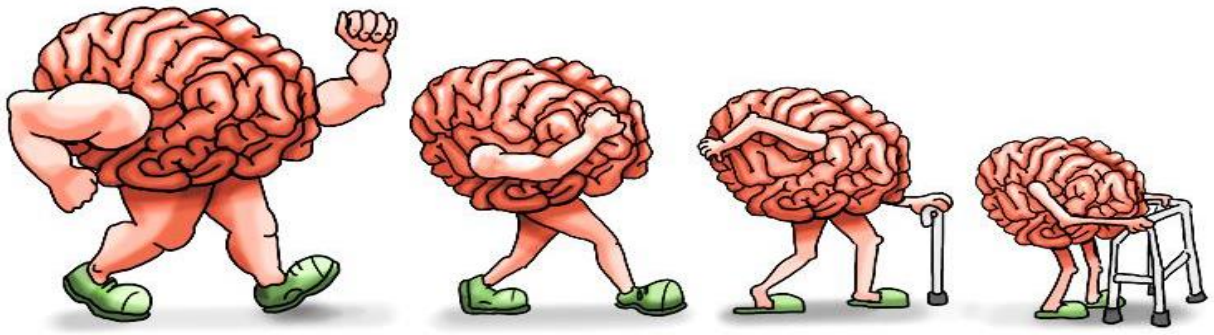
Neurodegenerative diseases

Anna Drabik

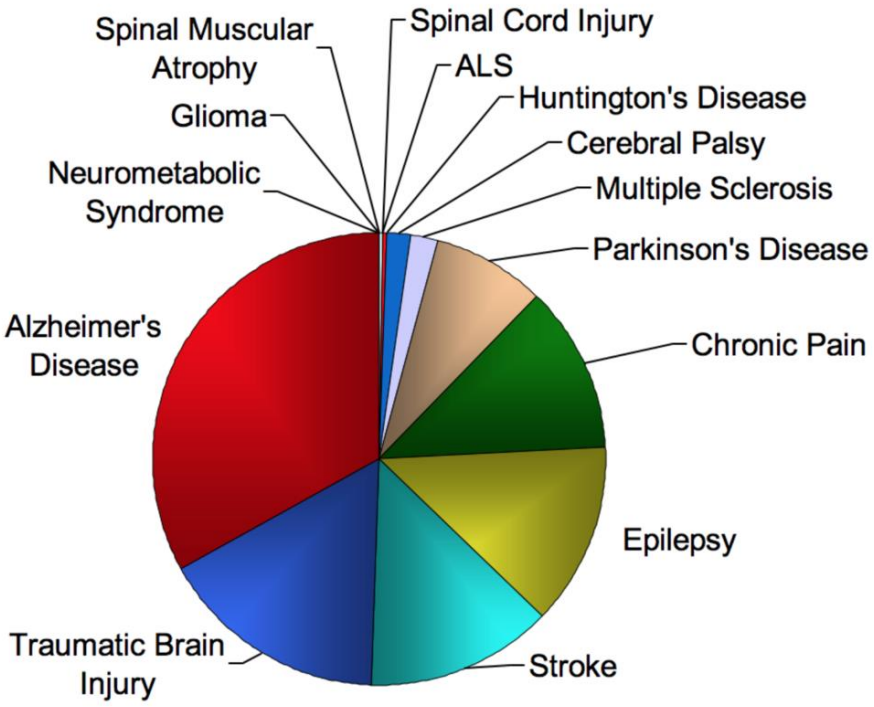
Neurodegenerative diseases

Neurodegenerative diseases occur when nerve cells in the brain or peripheral nervous system lose function over time and ultimately die.

Although treatments may help relieve some of the physical or mental symptoms associated with neurodegenerative diseases, there is currently no way to slow disease progression and no known cures.

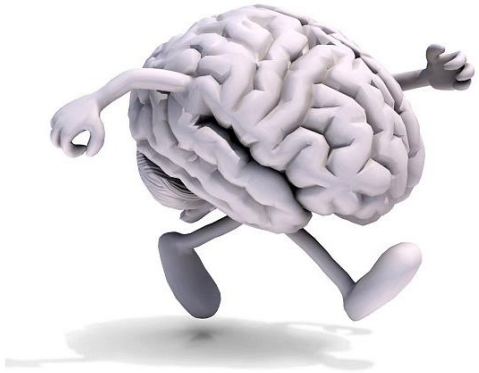


Neurodegenerative diseases



- Alzheimer’s AD
- Parkinson’s PD
- Huntington’s HD
- Spinocerebellar ataxia SCA
- Prion disease
- Motor neurone diseases MND

Neurodegenerative diseases Risk factors



Age

Gender

Family history

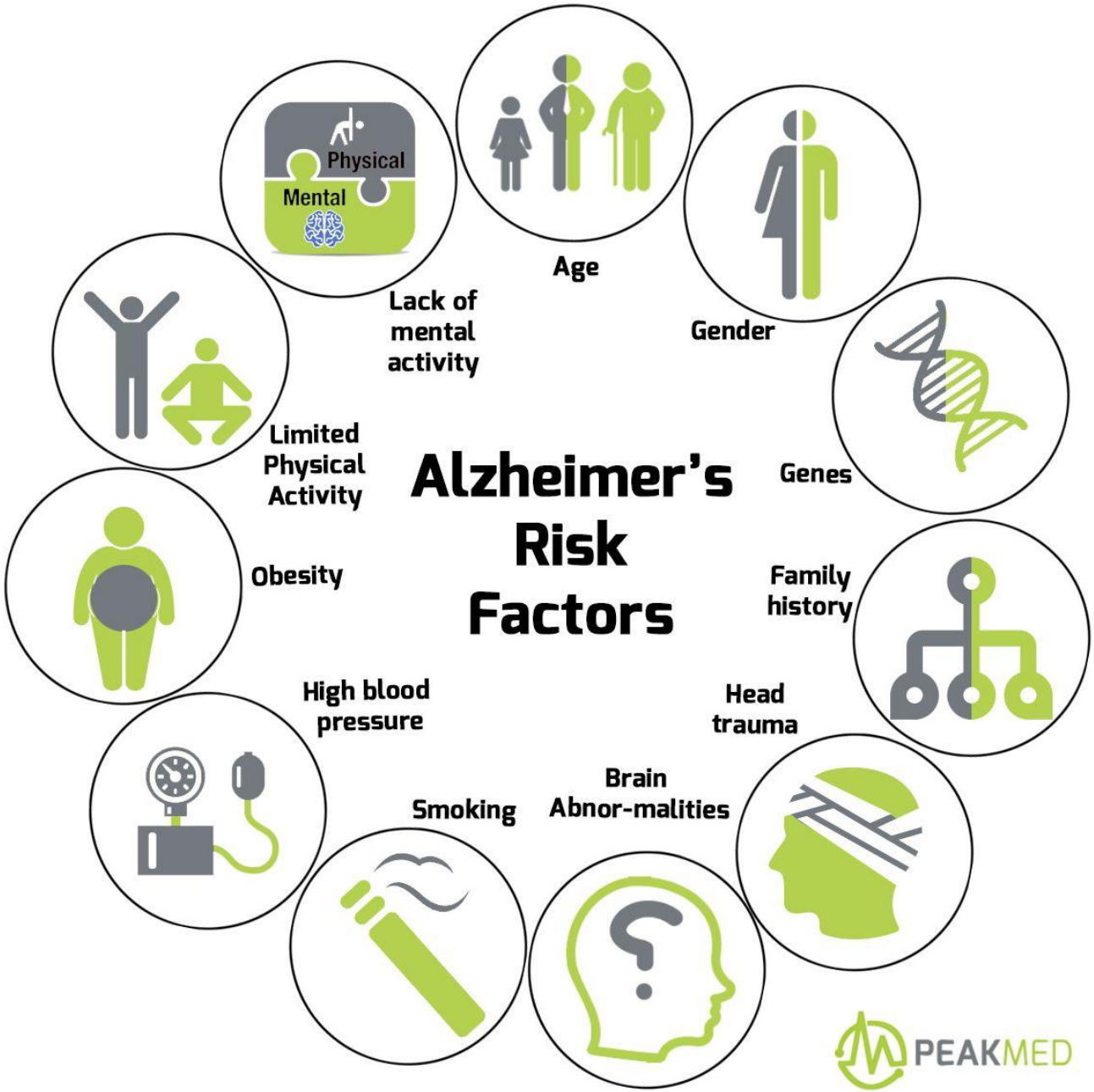
Environmental factors:

- Pesticides, fungicides, and insecticides
- Metals (e.g., arsenic, lead, manganese)
- Chemicals used in industry or consumer products (e.g., polychlorinated biphenyls (PCBs)
- Air pollution
- Biological factors (e.g., endotoxins produced by bacteria)
- Dietary and lifestyle factors (e.g., caffeine, tobacco smoke, dietary antioxidants)

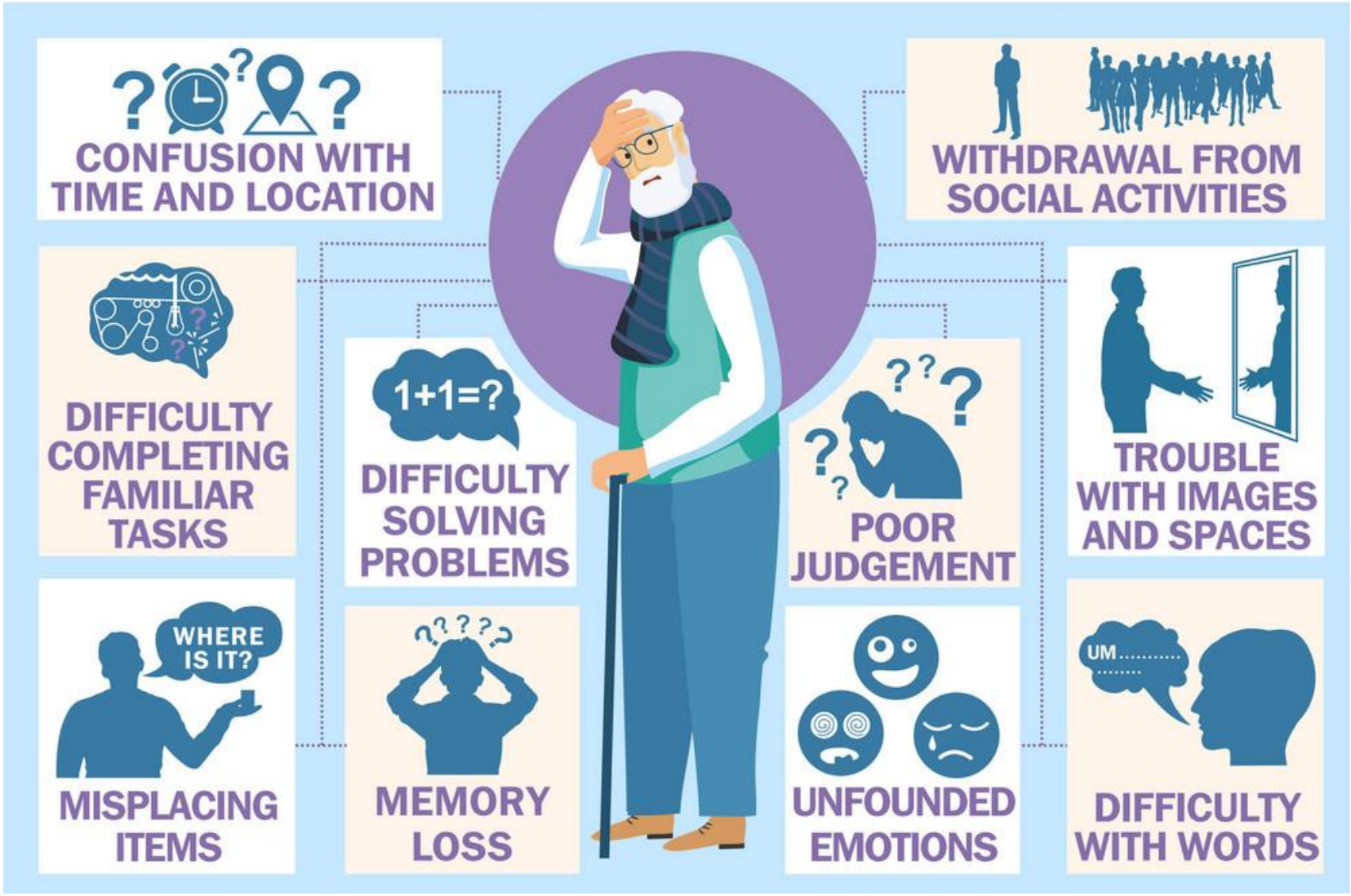
Head injury

Alzheimer's disease AD

- The most common form of dementia, affecting 400 000 Polish people over the age of 65, as well as thousands under the age of 65 who have early-onset AD.
- Women account for almost two-thirds of patients with AD.
- AD is diagnosed by ruling out other conditions with similar symptoms, which allows for a diagnosis with up to 95 percent accuracy.
- Unlike other forms of dementia, AD does not affect patients' motor function until late stages of the disease.


Alzheimer's Risk Factors
Physical
Mental
Lack of mental activity
Age
Gender
Genes
Family history
Head trauma
Brain Abnor-malities
Smoking
High blood pressure
Obesity
Limited Physical Activity
PEAKMED

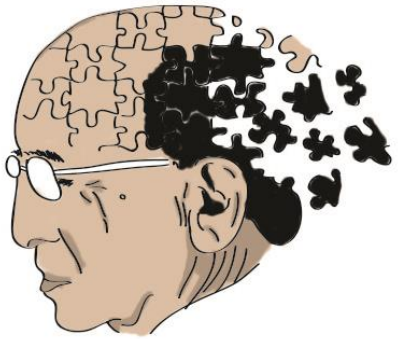
Alzheimer's Symptoms



Alzheimer's disease

Progressive, neurodegenerative disease that occurs when nerve cells in the brain die. The disease often results in the following behaviors:

- Impaired memory, thinking, and behavior
- Confusion
- Restlessness
- Personality and behavior changes
- Impaired judgment
- Impaired communication
- Inability to follow directions
- Language deterioration
- Impaired thought processes that involve visual and spatial awareness
- Emotional apathy



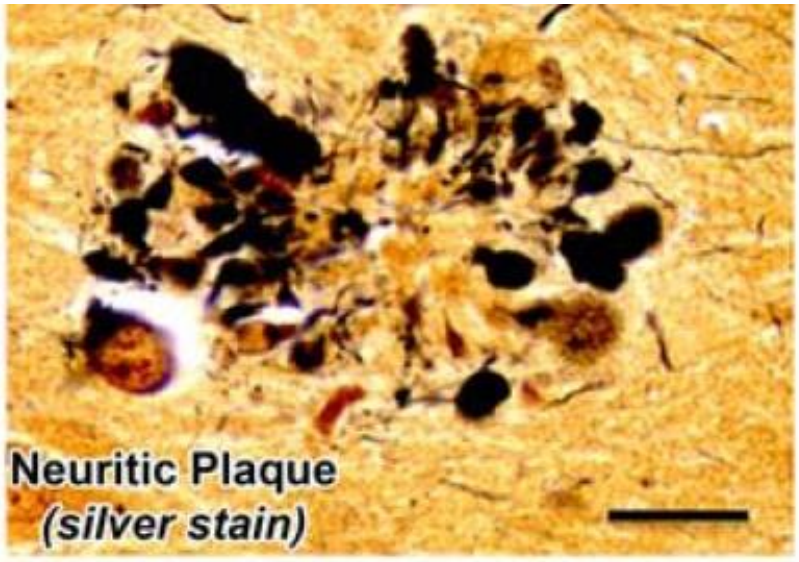
AD Facts

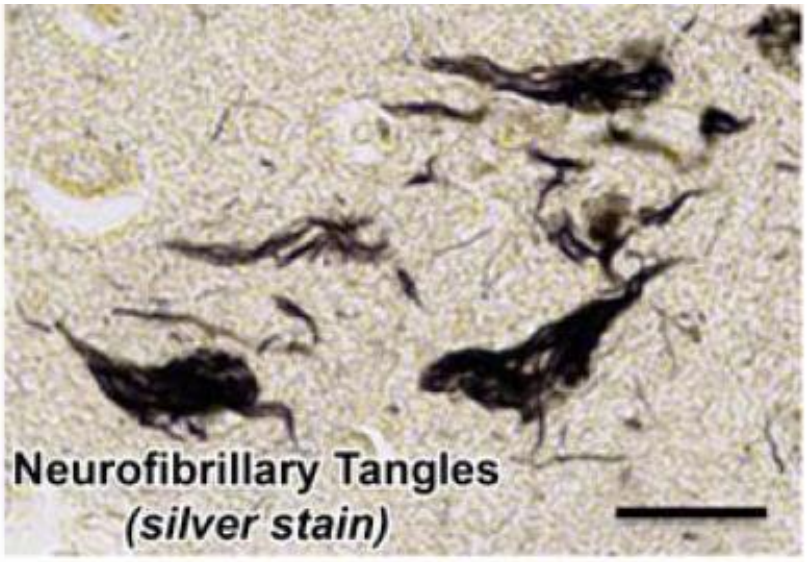
- An estimated half of million Poles age 65 and older are living with Alzheimer's dementia in 2020, over 1 million in 2050. Eighty percent are age 75 or older.
- One in 10 people age 65 has Alzheimer's dementia.
- Almost two-thirds of patients with Alzheimer's are women.
- Older African-Americans are about twice as likely to have Alzheimer's or other dementias as older whites.
- Hispanics are about one and one-half times as likely to have Alzheimer's or other dementias as older whites.

