 cloves garlic, chopped finely
- 1 tbsp olive oil
- 1 cup broccoli florets
- 1 cup cauliflower florets
- Salt and pepper to taste
- 1 tsp lemon juice

Method

Rub steak in olive oil, rosemary, lemon juice and garlic. Marinate for at least 30 minutes.

Heat a skillet on medium heat, and brown the steak for 4 minutes on each side. Season to taste

Serve with steamed cauliflower and broccoli florets. Recipe serves 2.

Apple & Cinnamon Crumble

Ingredients

- 2 green apples, diced
- 1 teaspoon cinnamon
- 1 tablespoon honey
- 1 cup walnuts, processed
- 2 tablespoons coconut oil

Method

Preheat the oven at 420 F. In a saucepan, combine apples, cinnamon, honey and 1 tablespoon of oil. Simmer on medium heat for a few minutes, until apples soften.

Process walnuts with a tablespoon of coconut oil until the mixture forms a crumble-like consistency.
Fill 4 individual sized ramekins with a layer of the apple mixture, followed by the walnut crumble. Broil the ramekins in the oven for a few minutes, until the top is turning golden brown. Serve warm. Recipe serves 4.

Superfood power truffles

Ingredients

- 1 cup dates, softened
- 2 tablespoons coconut oil
- 1 cup walnuts
- ¼ cup cocoa powder
- ¼ cup coconut, shredded

Method

In a food processor, combine all the ingredients until it forms a paste-like consistency.

Form the mixture into equal-sized balls. Refrigerate the balls on a baking tray for at least 4 hours. Recipe serves 10.

APPENDIX 1

FDA APPROVED DRUGS FOR DEMENTIA & ALZHEIMER'S

DRUG NAME	DRUG TYPE AND USE	HOW IT WORKS	COMMON SIDE EFFECTS
Aricept® (donepezil)	Cholinesterase inhibitor prescribed to treat symptoms of mild, moderate, and severe Alzheimer's	Prevents the breakdown of acetylcholine in the brain	Nausea, vomiting, diarrhea, muscle cramps, fatigue, weight loss
Exelon® (rivastigmine)	Cholinesterase inhibitor prescribed to treat symptoms of mild to moderate Alzheimer's (patch is also for severe Alzheimer's)	Prevents the breakdown of acetylcholine and butyrylcholine (a brain chemical similar to acetylcholine) in the brain	Nausea, vomiting, diarrhea, weight loss, indigestion, muscle weakness
Namenda® (memantine)	N-methyl-D-aspartate (NMDA) antagonist prescribed to treat symptoms of moderate to severe Alzheimer's	Blocks the toxic effects associated with excess glutamate and regulates glutamate activation	Dizziness, headache, diarrhea, constipation, confusion
Namzaric® (memantine extended-release and donepezil)	NMDA antagonist and cholinesterase inhibitor prescribed to treat symptoms of moderate to severe Alzheimer's (for patients stabilized on both memantine and donepezil taken separately)	Blocks the toxic effects associated with excess glutamate and prevents the breakdown of acetylcholine in the brain	Headache, nausea, vomiting, diarrhea, dizziness

Razadyne® (galantamine)	Cholinesterase inhibitor prescribed to treat symptoms of mild to moderate Alzheimer's	Prevents the breakdown of acetylcholine and stimulates nicotinic receptors to release more acetylcholine in the brain	Nausea, vomiting, diarrhea, decreased appetite, dizziness, headache

DRUG NAME	MANUFACTURER'S RECOMMENDED DOSAGE	FOR MORE INFORMATION
Aricept® (donepezil)	• Tablet*: Initial dose of 5 mg once a day • May increase dose to 6 mg/day (3 mg twice a day), 9 mg (4.5 mg twice a day), and 12 mg/day (6 mg twice a day) at minimum 2-week intervals if well tolerated • Patch*: Initial dose of 4.6 mg once a day; may increase dose to 9.5 mg once a day and 13.3 mg once a day at minimum 4-week intervals if well tolerated	For current information about this drug's safety and use, visit the **www.fda.gov/Drugs.** Click on "Drugs @ FDA," search for Exelon, and click on drug name links to see "Label Information."
Namenda® (memantine)	• Tablet*: Initial dose of 5 mg once a day • May increase dose to 10 mg/day (5 mg twice a day), 15 mg/day (5 mg and 10 mg as separate doses), and 20	Headache, nausea, vomiting, diarrhea, dizziness

Aricept® (donepezil)	mg/day (10 mg twice a day) at minimum 1-week intervals if well tolerated • Oral solution*: Same dosage as above • Extended-release capsule: Initial dose of 7 mg once a day; may increase dose to 14 mg/day, 21 mg/day, and 28 mg/day at minimum 1-week intervals if well tolerated	For current information about this drug's safety and use, visit **www.namenda.**com and **www.namendaxr.com**. See "Full Prescribing Information."
Namzaric® (memantine extended-release and donepezil)	• Capsule: 28 mg memantine extended-release + 10 mg donepezil once a day • 14 mg memantine extended-release + 10 mg donepezil once a day (for patients with severe renal impairment)	For current information about this drug's safety and use, visit **www.namzaric.com.** Click on "Full Prescribing Information" to see the drug label.
Razadyne® (galantamine)	• Tablet*: Initial dose of 8 mg/day (4 mg twice a day) • May increase dose to 16 mg/day (8 mg twice a day) and 24 mg/day (12 mg twice a day) at minimum 4-week intervals if well tolerated • Extended-release capsule*: Same dosage as above but once a day	For current information about this drug's safety and use, visit **www.janssenmd.com/razadyne.** Click on "full Prescribing Information" to see the drug label.

*Available as a generic drug.

Alzheimer's Disease Education and Referral (ADEAR) Center
A Service of the National Institute on Aging

National Institutes of Health
U.S. Department of Health and Human Services

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APPENDIX 2

THE GLYCEMIC INDEX

LOW GLYCEMIC INDEX (Less Than 55)

FRUITS
Apples
Apple juice
Apricots, dried
Bananas
Blueberries
Cherries
Coconut
Cranberries
Cranberry juice
Figs, dried

FRUITS
Grapefruit
Grapes
Orange juice
Oranges
Peaches
Pears, fresh
Plantains, raw
Plums
Strawberries

VEGETABLES
Artichokes
Asparagus
Bamboo shoots, raw
Beet greens
Broccoli
Broccoli rabe
Brussel sprouts
Butternut squash, baked
Cabbage, Chinese
Cabbage, savoy, boiled
Carrot juice
Carrots, raw
Cauliflower
Celery
Collard greens
Cucumber

FRUITS
Eggplant
Garlic
Green beans
Hubbard squash, baked
Kale
Leeks
Lettuce
Lima beans, baby, frozen
Okra, raw
Olives
Onions
Peppers
Pickles, dill
Turnip greens, boiled
Turnips, boiled
Snow peas
Summer squash
Tomato soup
Tomatoes
Spinach
Summer squash
Tomato soup
Tomatoes
Watercress
Zucchini

DAIRY AND DAIRY ALTERNATIVES
Soy milk
Almond milk

NUTS
Almonds
Cashews

SNACKS & SWEETS
Honey

MEDIUM GLYCEMIC INDEX (between 56 and 69)

FRUITS
Apricots, canned with light
syrup
Apricots, fresh
Cantaloupe
Fruit cocktail
Grapes
Mango juice, unsweetened
Mangoes
Oranges
Orange juice
Papaya, fresh
Peaches, fresh
Peaches, canned
Pineapple
Raisins

VEGETABLES
Sweet potato
Sponge cake
Sushi

HIGH GLYCEMIC INDEX (70 and higher)

FRUITS
Dates
Kiwifruit
Watermelon

VEGETABLES
Parsnips
Pumpkin
Rutabaga

DAIRY AND DAIRY ALTERNATIVES
Tofu
frozen dessert
non-dairy

http://universityhealthnews.com/daily nutrition/glycemic-index-chart/

[i] Kadakkuzha, Beena M; Akhmedov, Komolitdin (2013-12-14). "Age-associated bidirectional modulation of gene expression in single identified R15 neuron of Aplysia". BMC Genomics. **14** (1): 880. doi:10.1186/1471-2164-14-880. PMC 3909179 . PMID 24330282.