Alzheimer's disease diagnosis

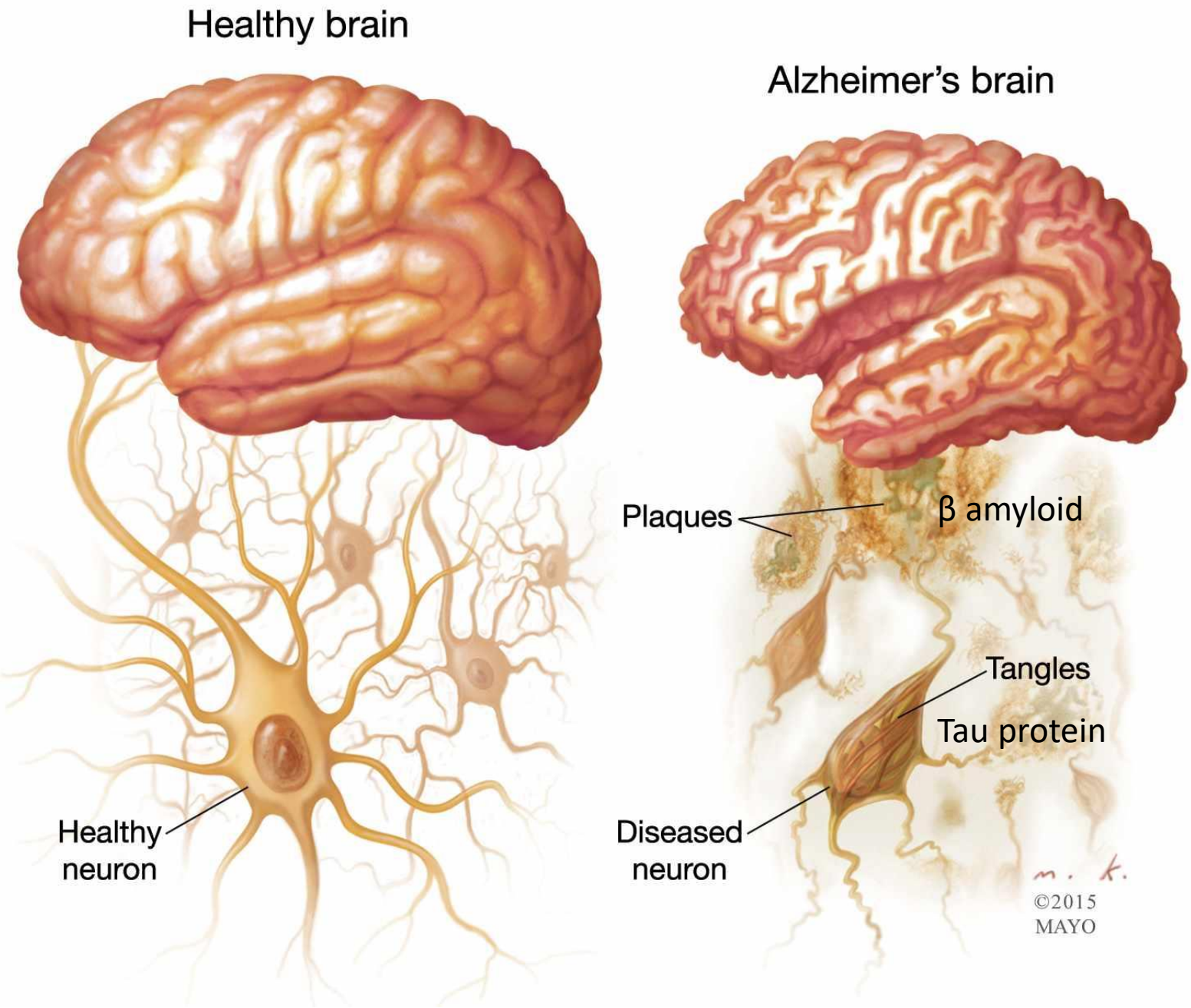
Brains affected by Alzheimer's disease often show presence of the following:

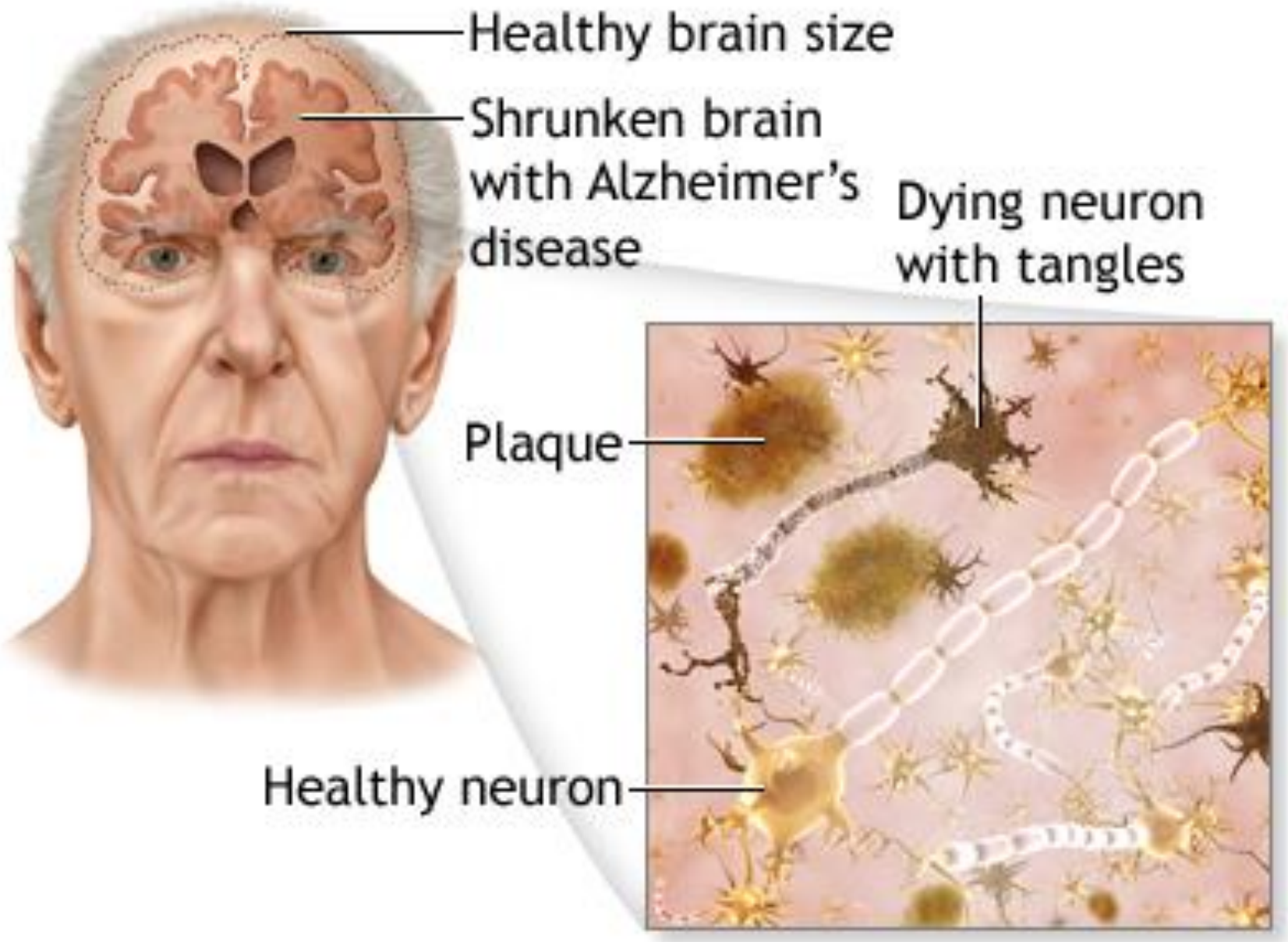
- Fiber tangles within nerve cells (neurofibrillary tangles)
- Clusters of degenerating nerve endings (neuritic plaques)

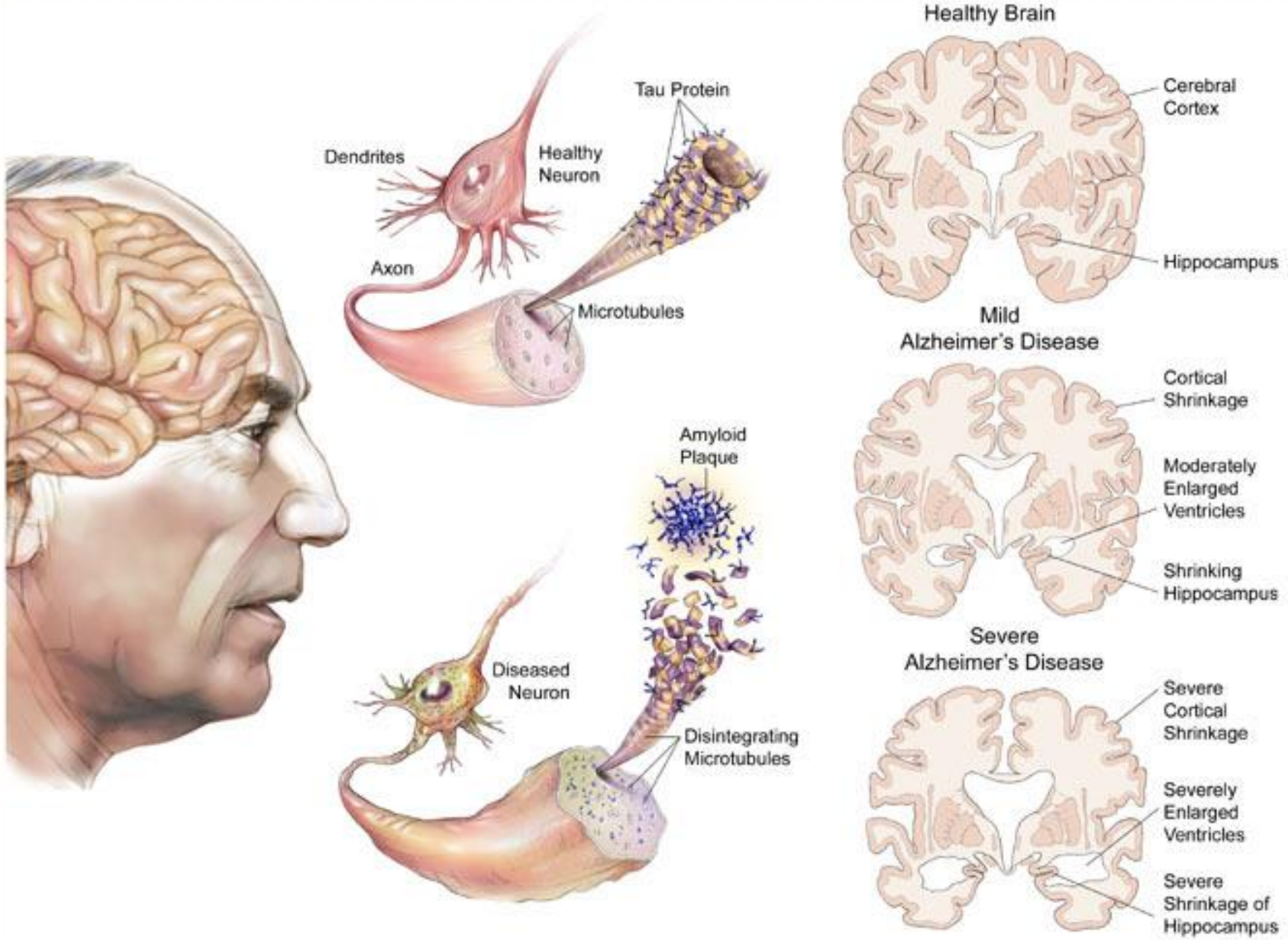


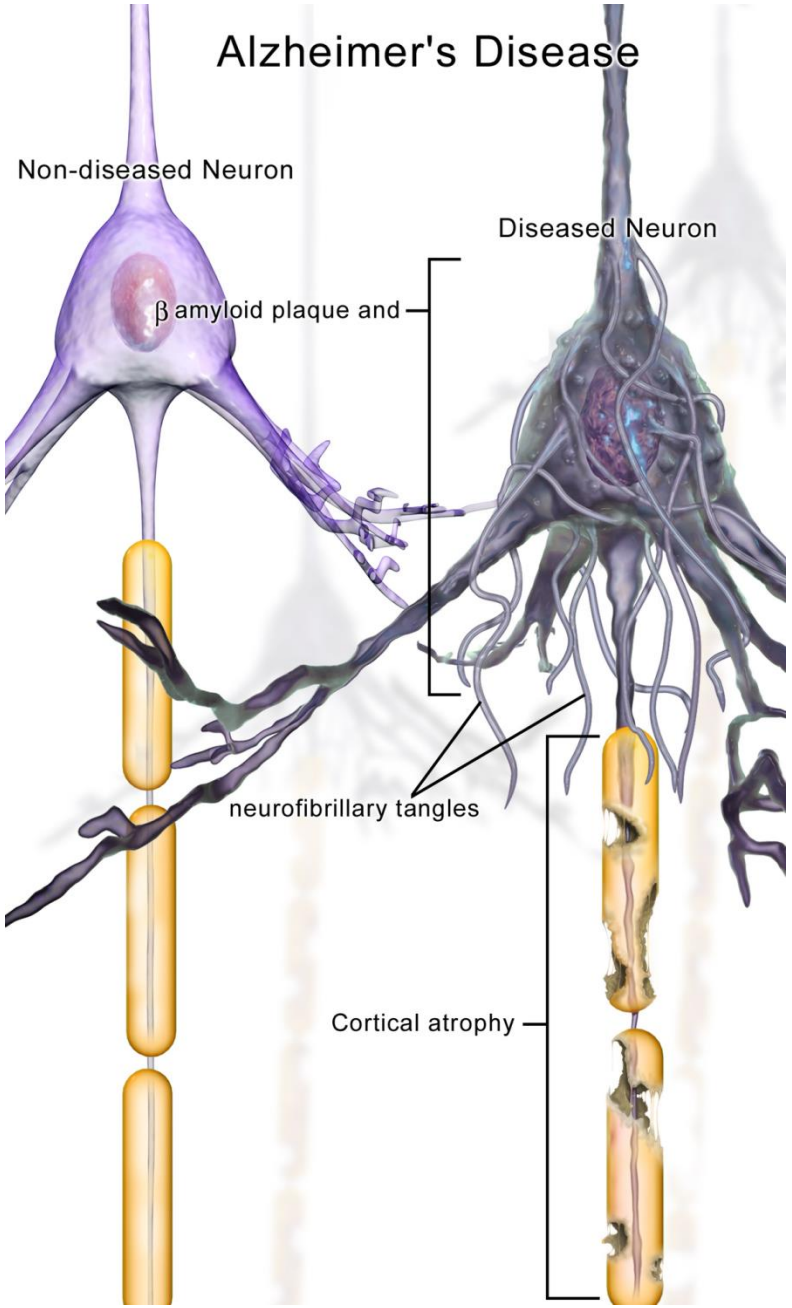


Also reduced production of acetylcholine, as well as norepinephrine, serotonin, and somatostatin is observed.