[ii] Craik, F.; Salthouse, T. (2000). The Handbook of Aging and Cognition (2nd ed.). Mahwah, NJ: Lawrence Erlbaum. ISBN 0-8058-2966-0. OCLC 44957002.

[iii] Craik, F.; Salthouse, T. (2000). The Handbook of Aging and Cognition (2nd ed.). Mahwah, NJ: Lawrence Erlbaum. ISBN 0-8058-2966-0. OCLC 44957002.

[iv] Raz, Naftali; et al. (2005). "Regional Brain Changes in Aging Healthy Adults: General Trends, Individual Differences and Modifiers". Cereb. Cortex. **15** (11): 1676–1689. doi:10.1093/cercor/bhi044. PMID 15703252.

[v] Raz, Naftali; Rodrigue, Karen M. (2006). "Differential aging of the brain: Patterns, cognitive correlates and modifiers" (PDF). Neuroscience & Biobehavioral Reviews. **30** (6): 730–748. doi:10.1016/j. neubiorev.2006.07.001. PMID 16919333.

[vi] Kolb, Bryan; Gibb, Robbin; Robinson, Terry E. (2003). "Brain plasticity and behavior". Current Directions in Psychological Science. **12** (1): 1–5. doi:10.1111/1467-8721.01210. ISSN 0963-7214.

[vii] Kolb, Bryan; Whishaw, Ian Q. (1998). "BRAIN PLASTICITY AND BEHAVIOR". Annual Review of Psychology. **49** (1): 43–64. doi:10.1146/annurev.psych.49.1.43. PMID 9496621.

[viii] Barnes, C.; Burke, S. (2006). "Neural plasticity in the ageing brain". Nature Reviews Neuroscience. **7** (1): 30–40. doi:10.1038/nrn1809. PMID 16371948.

[ix] Hof PR, Morrison JH (October 2004). "The aging brain: morphomolecular senescence of cortical circuits". Trends Neurosci. 27 (10): 607–13. doi:10.1016/j.tins.2004.07.013. PMID 15374672.

[x] Hof PR, Morrison JH (October 2004). "The aging brain: morphomolecular senescence of cortical circuits". Trends Neurosci. **27** (10): 607–13. doi:10.1016/j.tins.2004.07.013. PMID 15374672.

[xi] Sowell ER, Peterson BS, Thompson PM, Welcome SE, Henkenius AL, Toga AW (March 2003). "Mapping cortical change across the human life span". Nat. Neurosci. **6** (3): 309–15. doi:10.1038/nn1008. PMID 12548289.

[xii] Davis, P.; Morris, J.; et al. (1991). "The distribution of tangles, plaques, and related immunohistochemical markers in healthy aging and Alzheimer's disease". Neurobiology of Aging. **12** (4): 295–312. doi:10.1016/0197-4580(91)90006-6. PMID 1961359.

[xiii] Whalley LJ, Deary IJ, Appleton CL, Starr JM (November 2004). "Cognitive reserve and the neurobiology of cognitive aging". Ageing Res. Rev. **3** (4): 369–82. doi:10.1016/j.arr.2004.05.001. PMID 15541707.

[xiv] Keller JN, Schmitt FA, Scheff SW, et al. (April 2005). "Evidence of increased oxidative damage in subjects with mild cognitive impairment" (PDF). Neurology. **64** (7): 1152–6. doi:10.1212/01.WNL.0000156156.13641.BA. PMID 15824339.

[xv] Mobbs, Charles V.; Hof, Patrick R. (2009). Handbook of the neuroscience of aging. Amsterdam: Elsevier/Academic Press. ISBN 0-12-374898-4. OCLC 299710911.

[xvi] Kaasinen, V.; Vilkman, H.; Hietala, J.; Någren, K.; Helenius, H.; Olsson, H.; Farde, L.; Rinne, J. O. (2000). "Age-related dopamine D2/D3 receptor loss in extrastriatal regions of the human brain". Neurobiology of Aging. **21** (5): 683–688. doi:10.1016/S0197-4580(00)00149-4. PMID 11016537.

[xvii] Wang, E.; Snyder, S. D. (1998). Handbook of the aging brain. San Diego, California: Academic Press. ISBN 0-12-734610-4. OCLC 636693117.

(xviii) Iyo, M.; Yamasaki, T. (1993). "The detection of age-related decrease of dopamine, D1, D2 and serotonin 5-HT2 receptors in living human brain". Prog. Neuropsycopharmacol. Biol. Psychiatry. **17** (3): 415–421. doi:10.1016/0278-5846(93)90075-4. PMID 8475323.

(xix) Wong, D. F.; et al. (1984). "Effects of age on dopamine and serotonin receptors measured by positron tomography in the living human brain". Science. **226** (4681): 1393–1396. doi:10.1126/science.6334363. PMID 6334363.

(xx) RMarcusson, J.; Oreland, L.; Winblad, B. (1984). "Effect of age on human brain serotonin (S-1) binding sites". Journal of Neurochemistry. **43** (6): 1699–1705. doi:10.1111/j.1471-4159.1984.tb06098.x. PMID 6491674. neubiorev.2006.07.001. PMID 16919333.

(xxi) Kaiser LG, Schuff N, Cashdollar N, Weiner MW (May 2005). "Age-related glutamate and glutamine concentration changes in normal human brain: 1H MR spectroscopy study at 4 T". Neurobiol. Aging. 26 (5): 665–72. doi:10.1016/j.neurobiolaging.2004.07.001. PMC 2443746 . PMID 15708441.

(xxii) Sailasuta N, Ernst T, Chang L (June 2008). "Regional variations and the effects of age and gender on glutamate concentrations in the human brain". Magn Reson Imaging. **26** (5): 667–75. doi:10.1016/j.mri.2007.06.007. PMC 2712610 . PMID 17692491.

(xxiii) Chang L, Jiang CS, Ernst T (January 2009). "Effects of age and sex on brain glutamate and other metabolites". Magn Reson Imaging. **27** (1): 142–5. doi:10.1016/j.mri.2008.06.002. PMC 3164853. PMID 18687554.

(xxiv) Lezak, M.D; Howieson, D.B.; Loring, D.W. (2004). Neuropsychological Assessment (4th ed.). Oxford: Oxford University Press. ISBN 978-0-19-511121-7.

(xxv) Alverzo JP (2006). "A review of the literature on orientation as an indicator of level of consciousness". J Nurs Scholarsh. **38** (2): 159–164. doi:10.1111/j.1547-5069.2006.00094.x. PMID 16773920.

(xxvi) Ishizaki, J.; Meguro, K.; Ambo, H.; Shimada, M.; Yamaguchi, S.; Harasaka, C.; et al. (1998). "A normative community based study of mini-mental state in elderly adults: The effect of age and educational level.". Journal of Gerontology. **53**: 359–363.

(xxvii) Sweet, J.J.; Such, Y.; Leahy, B.; Abramowitz, C.; Nowinski, C.J. (1999). "Normative clinical relationships between orientation and memory: Age as an important moderator variable.". The Clinical Neuropsychologist. **13** (4): 495–508. doi:10.1076/1385-4046(199911)13:04;1-y;ft495.

(xxviii) Benton, A.L.; Eslinger, P.; Damasio, A. (1981). "Normative observations on neuropsychological test performances in old age.". Journal of Clinical Neuropsychology. **3**: 33–42. doi:10.1080/01688638108403111.

(xxix) Banich, M. T.; Compton, R. J. (2011). Cognitive neuroscience. Belmont,CA: Wadsworth.

(xxx) Light, L.L. (1991). "Memory and aging: Four hypotheses in search of data". Annual Review of Psychology. **42**: 333–376.doi:10.1146/annurev.ps.42.020191.002001.

(xxxi) Carrier, J. S. A.; Cheyne, A.; Solman, G. J. F.; Smilek, D. (2010). "Age trends for failures of sustained attention". Psychology and Aging. **25** (3): 569–574. doi:10.1037/a0019363. PMID 20677878.

(xxxii) Kensinger, E.A (2009). Cognition in aging and age related disease. In P. R. Hof & C. V. Mobbs (Eds.), Handbook of the neuroscience of aging (249-256). London: Elsevier Press.

(xxxiii) Portet, F.; Ousset, PJ.; Visser, PJ.; Frisoni, GB.; Nobili, F.; Scheltens, P.; Vellas, B.; Touchon, J. (Jun 2006). "Mild cognitive impairment (MCI) in medical practice: a critical review of the concept and new diagnostic procedure. Report of the MCI Working Group of the European Consortium on Alzheimer's Disease.". J Neurol Neurosurg Psychiatry. **77** (6): 714–8. doi:10.1136/jnnp.2005.085332. PMC 2077456 . PMID 16549412.

(xxxiv) Crosson, B., Garcia, A., Mcgregor, K., & Wierenga, C. E. (2013). The Impact of Aging on Neural Systems for Language. In M. F. G. Sandra Koffler, Joel Morgan, Ida Sue Baron (Ed.), Neuropsychology, Volume 1 (pp. 149–187). Oxford University Press.

(xxxv) Anderton BH (April 2002). "Ageing of the brain". Mech. Ageing Dev. **123** (7): 811–7. doi:10.1016/S0047-6374(01)00426-2. PMID 11869738

(xxxvi) Scarmeas, N.; Stern, Y. (2003). "Cognitive reserve and lifestyle". Journal of Clinical and Experimental Neuropsychology. **25** (5): 625–633. doi:10.1076/jcen.25.5.625.14576. PMC 3024591 . PMID 12815500

(xxxvii) Baker, L.D.; Frank, L.L.; Foster-Schubert, K.; Green, P.S.; Wilinson, C.W.; McTiernan, A.; et al. (2010). "Effects of aerobic exercise on mile cognitive impairment: A controlled trial". Archives of Neurology. **67** (1): 71–79. doi:10.1001/archneurol.2009.307

(xxxviii) Hall, C.B.; Lipton, R. B.; Sliwinski, M.; Katz, M. J.; Derby, C. A.; Verghese, J. (2009). "Cognitive activities delay onset of memory decline in persons who develop dementia". Neurology. **73** (5): 356–361. doi:10.1212/wnl.0b013e3181b04ae3.

(xxxix) Barnes, L. L.; Mendes de Leon, C.F.; Wilson, R. S.; Bienias, J. L.; Evans, D. A. (2004). "Social resources and cognitive decline in a population of older African Americans and whites".Neurology. **63** (12): 2322–2326.

(xl) Gabrieli, J.; Hedden, T. (2004). "Insights into the ageing mind: a view from cognitive neuroscience". Nature Reviews. **5** (2): 87–96. doi:10.1038/nrn1323. PMID 14735112.

(xli) Zhang Guo, Guo; Li, Juxue; Purkayastha, Purkayastha; Tang, Yizhe; Zhang, Hai; Yin, Ye; Li, Bo; et al. (2013). "Hypothalamic programming of systemic ageing involving IKK-[bgr], NF-[kgr] B and GnRH". Nature. **497**: 211–216. doi:10.1038/nature12143.

(xlii) http://www.apa.org/pi/aging/memory-and-aging.pdf

(xliii) https://www.nia.nih.gov/alzheimers/publication/alzheimers-disease-genetics-fact-

(xliv) Daviglus ML, et al. "Risk Factors and Preventive Interventions for Alzheimer's disease: State of the Science." Arch Neurol. 68.9 (2011): 1185-90

(xlv) Harrison FE. "A critical review of vitamin C for the prevention of age-related cognitive decline and Alzheimer's disease." J Alzheimer's Dis. 29.4 (2012): 711-26

(xlvi) Kalaria RN, Akinyemi R, and Ihara M. Does Vascular Pathology Contribute to Alzheimer Changes? [In Eng] J Neurol Sci. 2012 Aug 9

(xlvii) Blum S, et al. "Memory after Silent Stroke: Hippocampus and Infarcts Both Matter." Neurology. 78.1 (2012): 38-46

(xlviii) Massoud F, Gauthier S. Update on the pharmacological treatment of Alzheimer's disease. Curr Neuropharmacol. 2010 Mar;8(1):69-80

(xlix) Marchesi VT. "Alzheimer's disease 2012: the great amyloid gamble." Am J Pathol. 180.5 (2012): 1762-7

(l) Crespo-Biel N, Theunis C, and Van Leuven F. Protein tau: prime cause of synaptic and neuronal degeneration in Alzheimer's disease. Int J Alzheimer's Dis. 2012;2012:251426

(li) Muñoz-Torrero D. Acetylcholinesterase inhibitors as disease-modifying therapies for Alzheimer's disease. Curr Med Chem. 2008;15(24):2433-55

(lii) Nieoullon A. [Acetylcholinesterase inhibitors in Alzheimer's disease: further comments on their mechanisms of action and therapeutic consequences]. Psychol Neuropsychiatr Vieil. 2010 Jun;8(2):123-31

(liii) Dong-gyu J, et al. "Evidence That γ-Secretase Mediates Oxidative-Stress Induced β-Secretase Expression in Alzheimer's disease." Neurobiol Aging. 2010 Jun;31(6):917-25

[liv] Mandel S, et al. "Green Tea Catechins as Brain-Permeable, Natural Iron Chelators-Antioxidants for the Treatment of Neurodegenerative Disorders." Mol Nutr Food Res. 50.2 (2006): 229-34

[lv] Salminen A, et al. "Inflammation in Alzheimer's disease: Amyloid-β Oligomers Trigger Innate Immunity Defence via Pattern Recognition Receptors." Prog Neurobiol. 87.3 (2009):181-194

[lvi] Caselli RJ, Reiman EM. "Characterizing the Preclinical Stages of Alzheimer's Disease and the Prospect of Presymptomatic Intervention." J Alzheimer's Dis. (2012)

[lvii] Leuner K, Müller WE, and Reichert AS. From Mitochondrial Dysfunction to Amyloid Beta Formation: Novel Insights into the Pathogenesis of Alzheimer's Disease. Mol Neurobiol. 2012 Jul 26

[lviii] Danysz W, Parsons CG. "Alzheimer's disease, β-amyloid, glutamate, NMDA receptors and memantine – searching for the connections." Br J Pharmacol. 2012 May 30. doi: 10.1111/j.1476-5381.2012.02057

[lix] Barron AM, Pike CJ. "Sex hormones, aging, and Alzheimer's disease." Front Biosci (Elite Ed). 2012 Jan 1;4:976-97

[lx] Miklossy J. Alzheimer's disease - a neurospirochetosis. Analysis of the evidence following Koch's and Hill's criteria. J Neuroinflammation. 2011 Aug 4;8:90

[lxi] Honjo K, van Reekum R, Verhoeff NP. Alzheimer's disease and infection: do infectious agents contribute to progression of Alzheimer's disease? Alzheimer's Dement. 2009 Jul;5(4):348-60