Healthy brain
Alzheimer's brain
Plaques
β amyloid
Tangles
Tau protein
Healthy neuron
Diseased neuron
©2015 MAYO

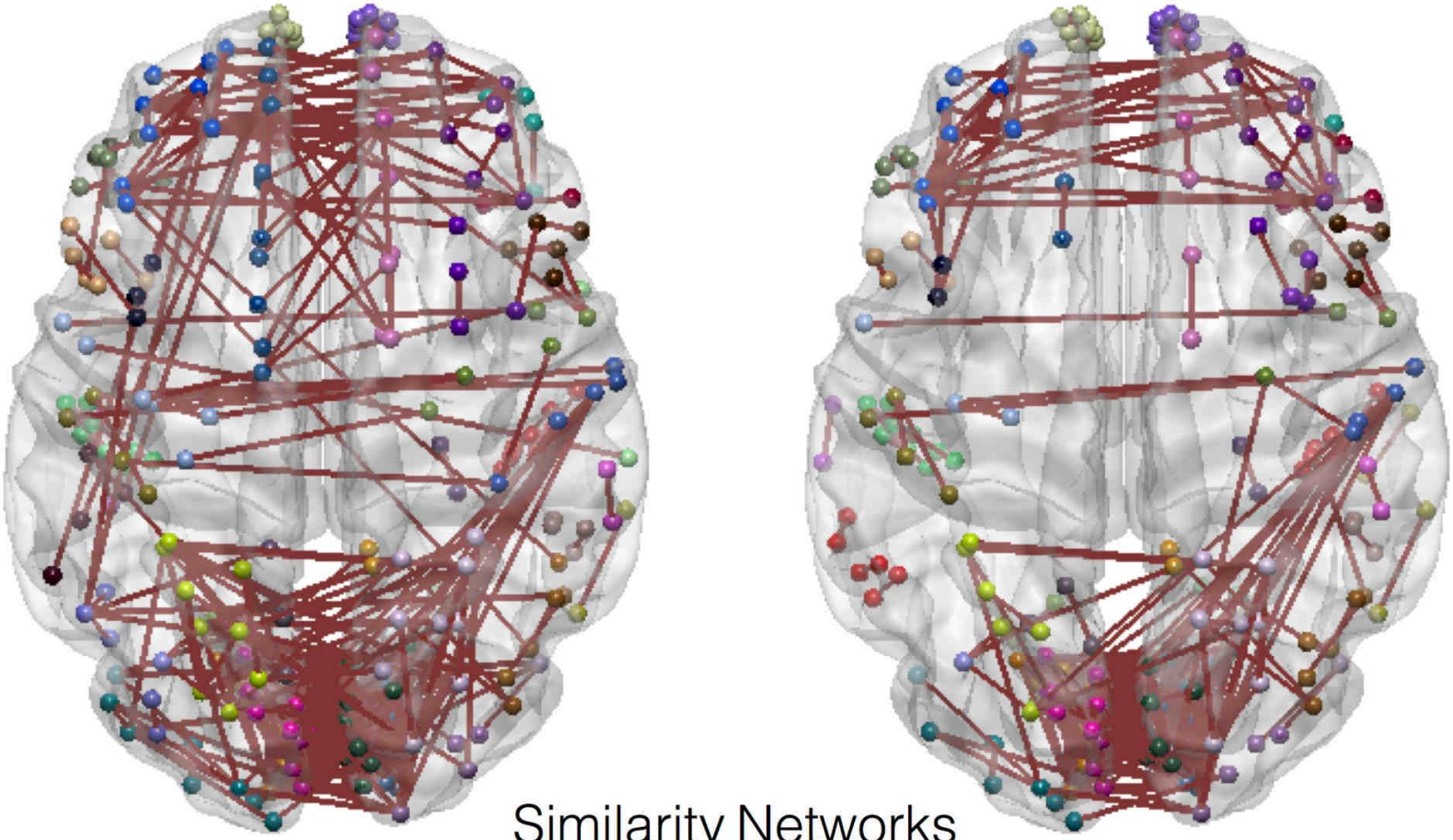

Healthy brain size
Shrunken brain with Alzheimer's disease
Dying neuron with tangles
Plaque
Healthy neuron


Tau Protein
Dendrites
Healthy Neuron
Axon
Microtubules
Amyloid Plaque
Diseased Neuron
Disintegrating Microtubules
Healthy Brain
Cerebral Cortex
Hippocampus
Mild Alzheimer's Disease
Cortical Shrinkage
Moderately Enlarged Ventricles
Shrinking Hippocampus
Severe Alzheimer's Disease
Severe Cortical Shrinkage
Severely Enlarged Ventricles
Severe Shrinkage of Hippocampus


Alzheimer's Disease
Non-diseased Neuron
Diseased Neuron
β amyloid plaque and
neurofibrillary tangles
Cortical atrophy

Healthy

Alzheimer's



Similarity Networks

ALHZHEIMERS

TOP 10 EARLY SIGNS

MEMORY LOSS

CHANGES IN MOOD

MISPLACING BELONGINGS

HARD TO COMPLETE FAMILIAR TASK

CONFUSION OF TIME AND PLACE

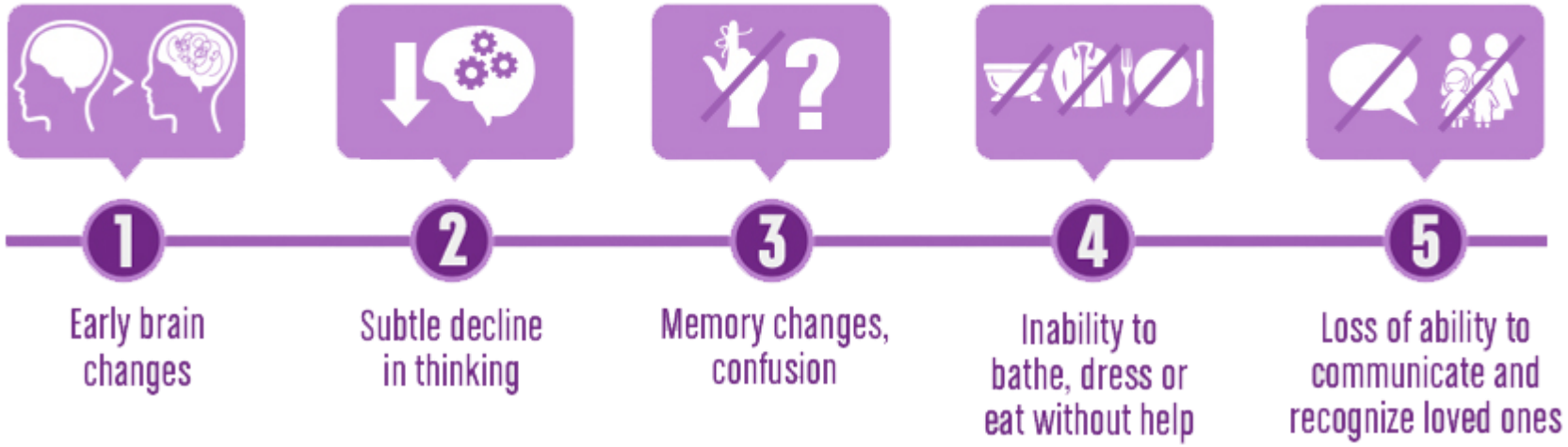
SOCIAL WITHDRAWAL

POOR JUDGEMENT

STRUGGLING TO COMMUNICATE

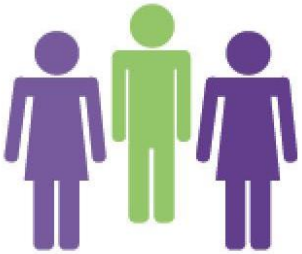
CHANGES IN VISION

THE ALZHEIMER'S TIMELINE



Diagnosis:
interview with patient
CT scan or MRI

Alzheimer's disease is the 6th leading cause of death in the United States and **KILLS MORE PEOPLE THAN BREAST CANCER AND PROSTATE CANCER COMBINED.**



ALMOST TWO THIRDS
of Americans with Alzheimer's disease are women.

EVERY
67
SECONDS
someone in the United States develops the disease.

SENIORS
dies with Alzheimer's or another dementia.

In thirteen years, Alzheimer's
DEATHS INCREASED BY
71%

By 2050, these costs could rise as high as **$1.1 TRILLION.**

Only
45%
of people with **ALZHEIMER'S** disease or their caregivers report **BEING TOLD OF THEIR DIAGNOSIS.**

More than
90%
of people with the four most common types of **CANCER** have been **TOLD OF THEIR DIAGNOSIS.**

Of America's top 10 causes of death, Alzheimer's disease is the only one that

CANNOT BE PREVENTED, CURED OR SLOWED.

REVIEW

NO PREVENTION

NO CURE

NO SLOWING

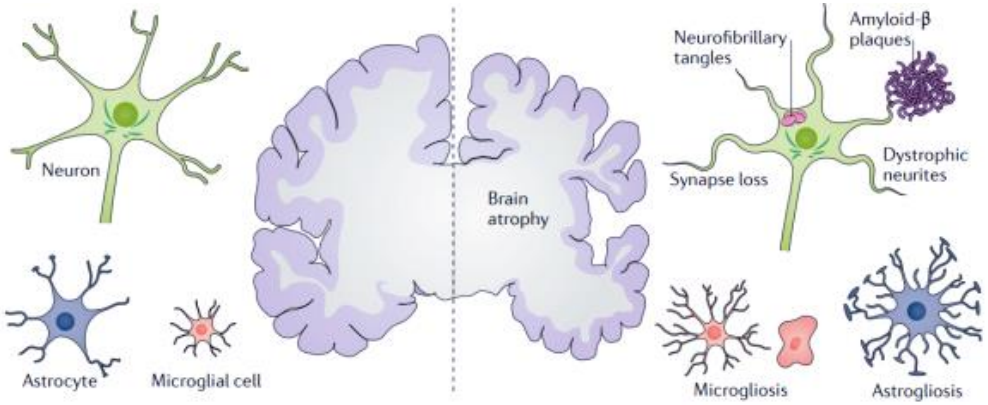
250,000

CHILDREN & YOUNG ADULTS

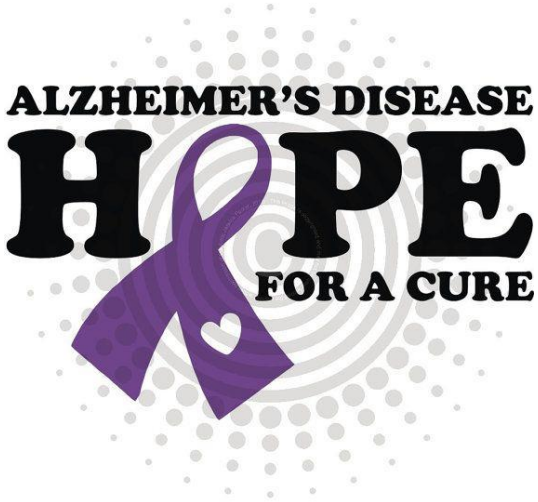
between ages 8 and 18 provide help to someone with Alzheimer's disease or another dementia.

Alzheimer's disease treatment

- Ephexin 5 protein appears in greater amounts in the brains of people with Alzheimer's disease. Blocking this protein in mice seems to prevent the development of memory loss.



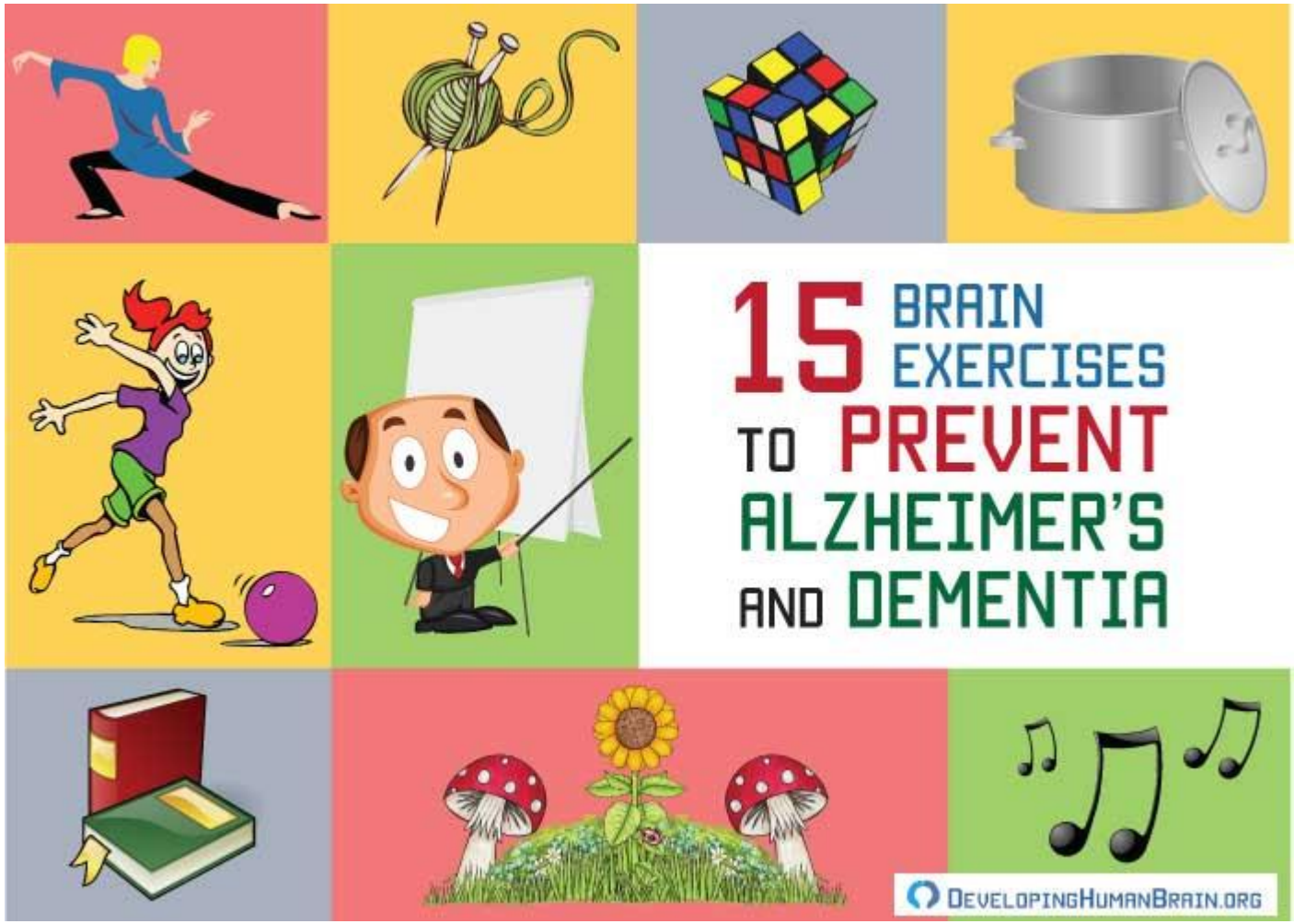
- Cholinesterase inhibitors are prescribed for mild AD, as they help to reduce some symptoms and control some behavioral symptoms. Razadyne® (galantamine), Exelon® (rivastigmine), and Aricept® (donepezil) block the breakdown of acetylcholine, a brain chemical believed to be important for memory and thinking.



Alzheimer's disease treatment

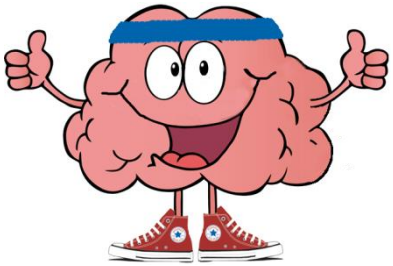
- Namenda® (memantine), an N-methyl D-aspartate (NMDA) antagonist, is prescribed to treat moderate to severe AD. This drug's main effect is to maintain daily functions a little longer (ability to use the bathroom independently for several more months). The FDA has also approved Aricept®, the Exelon® patch, and Namzaric®, a combination of Namenda® and Aricept®.

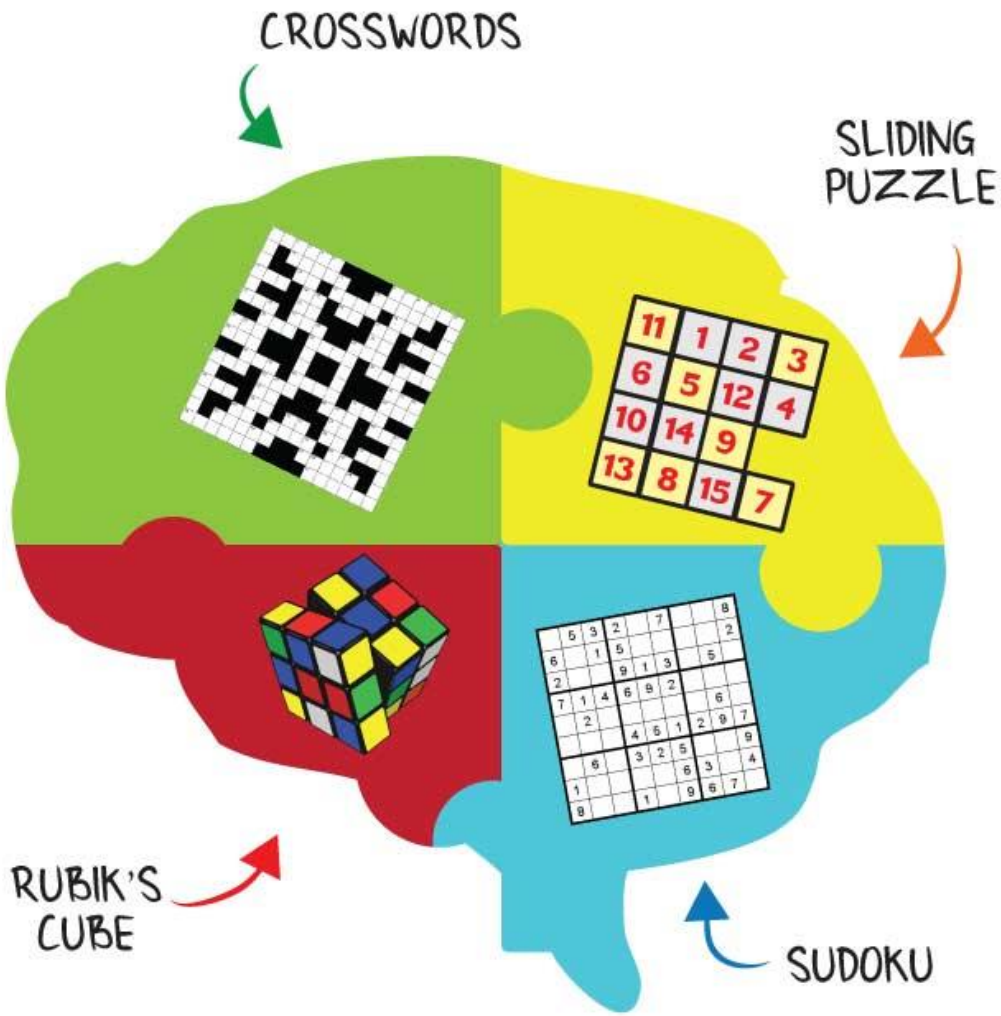
- Common behavioral symptoms of Alzheimer's include sleeplessness, wandering, agitation, anxiety, aggression, restlessness, and depression. Sleep aids, Anti-anxiety drugs (to treat agitation), Anticonvulsants (severe aggression), Antipsychotics (paranoia, hallucinations, agitation, and aggression).


15 BRAIN EXERCISES TO PREVENT ALZHEIMER'S AND DEMENTIA
DevelopingHumanBrain.org

HOW TO EXERCISE YOUR BRAIN ?

DevelopingHumanBrain.org





PUZZLES THAT STIMULATE OUR BRAIN

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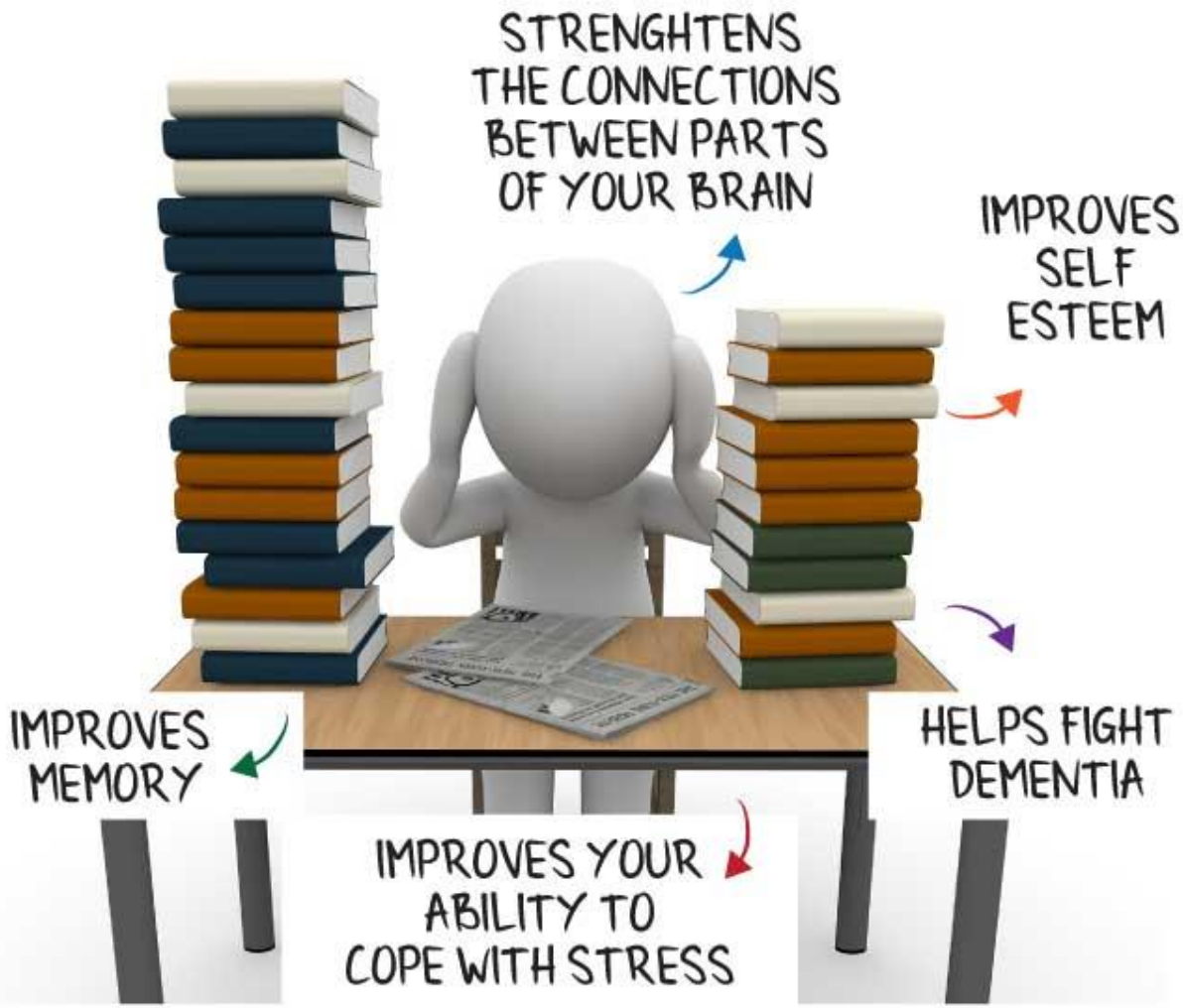

LEARNING A NEW SKILL AND BRAIN HEALTH

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READING AND BRAIN HEALTH

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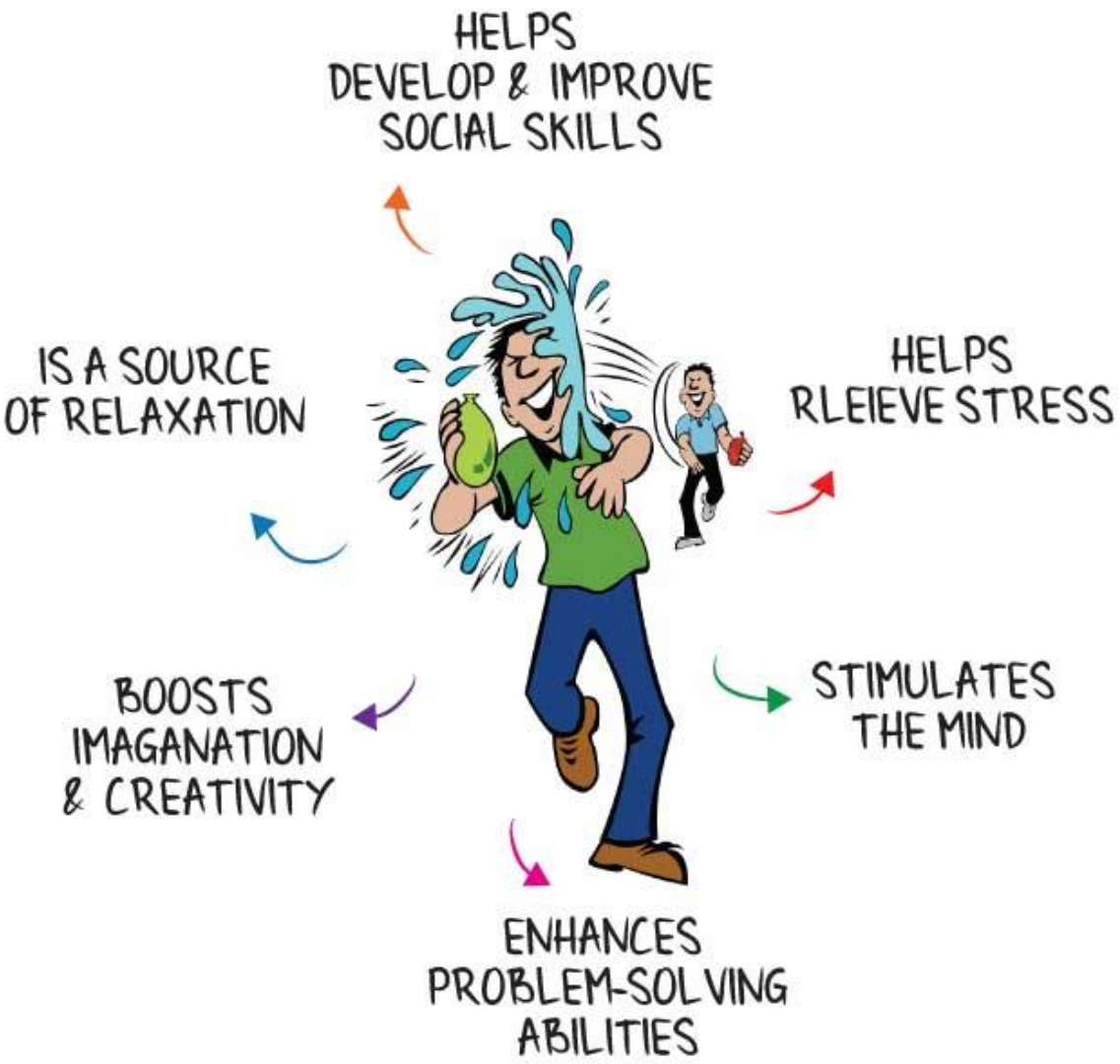

PLAY IS NOT JUST FOR KIDS

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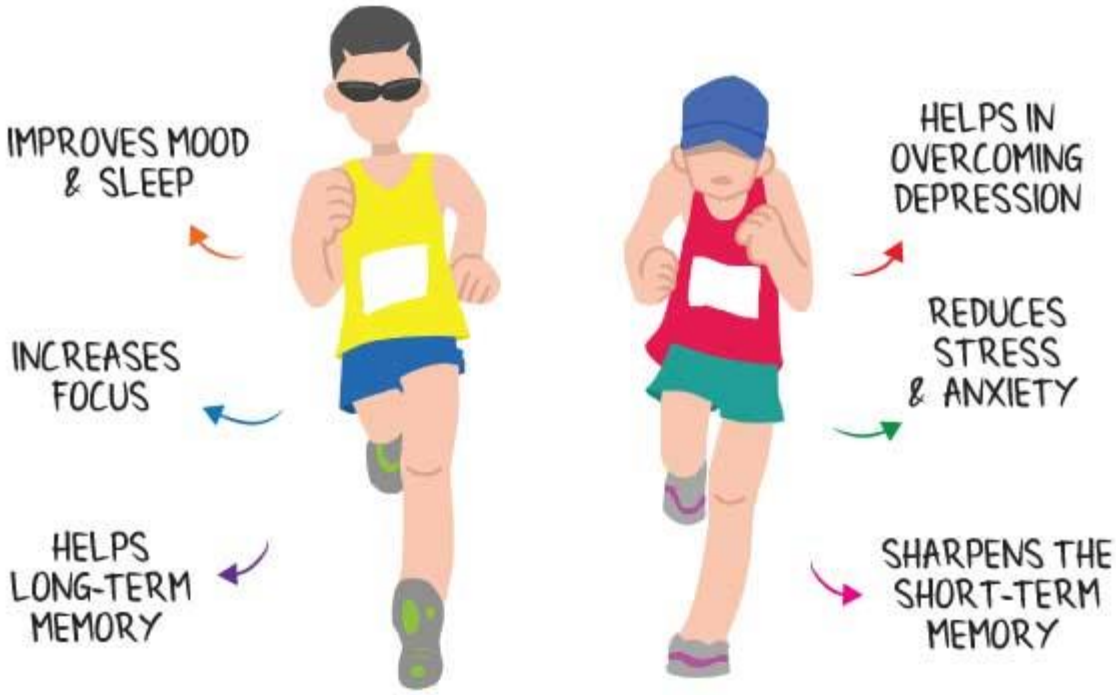

PHYSICAL EXERCISE IS THE KEY TO BRAIN HEALTH

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GARDENING AND BRAIN HEALTH

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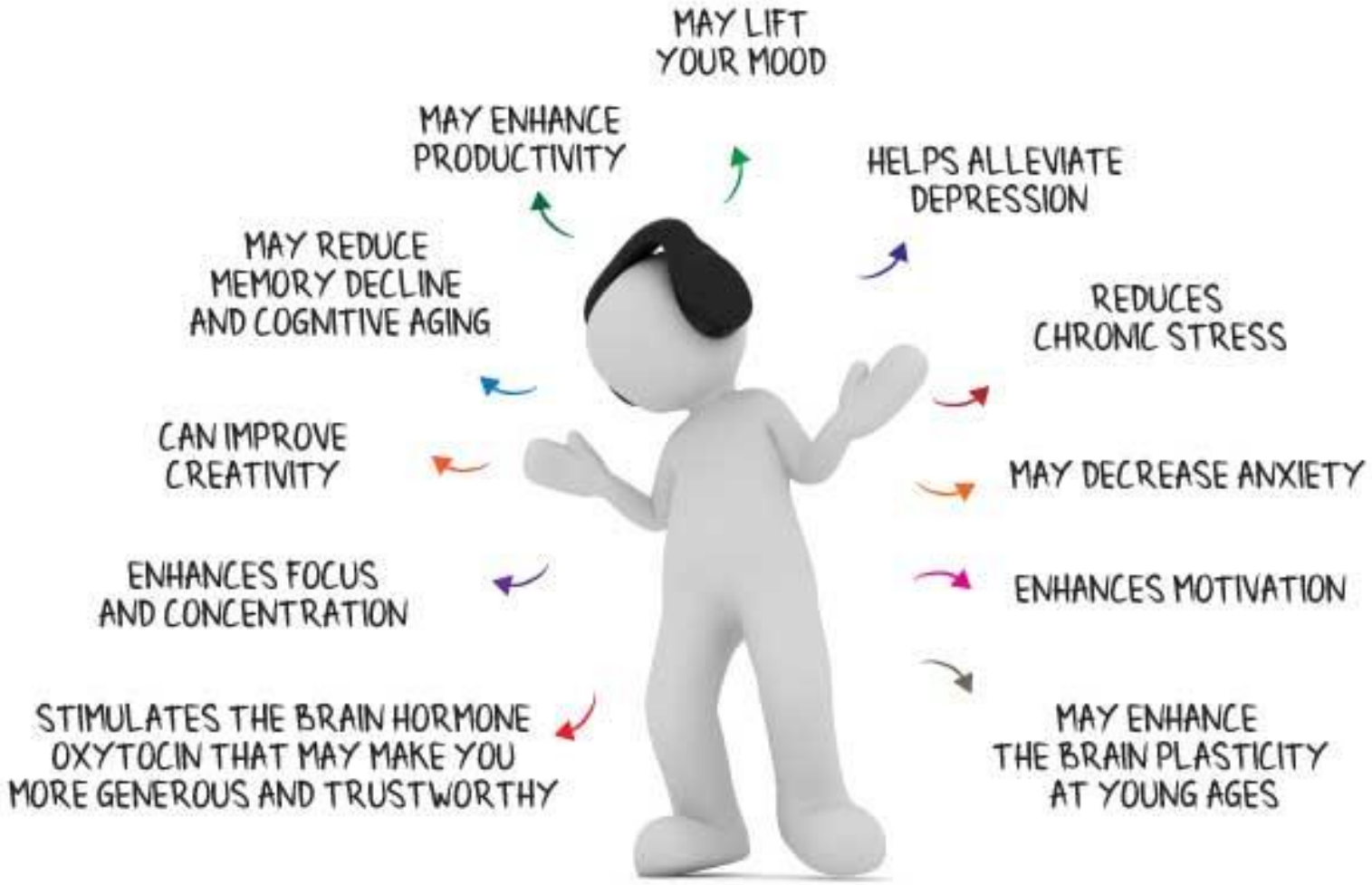

LISTENING TO MUSIC AND BRAIN HEALTH

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COOKING SOMETHING NEW?