[lxii] Kasper DL, et al., eds. Harrison's Principles of Internal Medicine, 16th ed. McGraw-Hill, New York, 2004

[lxiii] www.nia.nih.gov/alzheimers

[lxiv] http://www.cdc.gov/prions/

[lxv] http://www.altmedrev.com publications/4/3/144.pdf

[lxvi] **Alzheimer's Disease Education and Referral (ADEAR) Center**

[lxvii] https://www.nia.nih.gov/alzheimers/publication/alzheimers-disease-medications-fact-sheet

[lxviii] Sadowsky CH, Galvin JE. "Guidelines for management of cognitive and behavioral problems in dementia." J Am Board Fam Med. 25.3 (2012): 350-66

[lxix] http://www.telegraph.co.uk/health/healthnews/6250641/Extra-virgin-olive-oilcould-prevent-Alzheimers-disease.html

[lxx] http://www.huffingtonpost.com/2013/03/31/olive-oil-alzheimers-disease-extravirgin-amyloid-beta_n_2935483.html

[lxxi] http://www.thedenverchannel.com/lifestyle/health/couple-inspire-study- ofcoconut-oil-alzheimers-disease-061313

[lxxii] Del Pozo-Insfran D, Percival SS, Talcott ST. Acai (Euterpe oleracea Mart.) polyphenolics in their glycoside and aglycone forms induce apoptosis of HL-60 leukemia cells. J Agric Food Chem. 2006 Feb 22;54(4):1222-9.

[lxxiii] http://www.ars.usda.gov/SP2UserFiles/Place/12354500/Data/ORAC/ORAC07.pdf. Accessed July 1, 2008

[lxxiv] Ohgami K, Ilieva I, Shiratori K, et al. Anti-inflammatory effects of aronia extract on rat endotoxin-induced uveitis. Invest Ophthalmol Vis Sci. 2005 Jan;46(1):275-81.

[lxxv] Lala G, Malik M, Zhao C, et al. Anthocyanin-rich extracts inhibit multiple biomarkers of colon cancer in rats. Nutr Cancer. 2006;54(1):84-93.

[lxxvi] Valcheva-Kuzmanova S, Kuzmanov K, Tancheva S, Belcheva A. Hypoglycemic and hypolipidemic effects of Aroniamelanocarpa fruit juice in streptozotocin-induced diabetic rats. Methods Find ExpClinPharmacol. 2007 Mar;29(2):101-5.

[lxxvii] http://news.bbc.co.uk/2/hi/uk_news/scotland/tayside_and_central/5234108.stm. Accessed July 2, 2008

(lxxviii) Nakaishi H, Matsumoto H, Tominaga S, Hirayama M. Effects of black current anthocyanoside intake on dark adaptation and VDT work-induced transient refractive alteration in healthy humans. Altern Med Rev. 2000 Dec;5(6):553-62.

(lxxix) Marniemi J, Hakala P, Maki J, Ahotupa M. Partial resistance of low density lipoprotein to oxidation in vivo after increased intake of berries. NutrMetabCardiovasc Dis. 2000 Dec;10(6):331-7.

(lxxx) Matsumoto H, Takenami E, Iwasaki-Kurashige K, et al. Effects of blackcurrant anthocyanin intake on peripheral muscle circulation during typing work in humans. Eur J Appl Physiol. 2005 May;94(1-2):36-45.

(lxxxi) Yuko N, Hitoshi M, Kazuo T. Endothelium-dependent vasorelaxation induced by blackcurrant concentrate in rat thoracic aorta. Japan J Pharm. 2002;89(1):29-35.

(lxxxii) Jones E, Hughes RE. Quercetin, flavonoids and the life-span of mice. ExpGerontol. 1982;17(3):213-7.

(lxxxiii) Puupponen-Pimia R, Nohynek L, Meier C, et al. Antimicrobial properties of phenolic compounds from berries. J ApplMicrobiol. 2001 Apr;90(4):494-507.

(lxxxiv) http://www.cas.flinders.edu.au/sanra/research/proj0026.html. Accessed July 2, 2008. -60

(lxxxv) Seeram NP, Adams LS, Zhang Y, et al. Blackberry, black raspberry, blueberry, cranberry, red raspberry, and strawberry extracts inhibit growth and stimulate apoptosis of human cancer cells in vitro. J Agric Food Chem. 2006 Dec 13;54(25):9329-39.

(lxxxvi) Cavanagh HM, Hipwell M, Wilkinson JM. Antibacterial activity of berry fruits used for culinary purposes. J Med Food. 2003;6(1):57-61.

(lxxxvii) Tsuda T, Shiga K, Ohshima K, Kawakishi S, Osawa T. Inhibition of lipid peroxidation and the active oxygen radical scavenging effect of anthocyanin pigments isolated from Phaseolus vulgaris L. BiochemPharmacol. 1996 Oct 11;52(7):1033-9.

(lxxxviii) Tsuda T, Horio F, Kitoh J, Osawa T. Protective effects of dietary cyanidin 3-O-beta-D-glucoside on liver ischemia-reperfusion injury in rats. Arch BiochemBiophys. 1999 Aug 15;368(2):361-6.

(lxxxix) Tsuda T, Horio F, Osawa T. Dietary cyanidin 3-O-beta-D-glucoside increases ex vivo oxidation resistance of serum in rats. Lipids. 1998 Jun;33(6):583-8.

(xc) Tsuda T, Horio F, Osawa T. Cyanidin 3-O-beta-D-glucoside suppresses nitric oxide production during a zymosan treatment in rats. J NutrSciVitaminol (Tokyo). 2002 Aug;48(4):305-10.

(xci) Serraino I, Dugo L, Dugo P, et al. Protective effects of cyanidin-3-O-glucoside from blackberry extract against peroxynitrite-induced endothelial dysfunction and vascular failure. Life Sci. 2003 Jul 18;73(9):1097-114.

(xcii) Bere E. Wild berries: a good source of omega-3. Eur J ClinNutr. 2007 Mar;61(3):431-3.

(xciii) Kalea AZ, Lamari FN, Theocharis AD, et al. Wild blueberry (Vacciniumangustifolium) consumption affects the composition and structure of glycosaminoglycans in Sprague-Dawley rat aorta. J NutrBiochem. 2006 Feb;17(2):109-16.

(xciv) Shukitt-Hale B, Carey AN, Jenkins D, Rabin BM, Joseph JA. Beneficial effects of fruit extracts on neuronal function and behavior in a rodent model of accelerated aging. Neurobiol Aging. 2007 Aug;28(8):1187-94.

(xcv) Kelley DS, Rasooly R, Jacob RA, Kader AA, Mackey BE. Consumption of Bing sweet cherries lowers circulating concentrations of inflammation markers in healthy men and women. J Nutr. 2006 Apr;136(4):981-6.

(xcvi) http://www.sciencedaily.com/releases/2007/04/070430074703.htm. Accessed July 2, 2008.

(xcvii) Jayaprakasam B, Vareed SK, Olson LK, Nair MG. Insulin secretion by bioactive anthocyanins and anthocyanidins present in fruits. J Agric Food Chem. 2005 Jan 12;53(1):28-31.

(xcviii) Clark SS. Perillyl alcohol induces c-Myc-dependent apoptosis in Bcr/Abl-transformed leukemia cells. Oncology. 2006;70(1):13-8.