HOW IT AFFECTS YOUR BRAIN?

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YOGA AND BRAIN HEALTH

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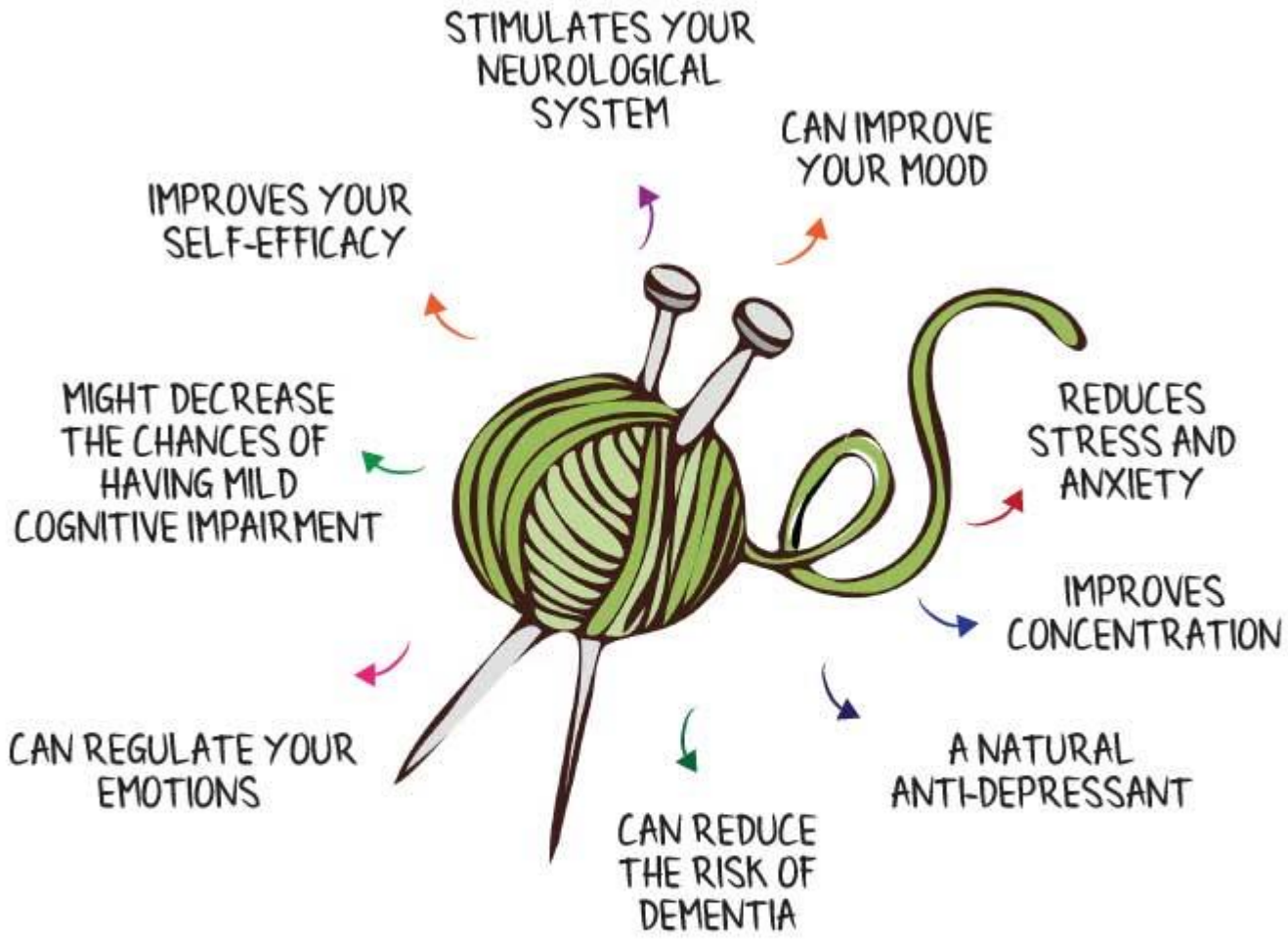
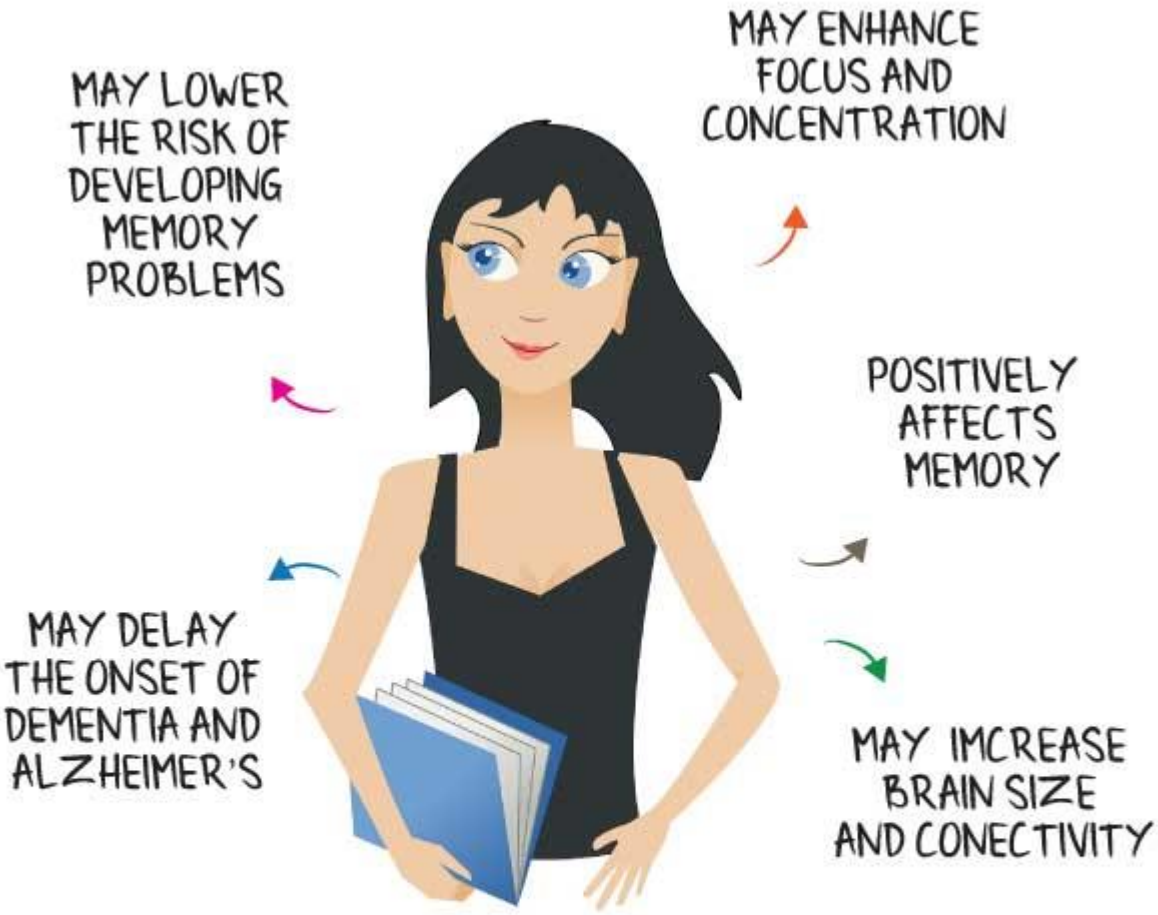


CRAFTING AND BRAIN HEALTH

DEVELOPINGHUMANBRAIN.ORG



LEARNING A NEW LANGUAGE AND THE BRAIN

DevelopingHumanBrain.org

Dementia.
How to reduce the risk?

Look after your heart

Be physically active

Follow a healthy diet

Challenge your brain

Enjoy social activity

Growing evidence indicates that people can reduce their risk of cognitive decline by adopting key lifestyle habits. When possible, combine these habits to achieve maximum benefit for the brain and body.

HIT THE BOOKS

Formal education will help reduce risk of cognitive decline and dementia. Take a class at a local college, community center or online.

BUTT OUT

Smoking increases risk of cognitive decline. Quitting smoking can reduce risk to levels comparable to those who have not smoked.

BREAK A SWEAT

Engage in regular cardiovascular exercise that elevates heart rate and increases blood flow. Studies have found that physical activity reduces risk of cognitive decline.

FOLLOW YOUR HEART

Risk factors for cardiovascular disease and stroke – obesity, high blood pressure and diabetes – negatively impact your cognitive health.

STUMP YOURSELF

Challenge your mind. Build a piece of furniture. Play games of strategy, like bridge.

HEADS UP!

Brain injury can raise risk of cognitive decline and dementia. Wear a seat belt and use a helmet when playing contact sports or riding a bike.

BUDDY UP

Staying socially engaged may support brain health. Find ways to be part of your local community or share activities with friends and family.

FUEL UP RIGHT

Eat a balanced diet that is higher in vegetables and fruit to help reduce the risk of cognitive decline.

TAKE CARE OF YOUR MENTAL HEALTH

Some studies link depression with cognitive decline, so seek treatment if you have depression, anxiety or stress.

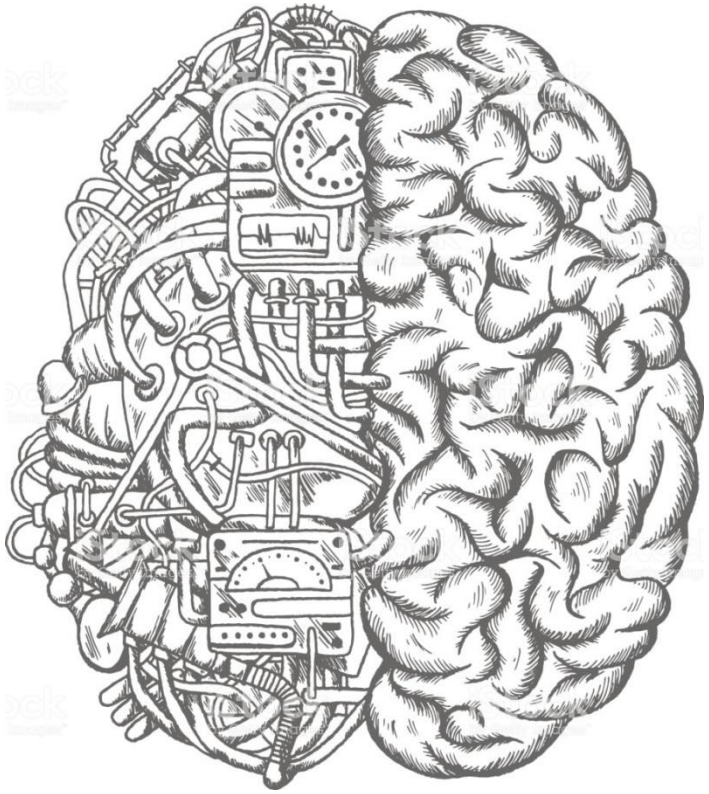
CATCH SOME ZZZ'S

Not getting enough sleep may result in problems with memory and thinking.

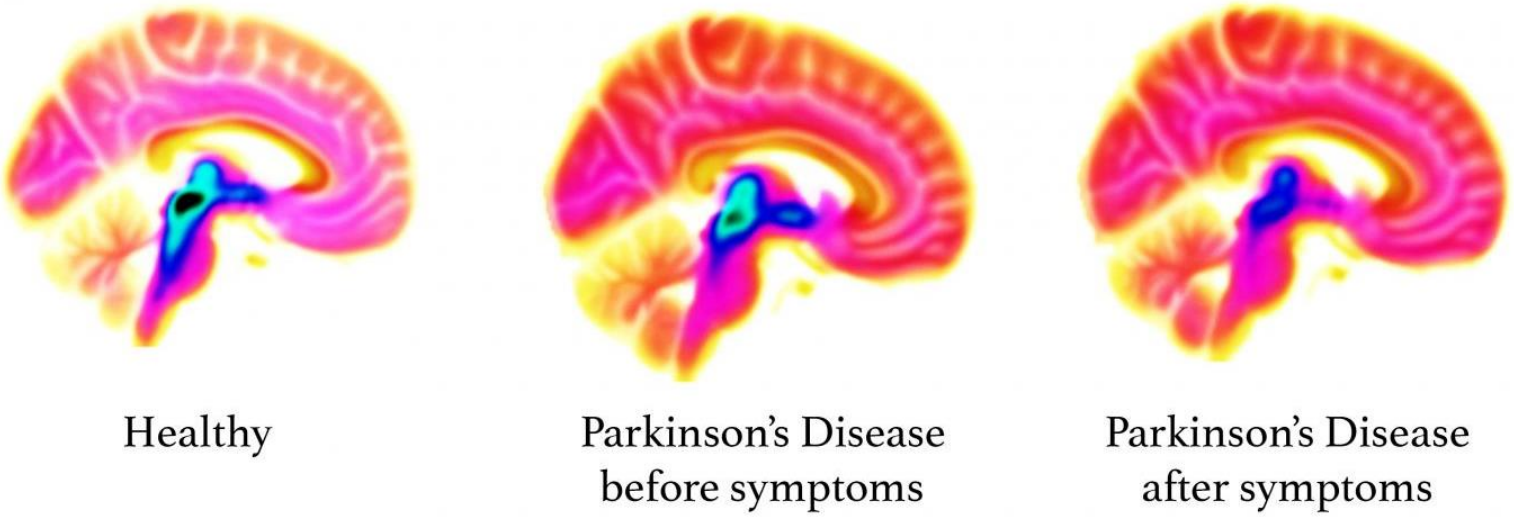

Memories are Priceless
Help Cure Alzheimers

Neurodegenerative diseases

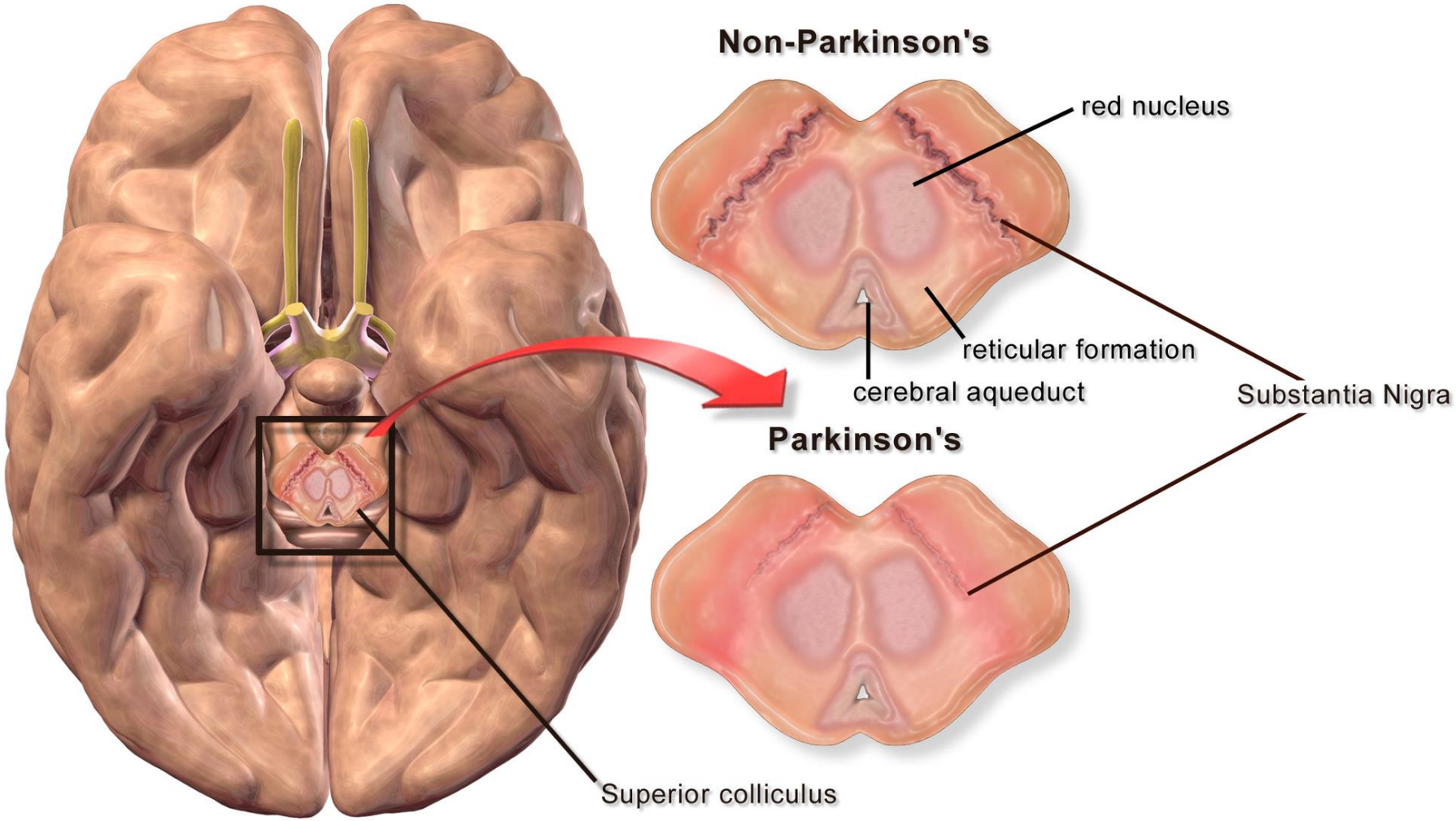
- Alzheimer's AD
- Parkinson's PD
- Huntington's HD
- Spinocerebellar ataxia SCA
- Prion disease
- Motor neurone diseases MND