(xcix) Jepson RG, Craig JC. A systematic review of the evidence for cranberries and blueberries in UTI prevention. MolNutr Food Res. 2007 Jun;51(6):738-45.
(c) Liu Y, Gallardo-Moreno AM, Pinzon-Arango PA, Reynolds Y, Rodriguez G, Camesano TA. Cranberry changes the physicochemical surface properties of E. coli and adhesion with uroepithelial cells. Colloids Surf B Biointerfaces. 2008 Aug 1;65(1):35-42.2057
(ci) Neto CC. Cranberry and its phytochemicals: a review of in vitro anticancer studies. J Nutr. 2007 Jan;137(1 Suppl):186S-93S
(cii) Burger O, Weiss E, Sharon N, Tabak M, Neeman I, Ofek I. Inhibition of Helicobacter pylori adhesion to human gastric mucus by a high-molecular-weight constituent of cranberry juice. Crit Rev Food SciNutr. 2002;42(3 Suppl):279-84
(ciii) McKay DL, Blumberg JB. Cranberries (Vacciniummacrocarpon) and cardiovascular disease risk factors. Nutr Rev. 2007 Nov;65(11):490-502.-60
(civ) Zakay-Rones Z, Varsano N, Zlotnik M, et al. Inhibition of several strains of influenza virus in vitro and reduction of symptoms by an elderberry extract (Sambucusnigra L.) during an outbreak of influenza B Panama. J Altern Complement Med. 1995;1(4):361-9.
(cv) Bell DR, Gochenaur K. Direct vasoactive and vasoprotective properties of anthocyanin-rich extracts. J Appl Physiol. 2006 Apr;100(4):1164-70.
(cvi) Freedman JE, Parker C, III, Li L, et al. Select flavonoids and whole juice from purple grapes inhibit platelet function and enhance nitric oxide release. Circulation. 2001 Jun 12;103(23):2792-8.
(cvii) Day AP, Kemp HJ, Bolton C, Hartog M, Stansbie D. Effect of concentrated red grape juice consumption on serum antioxidant capacity and low-density lipoprotein oxidation. Ann NutrMetab. 1997;41(6):353-7.
(cviii) Olson ER, Naugle JE, Zhang X, Bomser JA, Meszaros JG. Inhibition of cardiac fibroblast proliferation and myofibroblast differentiation by resveratrol. Am J Physiol Heart Circ Physiol. 2005 Mar;288(3):H1131-8.
(cix) Lu KT, Chiou RY, Chen LG, et al. Neuroprotective effects of resveratrol on cerebral ischemia-induced neuron loss mediated by free radical scavenging and cerebral blood flow elevation. J Agric Food Chem. 2006 Apr 19;54(8):3126-31.
(cx) Miura D, Miura Y, Yagasaki K. Hypolipidemic action of dietary resveratrol, a phytoalexin in grapes and red wine, in hepatoma-bearing rats. Life Sci. 2003 Aug 1;73(11):1393-400.
(cxi) Szewczuk LM and Penning TM. Mechanism-based inactivation of COX-1 by red wine m-hydroquinones: a structure-activity relationship study. J Nat Prod. 2004 Nov;67(11):1777-82
(cxii) Hu H, Qin YM. Grape seed proanthocyanidin extract induced mitochondria-associated apoptosis in human acute myeloid leukaemia 14.3D10 cells. Chin Med J (Engl.). 2006 Mar 5;119(5):417-21.
(cxiii) Aviram M, Dornfeld L. Pomegranate juice consumption inhibits serum angiotensin converting enzyme activity and reduces systolic blood pressure. Atherosclerosis. 2001 Sep;158(1):195-8.
(cxiv) Aviram M, Rosenblat M, Gaitini D, et al. Pomegranate juice consumption for 3 years by patients with carotid artery stenosis reduces common carotid intima-media thickness, blood pressure and LDL oxidation. ClinNutr. 2004 Jun;23(3):423-33.

[cxv] Malik A, Afaq F, Sarfaraz S et al. Pomegranate fruit juice for chemoprevention and chemotherapy of prostate cancer. ProcNatlAcadSci USA. 2005 Oct 11;102(41):14813-8.
[cxvi] Kim ND, Mehta R, Yu W, et al. Chemopreventive and adjuvant therapeutic potential of pomegranate (Punicagranatum) for human breast cancer. Breast Cancer Res Treat. 2002 Feb;71(3):203-17.
[cxvii] Khan N, Hadi N, Afaq F, et al. Pomegranate fruit extract inhibits prosurvival pathways in human A549 lung carcinoma cells and tumor growth in athymic nude mice. Carcinogenesis. 2007 Jan;28(1):163-73.
[cxviii] Kohno H, Suzuki R, Yasui Y, et al. Pomegranate seed oil rich in conjugated linolenic acid suppresses chemically induced colon carcinogenesis in rats. Cancer Sci. 2004 Jun;95(6):481-6.
[cxix] Aslam MN, Lansky EP, Varani J. Pomegranate as a cosmeceutical source: pomegranate fractions promote proliferation and procollagen synthesis and inhibit matrix metalloproteinase-1 production in human skin cells. J Ethnopharmacol. 2006 Feb 20;103(3):311-8.
[cxx] Meyers KJ, Watkins CB, Pritts MP, Liu RH. Antioxidant and antiproliferative activities of strawberries. J Agric Food Chem. 2003 Nov 5;51(23):6887-92.
[cxxi] Shukitt-Hale B, Carey AN, Jenkins D, Rabin BM, Joseph JA. Beneficial effects of fruit extracts on neuronal function and behavior in a rodent model of accelerated aging. Neurobiol Aging. 2007 Aug;28(8):1187-94.
[cxxii] Naemura A, Mitani T, Ijiri Y, et al. Anti-thrombotic effect of strawberries. Blood Coagul Fibrinolysis. 2005 Oct;16(7):501-9.
[cxxiii] http://www.motherearthliving.com/health-and-wellness/inside-plants-rosemarycompounds-help-the-brain-manage-memory-16-rosemary-compounds-help-the-.aspx#axzz2Wo7CvY8m
[cxxiv] Wang XD, Zhang JM, Yang HH, et al. Modulation of NMDA receptor by huperzine A in rat cerebral cortex. Zhongguo Yao Li XueBao. 1999 Jan;20(1):31-5
[cxxv] Wang R, et al. "Progress in Studies of Huperzine A: A Natural Cholinesterase Inhibitor from Chinese Herbal Medicine." ActaPharmacol Sin. 27.1 (2006b): 1-26.
[cxxvi] Sun QQ, et al. "Huperzine-A Capsules Enhance Memory and Learning Performance in 34 Pairs of Matched Adolescent Students." Zhongguo Yao Li XueBao. 20.7 (1999): 601-3
[cxxvii] Wang BS, et al. "Efficacy and Safety of Natural Acetylcholinesterase Inhibitor Huperzine A in the Treatment of Alzheimer's Disease: An Updated Meta-Analysis." J Neural Transm. 116.4 (2009): 457-65.
[cxxviii] Christensen LP. "Ginsenosides chemistry, biosynthesis, analysis, and potential health effects." Adv Food Nutr Res. 55 (2009): 1-99.
[cxxix] Lee ST, et al. "Panax ginseng Enhances Cognitive Performance in Alzheimer Disease." Alzheimer Dis AssocDisord. 22.3 (2008): 222-6.
[cxxx] Ven Murthy MR, et al. "Scientific Basis for the Use of Indian Ayurvedic Medicinal Plants in the Treatment of Neurodegenerative Disorders: Ashwagandha." Cent NervSyst Agents Med Chem. 10.3 (2010): 238-46
[cxxxi] Sehgal N, Gupta A, ValliRK,et al.Withaniasomnifera reverses Alzheimer's disease pathology by enhancing low-density lipoprotein receptor-related protein in liver. ProcNatlAcadSci U S A. 2012 Feb 28;109(9):3510-5. Epub 2012 Jan 30
[cxxxii] Kuboyama T, et al. "Neuritic Regeneration and Synaptic Reconstruction Induced by Withanolide A." Br J Pharmacol. 144.7 (2005): 961-71