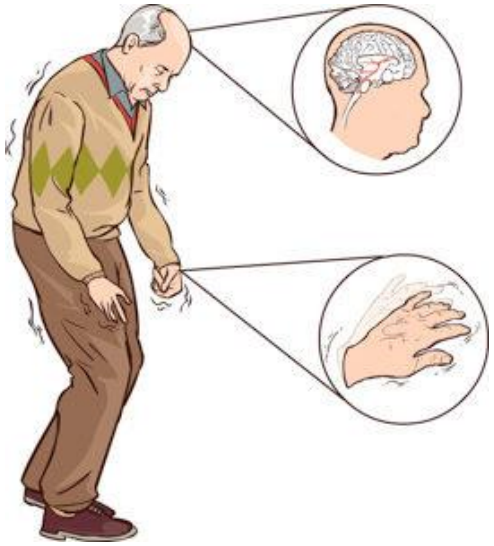
Parkinson’s disease PD



PD progresses slowly as small clusters of neurons in the midbrain die. The gradual loss of these neurons reduces levels of a chemical, called dopamine, which is responsible for transmitting messages to the parts of the brain that coordinate muscle movement.


Parkinsons Disease
Non-Parkinson's
red nucleus
reticular formation
cerebral aqueduct
Substantia Nigra
Parkinson's
Superior colliculus

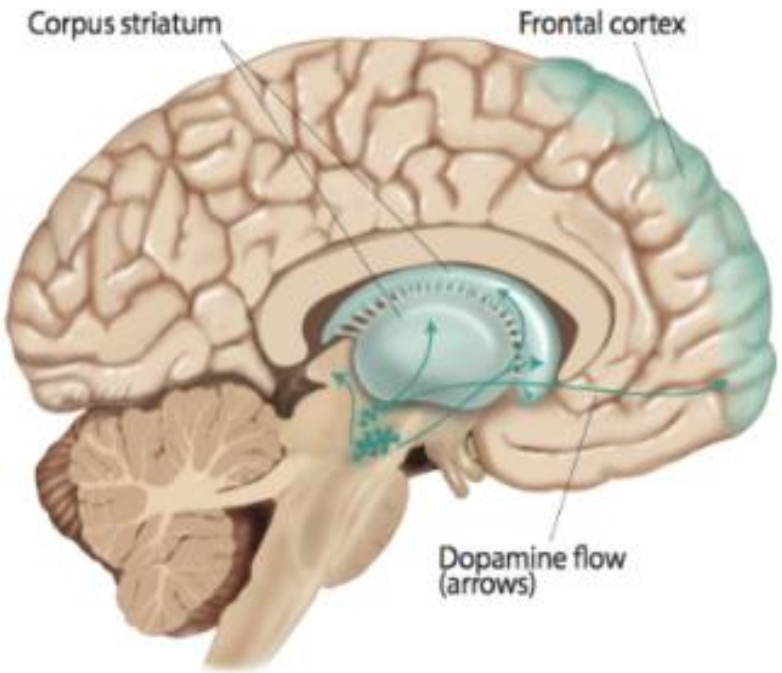
Parkinson’s disease PD

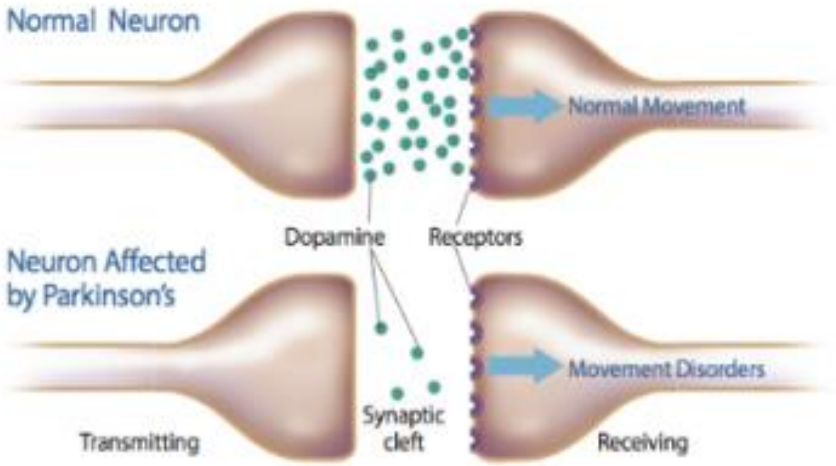


Common symptoms include tremors or shaking in hands, arms, legs, jaw and face; rigidity or stiffness of the limbs and trunk; bradykinesia, or slowness of movement; and difficulties with balance, speech and coordination.

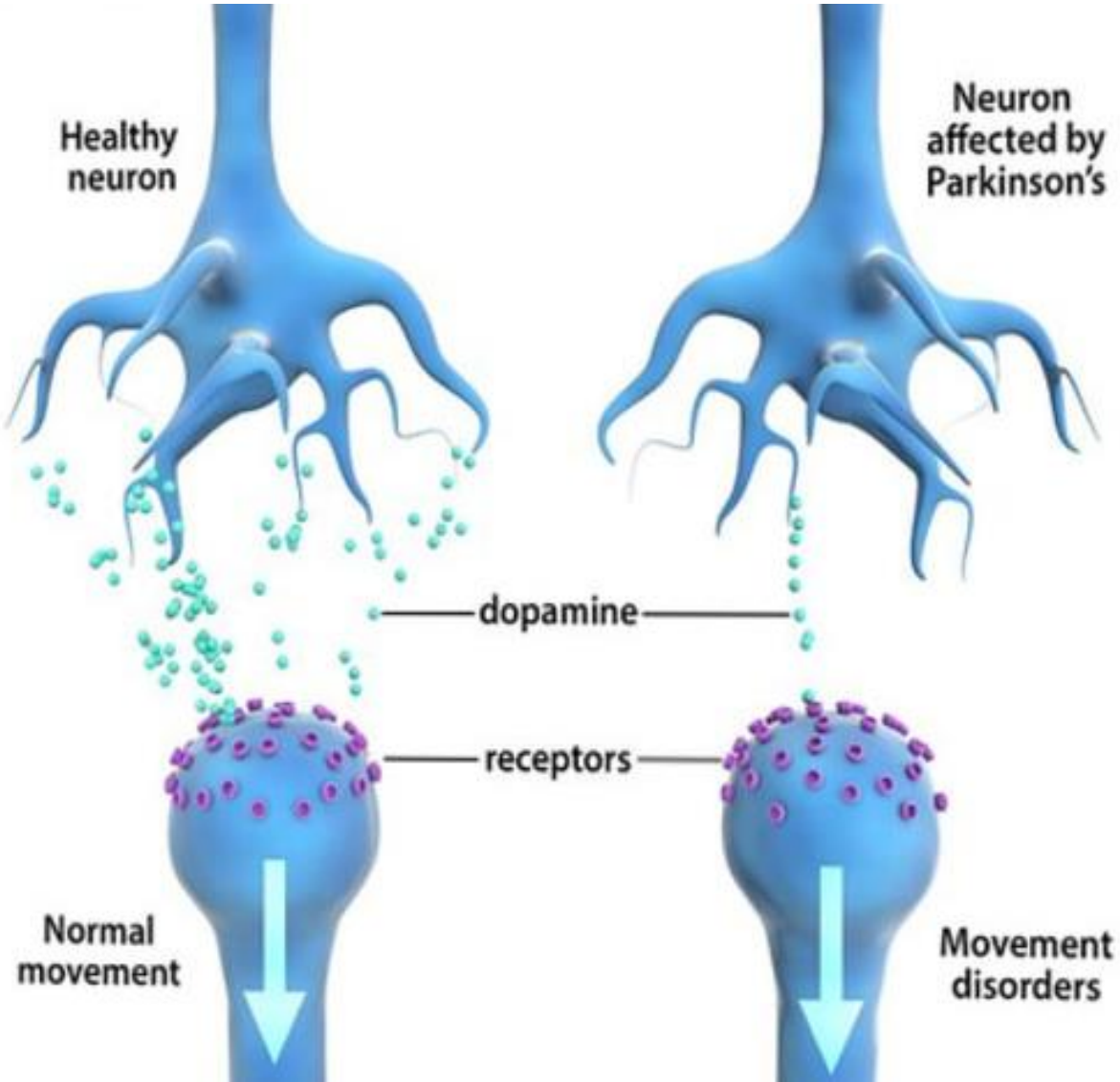
Symptoms of PD begin gradually and typically worsen over time.