(cxxxiii) Choudhary MI, et al. "Cholinesterase Inhibiting Withanolides from Withaniasomnifera." Chem Pharm Bull Tokyo. 52.11 (2004): 1358-61
(cxxxiv) http://www.mskcc.org/cancer-care/herb/ashwagandha
(cxxxvi) Perry EK, et al. "Medicinal Plants and Alzheimer's Disease: From Ethnobotany to Phytotherapy." J Pharm Pharmacol. 51.5 (1999): 527-34.
(cxxxvii) Diamond BJ, et al. "Ginkgo biloba Extract: Mechanisms and Clinical Indications." Arch Phys Med Rehab. 81.5 (2000): 668-78
(cxxxviii) Yao ZX, et al. "Ginkgo biloba Extract (Egb 761) Inhibits Beta-Amyloid Production by Lowering Free Cholesterol Levels." J NutrBiochem. 15.12 (2004): 749-56.
(cxxxix) Aranda-Abreu GE, et al. "Rehabilitating a Brain with Alzheimer's: A Proposal." ClinInterv Aging. 6 (2011): 53-9.
(cxl) Janssen IM, et al. "Ginkgo biloba in Alzheimer's Disease: A Systematic Review." Wien Med Wochenschr. 160.21-22 (2010): 539-46
(cxli) Yang F, et al. "Curcumin Inhibits Formation of Amyloid Beta Oligomers and Fibrils, Binds Plaques, and Reduces Amyloid in Vivo." J Biol Chem. 280.7 (2005): 5892-901
(cxlii) Baum L, et al. "Curcumin Interaction with Copper and Iron Suggests One Possible Mechanism of Action in Alzheimer's Disease Animal Models." J Alzheimers Dis. 6.4 (2004): 367-77
(cxliii) Begum AN, et al. "Curcumin Structure-Function, Bioavailability, and Efficacy in Models of Neuroinflammation and Alzheimer's Disease." J PharmacolExpTher. 326.1 (2008): 196-208
(cxliv) Yang F, et al. "Curcumin Inhibits Formation of Amyloid Beta Oligomers and Fibrils, Binds Plaques, and Reduces Amyloid in Vivo." J Biol Chem. 280.7 (2005): 5892-901
(cxlv) Mishra S, et al. "The Effect of Curcumin (Turmeric) on Alzheimer's Disease: An Overview." Ann Indian Acad Neurol. 11.1 (2008): 13-9
(cxlvi) Cole GM, et al. "NSAID and Antioxidant Prevention of Alzheimer's Disease: Lessons from in Vitro and Animal Models." Ann N Y Acad Sci. 1035 (2004): 68-84
(cxlvii) Aggarwal BB, et al. "Suppression of the Nuclear Factor-KappaB Activation Pathway by Spice-Derived Phytochemicals: Reasoning for Seasoning." Ann N Y Acad Sci. 1030 (2004): 434-41
(cxlviii) Butt MS, et al. "Coffee and Its Consumption: Benefits and Risks." Crit Rev Food SciNutr. 51.4 (2011): 363-73.
(cxlix) Cao C, et al. "Caffeine Synergizes with Another Coffee Component to Increase Plasma GCSF: Linkage to Cognitive Benefits in Alzheimer's Mice." J Alzheimers Dis. 25.2 (2011): 323-35
(cl) Montagnana M, et al. "Coffee Intake and Cardiovascular Disease: Virtue Does Not Take Center Stage." SeminThrombHemos. 38.2 (2012): 164-77. Epub February 18, 2012
(cli) Zapp LM, Slaga TJ, Zhao J. et al. Method for enhancing post-processing content of beneficial compounds in beverages naturally - United States Patent Application 20100183790. Publication Date: 2010-07-22. Available at: http://patent.ipexl.com/U2S/20100183790.html accessed 7/25/2012 al.Withaniasomnifera reverses
(clii) Mandel S, et al. "Green Tea Catechins as Brain-Permeable, Natural Iron Chelators-Antioxidants for the Treatment of Neurodegenerative Disorders." MolNutr Food Res. 50.2 (2006): 229-34.
(cliii) Kim TI, et al. "L-Theanine, an Amino Acid in Green Tea, Attenuates Beta-Amyloid-Induced Cognitive Dysfunction and Neurotoxicity: Reduction in Oxidative Damage and Inactivation of ERK/p38 Kinase and NF-kappaB Pathways." Free RadicBiol Med. 47.11 (2009): 1601-10.
(cliv) Rezai-Zadeh K, et al. "Green Tea Epigallocatechin-3-Gallate (EGCG) Modulates Amyloid Precursor Protein Cleavage and Reduces Cerebral Amyloidosis in Alzheimer Transgenic Mice." J Neurosci. 25.38 (2005): 8807-14.

[clv] Mandel SA, et al. "Understanding the Broad-Spectrum Neuroprotective Action Profile of Green Tea Polyphenols in Aging and Neurodegenerative Diseases." J Alzheimers Dis. 25.2 (2011): 187-208
[clvi] Haque AM, et al. "Green Tea Catechins Prevent Cognitive Deficits Caused by Abeta1-40 in Rats." J NutrBiochem. 19.9 (2008): 619-26
[clvii] Modi, K.K., Rangasamy, S.B., Dasarathi, S. et al. J NeuroimmunePharmacol (2016). doi:10.1007/s11481-016-9693-6
[clviii] Walcutt, D. (2009). Chocolate and Mood Disorders. Psych Central. Retrieved on October 16, 2016, from http://psychcentral.com/blog/archives/2009/04/27/chocolate-and-mood-disorders/
[clix] http://antoine.frostburg.edu/chem/senese/101/features/anandamide.shtml
[clx] http://news.bbc.co.uk/2/hi/health/6558775.stm
[clxi] Craig, Winston J.; Nguyen, Thuy T. (1984). "Caffeine and theobromine levels in cocoa and carob products". Journal of Food Science. **49** (1): 302–303.
[clxii] https://www.sciencedaily.com/releases/2007/02/070221101326.htm
[clxiii] https://www.ncbi.nlm.nih.gov/pubmed/16794461
[clxiv] https://www.ncbi.nlm.nih.gov/pubmed/23810791
[clxv] http://link.springer.com/article/10.1007/BF00210835
[clxvi] https://www.sciencedaily.com/releases/2008/12/081210091039.htm
[clxvii] http://link.springer.com/article/10.1023/A:1007614613771
[clxviii] http://evolutionarypsychiatry.blogspot.co.za/2010/10/magnesium-and-brain.html
[clxix] https://www.psychologytoday.com/blog/evolutionary-psychiatry/201106/magnesium-and-the-brain-the-orig
[clxx] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2769828/
[clxxi] http://ajcn.nutrition.org/content/93/1/62.full
[clxxii] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3575938/
[clxxiii] Milad SB, et al. "Inflammation and apoptosis in aortic tissues of aged type II diabetes: Amelioration with Mechanisms Underlying the Tissue-selective Agonist/Antagonist Activities of ." Life Sci. 86.23-24 (2010): 844-53.
[clxxiv] Holmquist L, et al. "Lipoic Acid as a Novel Treatment for Alzheimer's disease and Related Dementias." PharmacolThera. 113.1 (2007): 154-164.
[clxxv] Hager K, et al. "Alpha-Lipoic Acid as a New Treatment Option for Alzheimer [corrected] Type Dementia." Arch GerontolGeriatr. 32.3 (2001): 275-82.
[clxxvi] Hager K, et al. "Alpha-Lipoic Acid as a New Treatment Option for Alzheimer's Disease—A 48 Months Follow-up Analysis." J Neural Transm Suppl. 72 (2007): 189-93.
[clxxvii] Dhitavat S, et al. "Folate, Vitamin E, and Acetyl-L-Carnitine Provide Synergistic Protection against Oxidative Stress Resulting from Exposure of Human Neuroblastoma Cells to Amyloid-Beta." Brain Res. 1061.2 (2005): 114-7.
[clxxviii] Virmani MA, et al. "The Action of Acetyl-L-Carnitine on the Neurotoxicity Evoked by Amyloid Fragments and Peroxide on Primary Rat Cortical Neurones." Ann N Y Acad Sci. 939 (2001): 162-78.
[clxxix] Ames BN, et al. "Delaying the Mitochondrial Decay of Aging with Acetylcarnitine." Ann N Y Acad Sci. 1033 (2004): 108-16.
[clxxx] Zhou P, et al. "Acetyl-L-Carnitine Attenuates Homocysteine-Induced Alzheimer-Like Histopathological and Behavioral Abnormalities." Rejuvenation Res. 14.6 (2011): 669-79.
[clxxxi] Epis R, et al. "Modulatory Effect of Acetyl-L-Carnitine on Amyloid Precursor Protein Metabolism in Hippocampal Neurons." Eur J Pharmocol. 597.1-3 (2008): 51-53.
[clxxxii] http://www.altmedrev.com/publications/4/3/144.pdf
[clxxxiii] Eyles DW, et al. "Distribution of the Vitamin D Receptor and 1α-hydroxylase in Human Brain." J ChemNeuroanat. 29.1 (2005): 21-30
[clxxxiv] Ito S, et al. "1-alpha,25-Dihydroxyvitamin D3 Enhances Cerebral Clearance of Human Amyloid-B Peptide(1-40) from Mouse Brain across the Blood-Brain Barrier." Fluids Barriers CNS. 8 (2011): 20

(clxxxv) Annweiler C, Rolland Y, Schott AM, et al. Higher Vitamin D Dietary Intake Is Associated with Lower Risk of Alzheimer's Disease: A 7-Year Follow-Up. [In Eng] J GerontolABiolSci Med Sci. 2012 Apr 13
(clxxxvi) Ciabattoni G, et al. "Determinants of Platelet Activation in Alzheimer's Disease." Neurobiol Aging. 28.3 (2007): 336-72. Epub ahead of print January 24, 2006
(clxxxvii) Gehin A, et al. "Glyphosate-induced Antioxidant Imbalance in HaCaT: The Protective Effect of Vitamins C and E." Environ ToxicolPharmocol. 22.1 (2006): 27-34
(clxxxviii) Boothby LA, Doering PL. "Vitamin C and vitamin E for Alzheimer's disease." Ann Pharmacother. 39.12 (2005):2073-80
(clxxxix) Zandi PP, et al. "Reduced Risk of Alzheimer Disease in Users of Antioxidant Vitamin Supplements: The Cache County Study." Arch Neurol. 61.1 (2004): 82-8
(cxc) http://www.mskcc.org/cancer-care/herb/vitamin-e
(cxci) Swanson D, et al. "Omega-3 fatty acids DHA and EPA: Health Benefits throughout Life." AdvNutr. 3.1 (2012): 1-7
(cxcii) Young G, et al. "Omega-3 Fatty Acids and Neuropsychiatric Disorders." ReprodNutr Dev 45.1 (2005): 1-28
(cxciii) Lukiw WJ, et al. "A Role for Docosahexaenoic Acid-Derived Neuroprotectin D1 in Neural Cell Survival and Alzheimer Disease." J Clin Invest. 115.10 (2005): 2774-83
(cxciv) Akbar M, et al. "Docosahexaenoic Acid: A Positive Modulator of Akt Signaling in Neuronal Survival." ProcNatlAcadSci USA. 102.31 (2005): 10858-63.
(cxcv) Ma QL, et al. "Beta-Amyloid Oligomers Induce Phosphorylation of Tau and Inactivation of Insulin Receptor Substrate via C-Jun N-Terminal Kinase Signaling: Suppression by Omega-3 Fatty Acids and Curcumin." J Neurosci. 29.28 (2009): 9078-89
(cxcvi) http://www.altmedrev.com/publications/4/3/144.pdf
(cxcvii) Balestreri R, Fontana L, Astengo F. A double-blind placebo controlled evaluation of the safety and efficacy of vinpocetine in the treatment of patients with chronic vascular senile cerebral dysfunction. J Am Geriatr Soc. 1987 May;35(5):425-30
(cxcviii) Chowanadisai W, et al. "Pyrroloquinolinequinone stimulates mitochondrial biogenesis through cAMP response element-binding protein phosphorylation and increased PGC-1alpha expression." J Biol Chem. 285.1 (2010): 142-152
(cxcix) Tao R, et al. "Pyrroloquinolinequinone preserves mitochondrial function and prevents oxidative injury in Alzheimer's diseaseult rat cardiac myocytes." BiochemBiophys Res Commune. 363.2 (2007):257-262.
(cc) Kim J, et al. "The Inhibitory Effect of Pyrroloquinoline Quinone on the Amyloid Formation and Cytotoxicity of Truncated Alpha-Synuclein." MolNeurodegener. 5 (2010): 20
(cci) Nakano M, Ubukata K, Yamamoto T, Yamaguchi H. Effect of pyrroloquinolinequinone (PQQ) on mental status of middle-aged and elderly persons. FOOD Style. 2009;21:13(7):50-3
(ccii) Kato-Kataoka A, et al. "Soybean-derived Phosphatidylserine Improves Memory Functions of the Elderly Japanese Subjects with Memory Complaints." J ClinBiochemNutr. 47.3 (2010) 246-55
(cciii) Shyh-Hwa L, et al. "Docosahexaenoic Acid and Phosphatidylserine Supplementations Improve Antioxidant Activities and Cognitive Functions of the Developing Brain on Pentylenetetrazol-Induced Seizure Model." Brain Res. 1451. (2012): 19-26
(cciv) Kim D, et al. "SIRT1 Deacetylase Protects against Neurodegeneration in Models for Alzheimer's Disease and Amyotrophic Lateral Sclerosis." EMBO J. 26.13 (2007): 3169-79.