Parkinson's disease mechanism

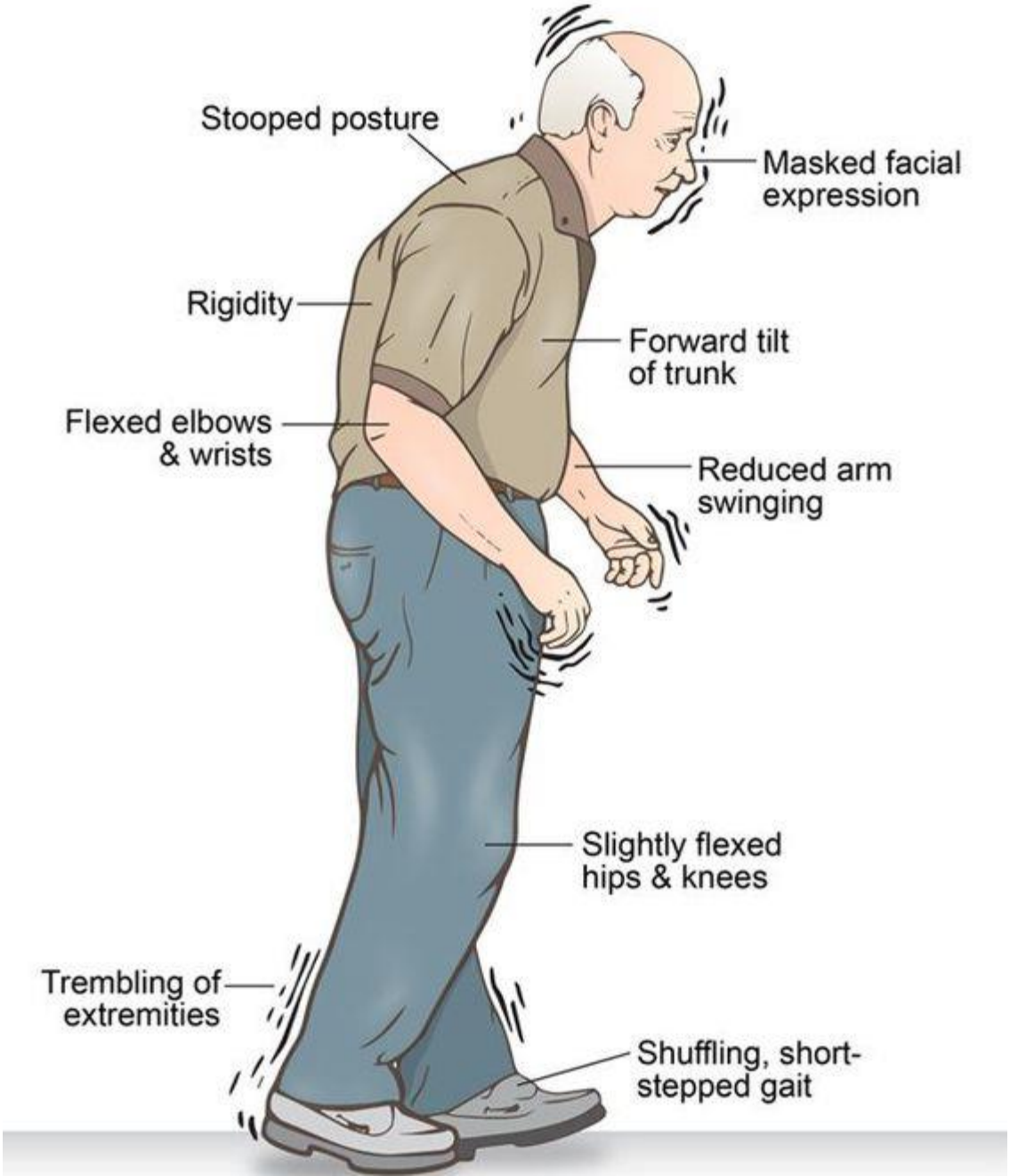




Parkinson's disease mechanism

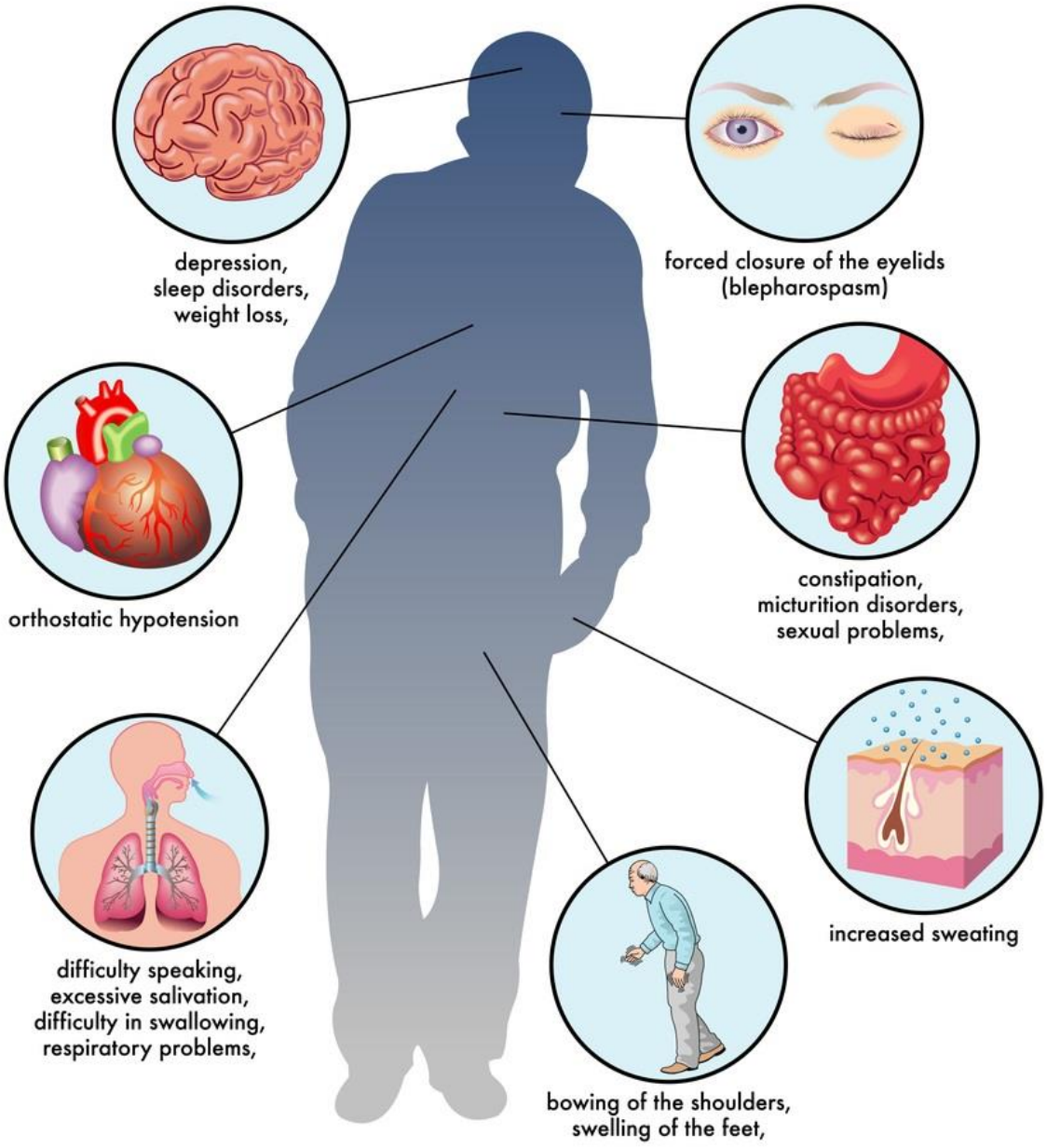


Typical appearance of Parkinson's disease

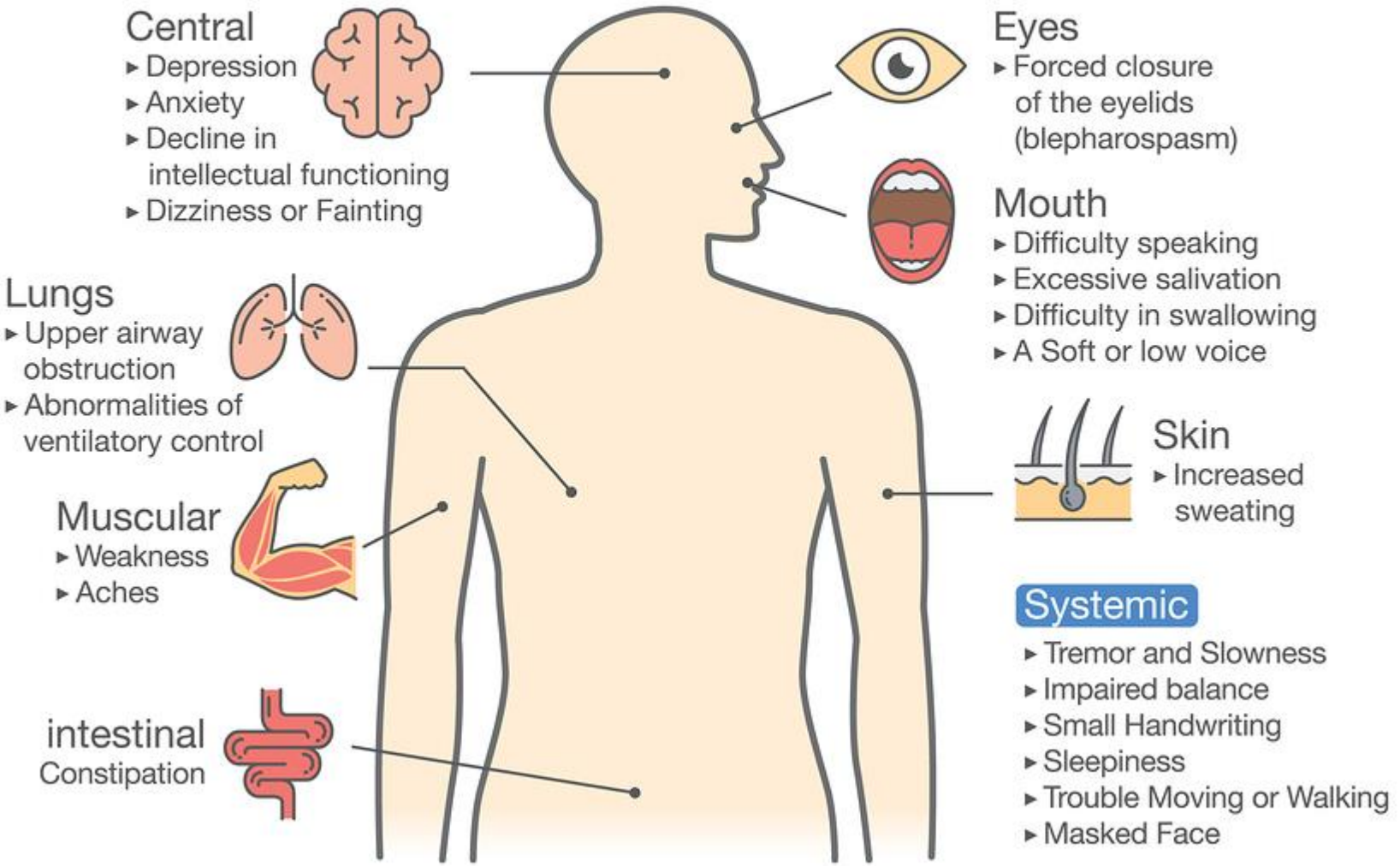


Parkinson's disease

non-motor disorders caused by Parkinson's disease

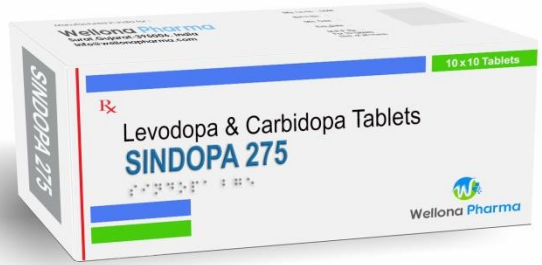


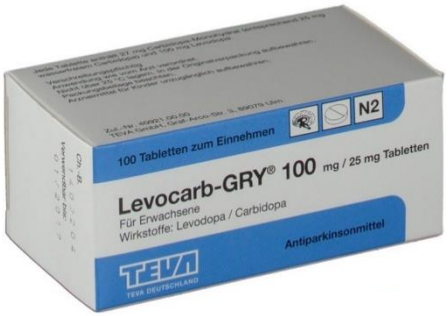
Symptoms of Parkinson's Disease

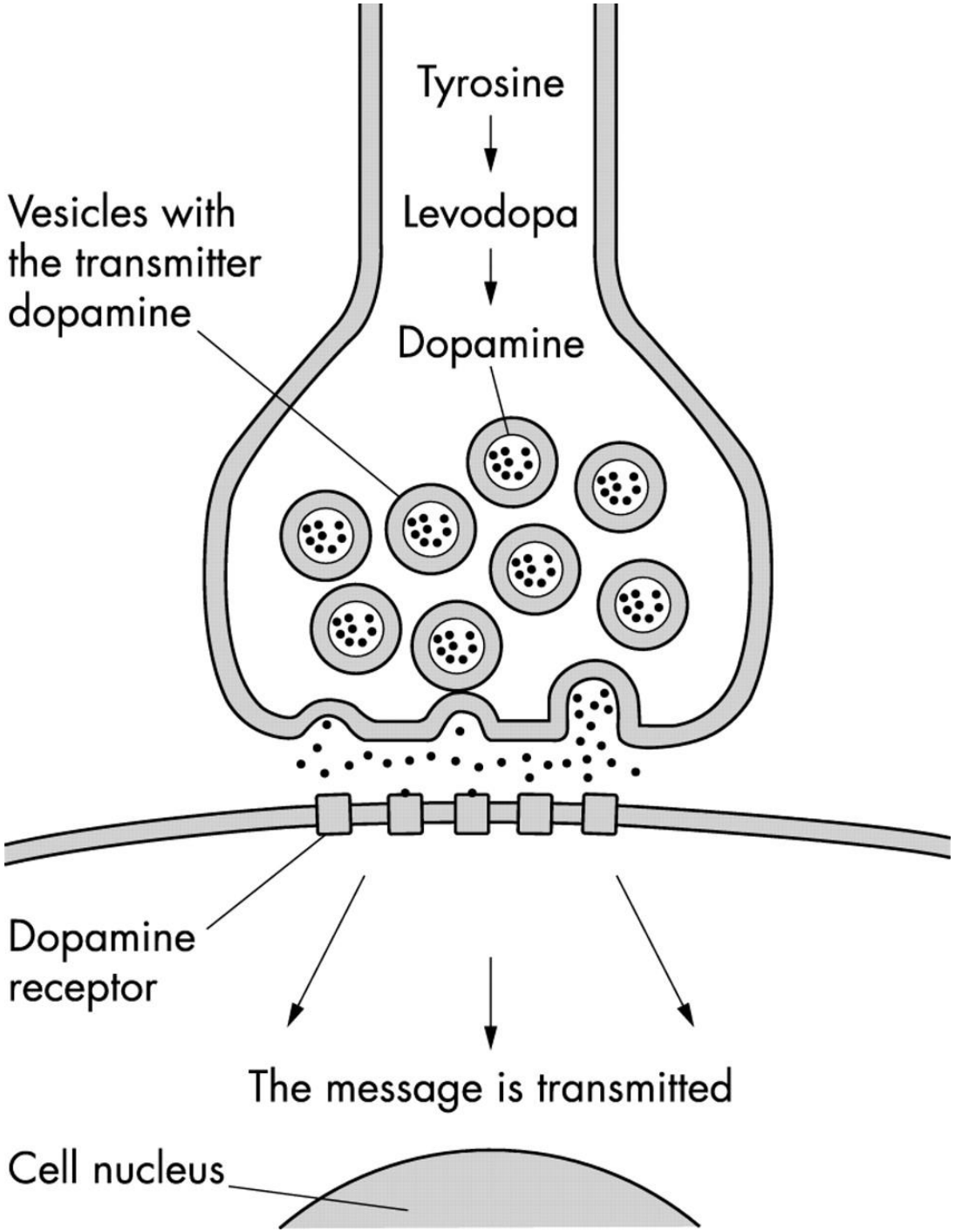


History of Parkinson's disease

- 1817: description of symptoms by James Parkinson
- 1862: Coined the name “Parkinson’s Disease” by Jean-Martin Charcot
- 1919: Degeneration of substantia nigra
- 1968: L-dopa was introduced
- 1979: MPTP was found to cause Parkinsonism
- 1997: PARK1 gene mutation was discovered

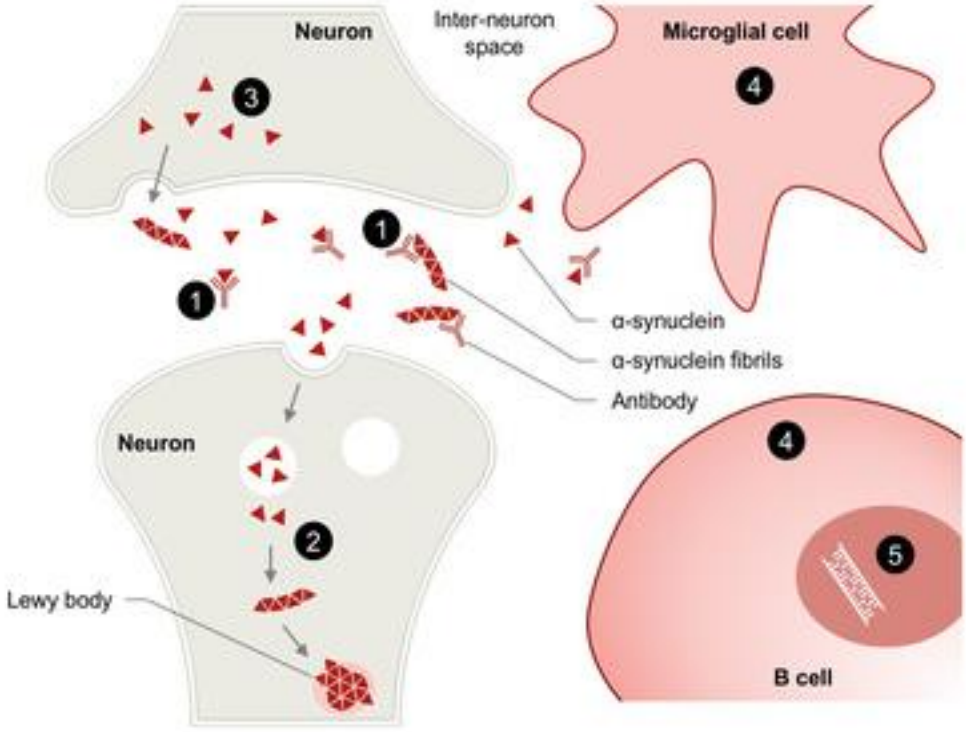

Wellona Pharma
10 x 10 Tablets
Levodopa & Carbidopa Tablets
SINDOPA 275
Wellona Pharma
SINDOPA 275


100 Tabletten zum Einnehmen
N2
Levocarb-GRY® 100 mg / 25 mg Tabletten
Für Erwachsene
Wirkstoffe: Levodopa / Carbidopa
Antiparkinsonmittel
TEVA
TEVA DEUTSCHLAND


Tyrosine
Levodopa
Dopamine
Vesicles with the transmitter dopamine
Dopamine receptor
The message is transmitted
Cell nucleus

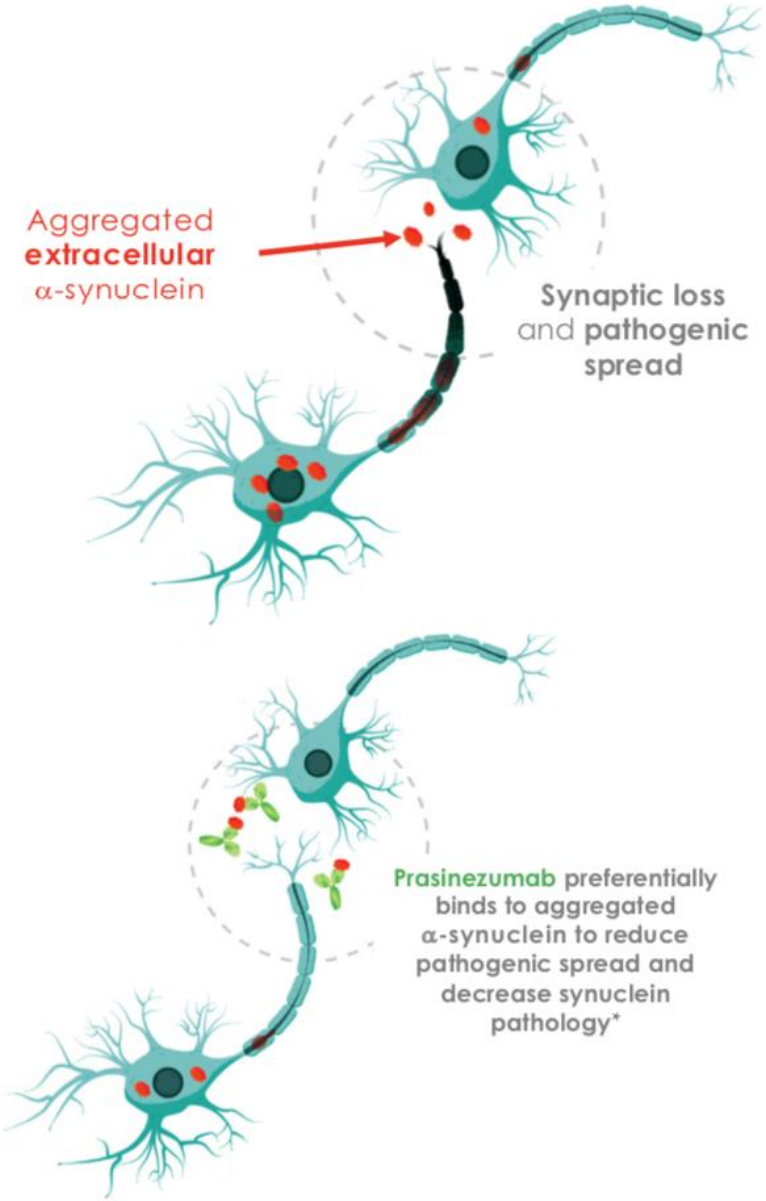
Novel therapeutic strategies for Parkinson's disease