(ccv) Vingtdeux V, Dreses-Werringloer U, Zhao H, et al. "Therapeutic potential of resveratrol in Alzheimer's disease." BMC Neurosci. 9(Suppl 2) (2008): S6
(ccvi) Ho L, et al. "Heterogeneity in Red Wine Polyphenolic Contents Differentially Influences Alzheimer's Disease-Type Neuropathology and Cognitive Deterioration." J Alzheimers Dis. 16.1 (2009): 59-72.
(ccvii) Wang J, et al. "Grape-Derived Polyphenolics Prevent Alpha Beta Oligomerization and Attenuate Cognitive Deterioration in a Mouse Model of Alzheimer's Disease." J Neurosci. 28.25 (2008): 6388-92
(ccviii) Bardgett ME, et al. "Magnesium Deficiency Impairs Fear Conditioning in Mice." Brain Res. 1038.1 (2005): 100-6.
(ccix) Barbagallo M, et al. "Altered Ionised Magnesium Levels in Mild-to-Moderate Alzheimer's Disease." Magnes Res. 24.3 (2011): S115-21.
(ccx) Corsonello A, et al. "Serum Magnesium Levels and Cognitive Impairment in Hospitalized Hypertensive Patients." Magnes Res. 14.4 (2001): 273-82.
(ccxi) Ravaglia G, et al. "Homocysteine and Folate as Risk Factors for Dementia and Alzheimer Disease." Am J ClinNutr. 82.3 (2005): 636-43
(ccxii) Tucker KL, et al. "High Homocysteine and Low B Vitamins Predict Cognitive Decline in Aging Men: The Veterans Affairs Normative Aging Study." Am J ClinNutr. 82.3 (2005): 627-35
(ccxiii) Engelborghs S, et al. "Correlations between Cognitive, Behavioural and Psychological Findings and Levels of Vitamin B12 and Folate in Patients with Dementia." Int J Geriatr Psychiatry. 19.4 (2004): 365-70
(ccxiv) Wang HX, et al. "Vitamin B(12) and Folate in Relation to the Development of Alzheimer's Disease." Neurology. 56.9 (2001): 1188-94
(ccxv) Mulder C, et al. "Low Vitamin B6 Levels Are Associated with White Matter Lesions in Alzheimer's Disease." J Am Geriatr Soc. 53.6 (2005): 1073-4
(ccxvi) Hinterberger M and Fischer P. "Folate and Alzheimer: when time matters." J Neural Transm. 2012 May 25
(ccxvii) Kado DM, et al. "Homocysteine versus the Vitamins Folate, B6, and B12 as Predictors of Cognitive Function and Decline in Older High-Functioning Adults: MacArthur Studies of Successful Aging." Am J Med. 118.2 (2005): 161-7
(ccxviii) Nakano M, Ubukata K, Yamamoto T, Yamaguchi H. Effect of pyrroloquinolinequinone (PQQ) on mental status of middle-aged and elderly persons. FOOD Style. 2009;21:13(7):50-3
(ccxix) Yang X, et al. "Coenzyme Q10 Attenuates Beta-Amyloid Pathology in the Aged Transgenic Mice with Alzheimer Presenilin 1 Mutation." J MolNeurosci. 34.2 (2008): 165-71
(ccxx) Ono K, et al. "Preformed Beta-Amyloid Fibrils Are Destabilized by Coenzyme Q10 in Vitro." BiochemBiophys Res Commun. 330.1 (2005): 111-6
(ccxxi) Gutzmann H, and Hadler D. Sustained Efficacy and Safety of Idebenone in the Treatment of Alzheimer's Disease: Update on a 2-Year Double-Blind Multicentre Study. [In eng] J Neural Transm Suppl. 1998 54(301-10
(ccxxii) http://www.liveinthenow.com/article/coq10-blocks-alzheimers-damage
(ccxxiii) Pocernich CB, et al. "Nutritional Approaches to Modulate Oxidative Stress in Alzheimer's Disease." Curr Alzheimer Res. 8.5 (2011): 452-69
(ccxxiv) Tchantchou F, et al. "N-Acetyl Cysteine Alleviates Oxidative Damage to Central Nervous System of Apoe-Deficient Mice Following Folate and Vitamin E-Deficiency." J Alzheimers Dis. 7.2 (2005): 135-8

[ccxxv] Lau FC, et al. "The Beneficial Effects of Fruit Polyphenols on Brain Aging." Neurobiol Aging. 26.1 (2005): 128-32
[ccxxvi] Casadesus G, Shukitt-Hale B, Stellwagen HM, et al. NutrNeurosci. 7.5-6 (2004): 309-16
[ccxxvii] Wu X, et al. "Lipophilic and Hydrophilic Antioxidant Capacities of Common Foods in the United States." J Agric Food Chem. 52.12 (2004b): 4026-37
[ccxxviii] Lee YM, Han SI, Song BC, Yeum KJ. Bioactives in Commonly Consumed Cereal Grains: Implications for Oxidative Stress and Inflammation. Journal of medicinal food.Nov 2015;18(11):1179-1186
[ccxxix] Nagatsu T, Sawada M. Molecular mechanism of the relation of monoamine oxidase B and its inhibitors to Parkinson's disease: possible implications of glial cells. Journal of neural transmission. Supplementum. 2006(71):53-65
[ccxxx] Cai Z. Monoamine oxidase inhibitors: promising therapeutic agents for Alzheimer's disease (Review). Molecular medicine reports. May 2014;9(5):1533-1541
[ccxxxi] Wong RH, Howe PR, Bryan J, Coates AM, Buckley JD, Berry NM. Chronic effects of a wild green oat extract supplementation on cognitive performance in older adults: a randomised, double-blind, placebo-controlled, crossover trial. Nutrients.May 2012;4(5):331-342
[ccxxxii] Chi Y, Sauve AA. Nicotinamideriboside, a trace nutrient in foods, is a vitamin B3 with effects on energy metabolism and neuroprotection. Current opinion in clinical nutrition and metabolic care.Nov 2013;16(6):657-661
[ccxxxiii] Gao J, Wang WY, Mao YW, et al. A novel pathway regulates memory and plasticity via SIRT1 and miR-134. Nature. Aug 26 2010;466(7310):1105-1109
[ccxxxiv] Deleglise B, Lassus B, Soubeyre V, et al. Synapto-protective drugs evaluation in reconstructed neuronal network. PloS one.2013;8(8):e71103
[ccxxxv] http://altmedicine.about.com/od/completeazindex/a/benefits_bacopa.htm
[ccxxxvi] https://www.ncbi.nlm.nih.gov/pubmed/10383479
[ccxxxvii] http://www.smart-publications.com/articles/bacopa-protects-against-depressionand-alzheimers-disease
[ccxxxviii] http://well.blogs.nytimes.com/2013/04/25/ask-well-exercises-to-prevent-dementia/
[ccxxxix] http://well.blogs.nytimes.com/2013/04/10/how-exercise-may-boost-the-brain/
[ccxl] http://www.webmd.com/brain/news/20090413/how-pcbs-may-hurt-the-brain
[ccxli] http://www.alz.org/braintour/plaques.asp
[ccxlii] http://www.livescience.com/9977-depression-increase-chances-alzheimer.html
[ccxliii] http://www.alzheimersprevention.org/research_meditation_study.htm
[ccxliv] Demarin V, Podobnik SS, Storga-Tomic D, Kay G. Treatment of Alzheimer's disease with stabilized oral nicotinamide adenine dinucleotide: a randomized, double-blind study. Drugs under experimental and clinical research.2004;30(1):27-33
[ccxlv] Gu Y, et al. "Dietary Patterns in Alzheimer's Disease and Cognitive Aging." Curr Alzheimer Res. 8.5 (2011): 510-9
[ccxlvi] Scarmeas N, et al. "Mediterranean Diet and Alzheimer Disease Mortality." Neurology. 69 (2007): 1084-93.
[ccxlvii] Scarmeas N, et al. "Mediterranean Diet and Mild Cognitive Impairment." Arch Neurol. 66.2 (2009): 216-25

[ccxlviii] Scarmeas N, et al. “Mediterranean Diet and Alzheimer Disease Mortality.” Neurology. 69 (2007): 1084-93
[ccxlix] Solfrizzi V, et al. “Mediterranean Diet in Predementia and Dementia Syndromes.” Curr Alzheimer Res. 2011 Aug;8(5):520-42
[ccl] Jóźwiak S, et al. “Dietary Treatment of Epilepsy: Rebirth of an Ancient Treatment.” Neurol Neurochir Pol. 45.4 (2011): 370-8
[ccli] Van der Auwera I, Wera S, Van Leuven F, et al. “A ketogenic diet reduces amyloid beta 40 and 42 in mouse model of Alzheimer’s disease.” Nutr Metab (Lond). 2 (2005):28
[cclii] Jóźwiak S, et al. “Dietary Treatment of Epilepsy: Rebirth of an Ancient Treatment.” Neurol Neurochir Pol. 45.4 (2011): 370-8
[ccliii] Pasinetti GM, Zhao Z, Qin W, et al. Caloric intake and Alzheimer’s disease. Experimental approaches and therapeutic implications. Interdiscip Top Gerontol. 2007;35:159-75
[ccliv] Geda Y, et al. “Caloric Intake, Aging, and Mild Cognitive Impairment: A Population-Based Study.” Presented at the 64th Annual Meeting of the American Academy of Neurology. New Orleans. April 21-28, 2012