Possible targets for PD drug development



1. Targeting extracellular α-synuclein – with antibodies that would prevent cell-to-cell transmission of α-synuclein
2. Targeting multimerization of α-synuclein – drugs that would prevent the α-synuclein combining to form fibrils
3. Targeting intracellular α-synuclein – to restore proper α-synuclein handling inside the neuron, drugs might enhance signalling or breakdown processes within the cell
4. Targeting neuroinflammation – the immune system is key in PD, so drugs that modulate immune action may be useful in the treatment of PD
5. Gene therapy – new treatment modalities could make manipulation of the genome a possibility

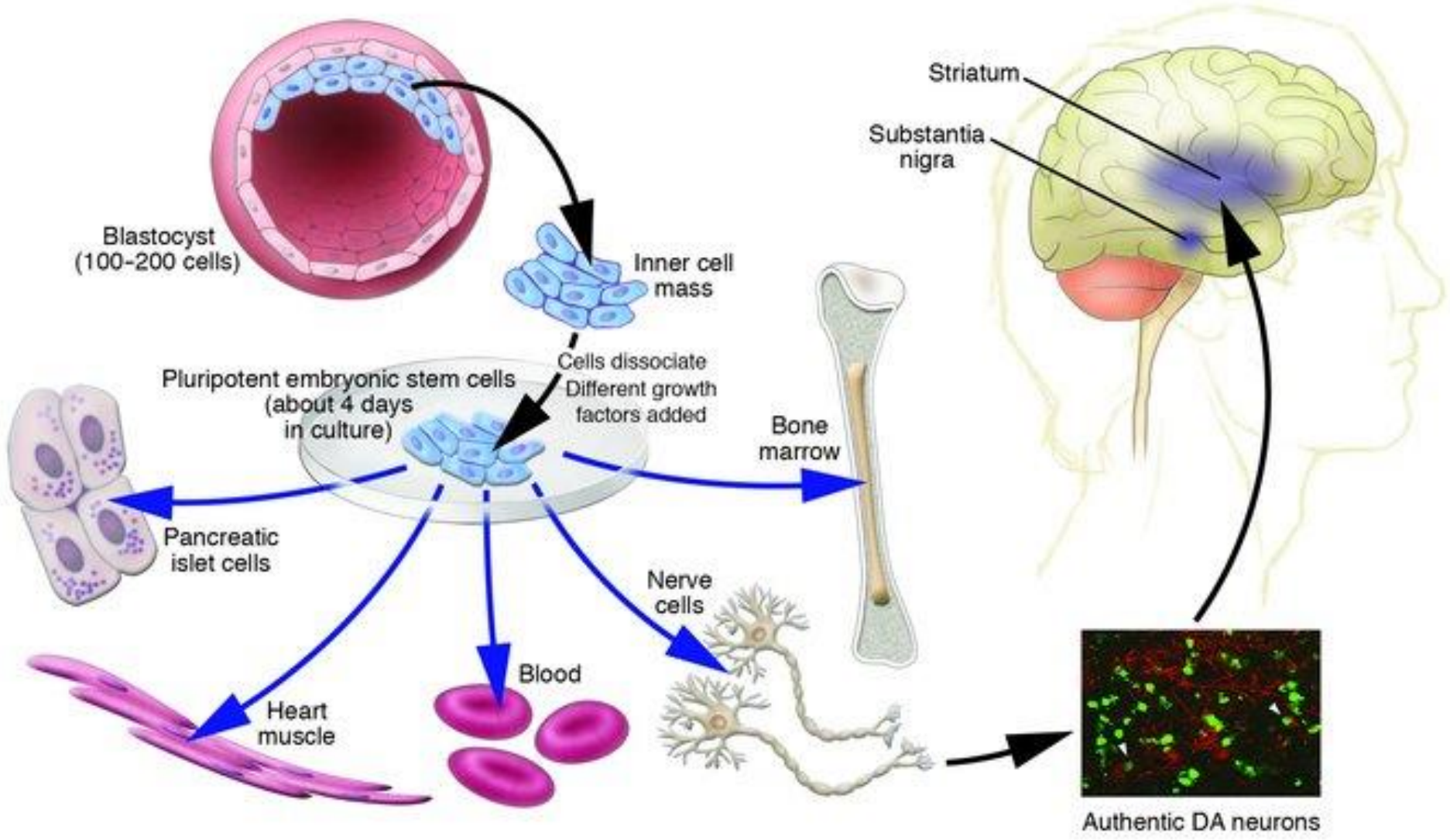
Others – including antioxidants, enzyme inhibitors, and metal-chelators



*Proposed mechanism of action

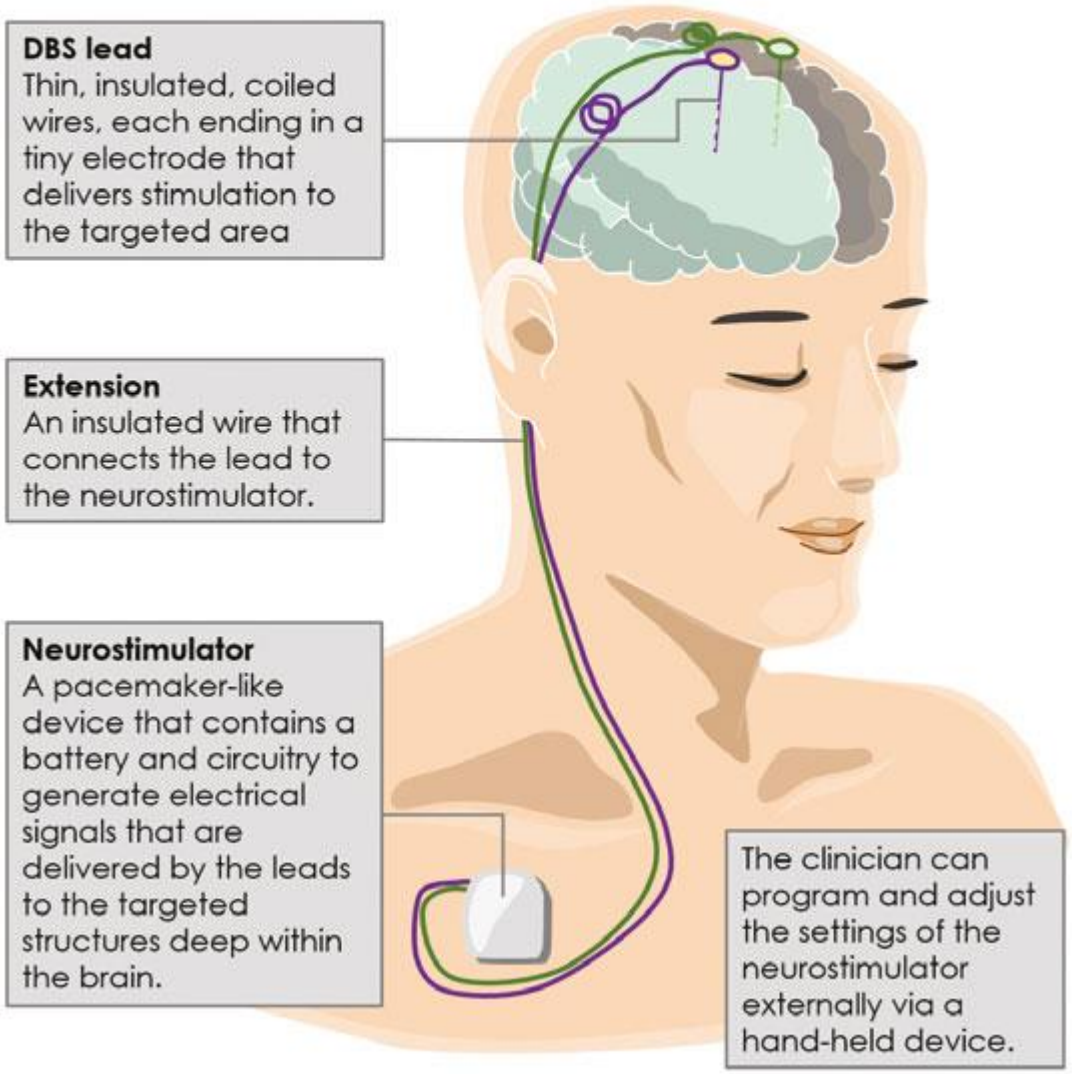
Prothena's investigational therapy prasinezumab is designed to slow or reduce the neurodegeneration associated with alpha-synuclein misfolding and/or its transmission, a process highly implicated in Parkinson disease pathology.

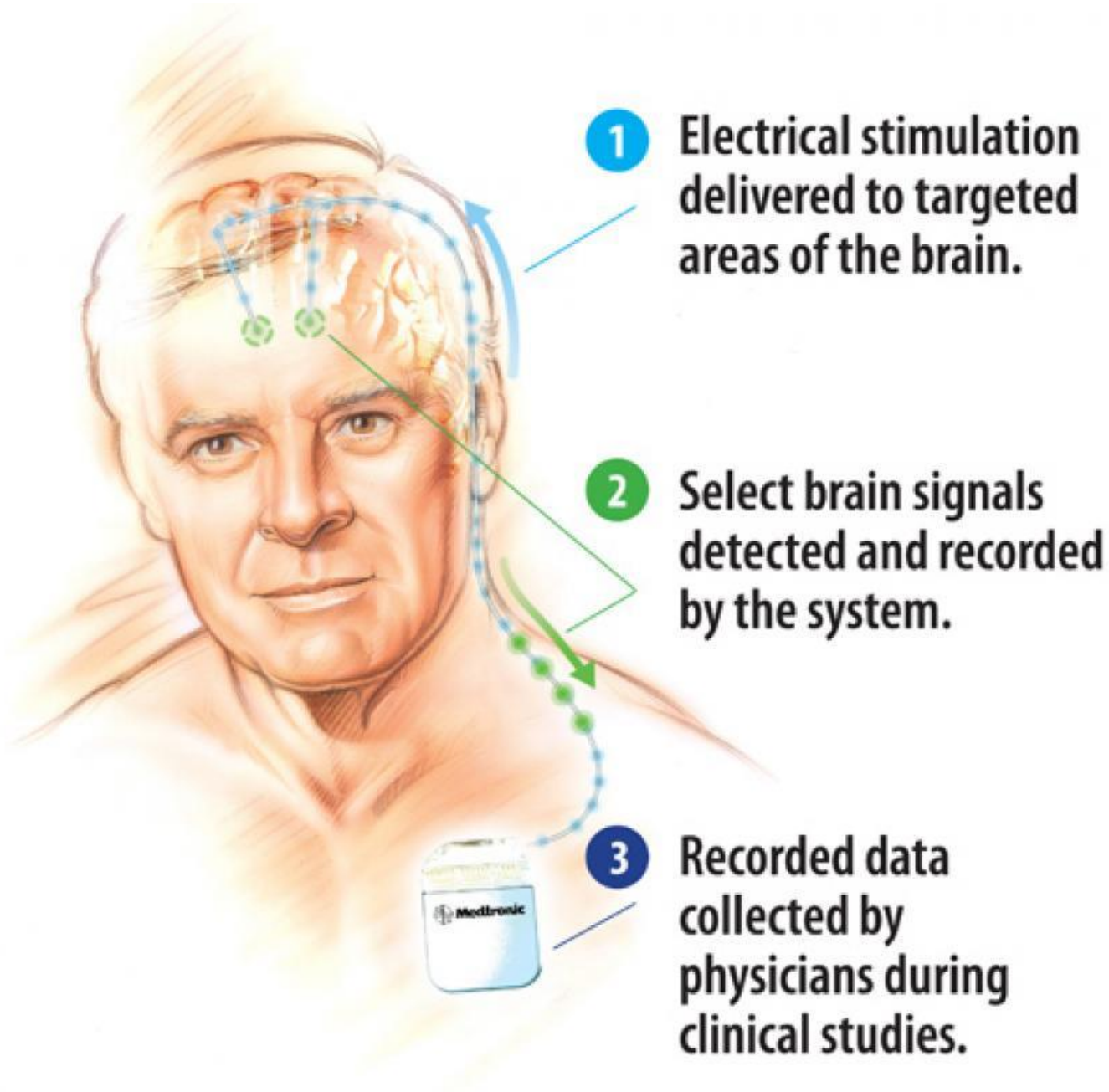
Stem cells treatment



Deep brain stimulation (DBS)

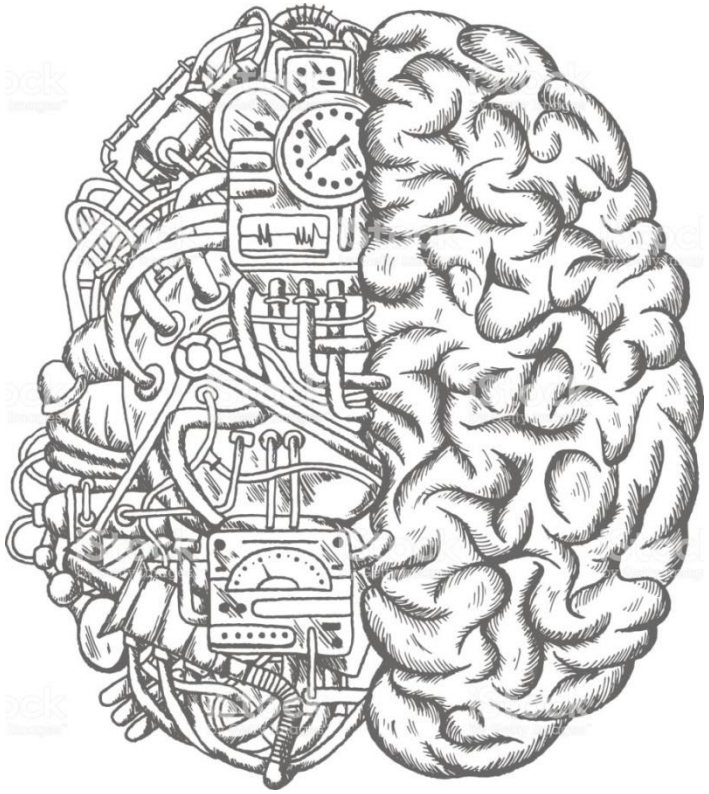
The DBS system is used to help control tremors and chronic movement disorders, like Parkinson's disease. Tiny electrodes are connected via a subcutaneous wire to a neurostimulator implanted under the skin near the clavicle.




1
Electrical stimulation delivered to targeted areas of the brain.
2
Select brain signals detected and recorded by the system.
3
Recorded data collected by physicians during clinical studies.
Medtronic

Neurodegenerative diseases

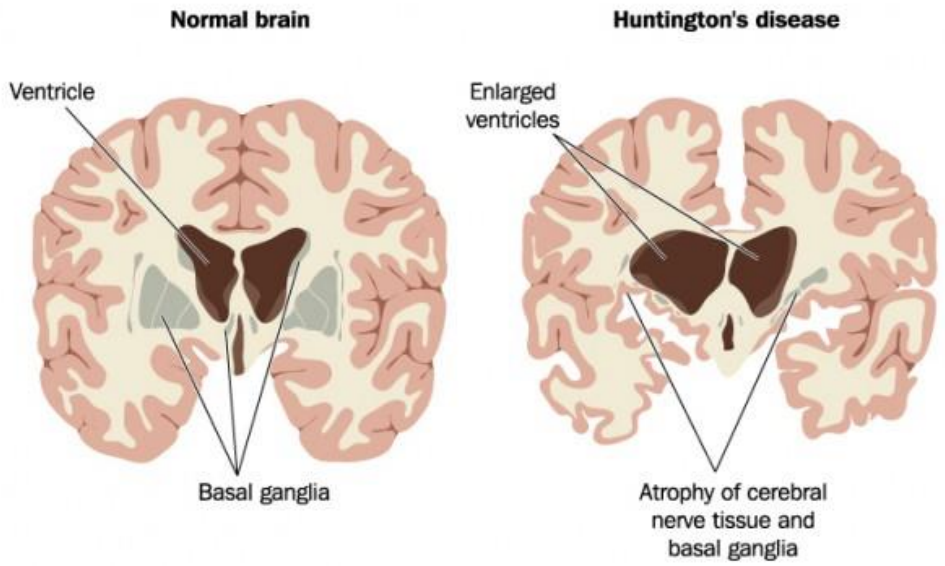
- Alzheimer's AD
- Parkinson's PD
- Huntington's HD
- Spinocerebellar ataxia SCA
- Prion disease
- Motor neurone diseases MND



Huntington’s disease HD

Huntington’s disease (HD) is a fatal genetic disorder that causes the progressive breakdown of nerve cells in the brain.

It deteriorates a person’s physical and mental abilities usually during their prime working years and has no cure.

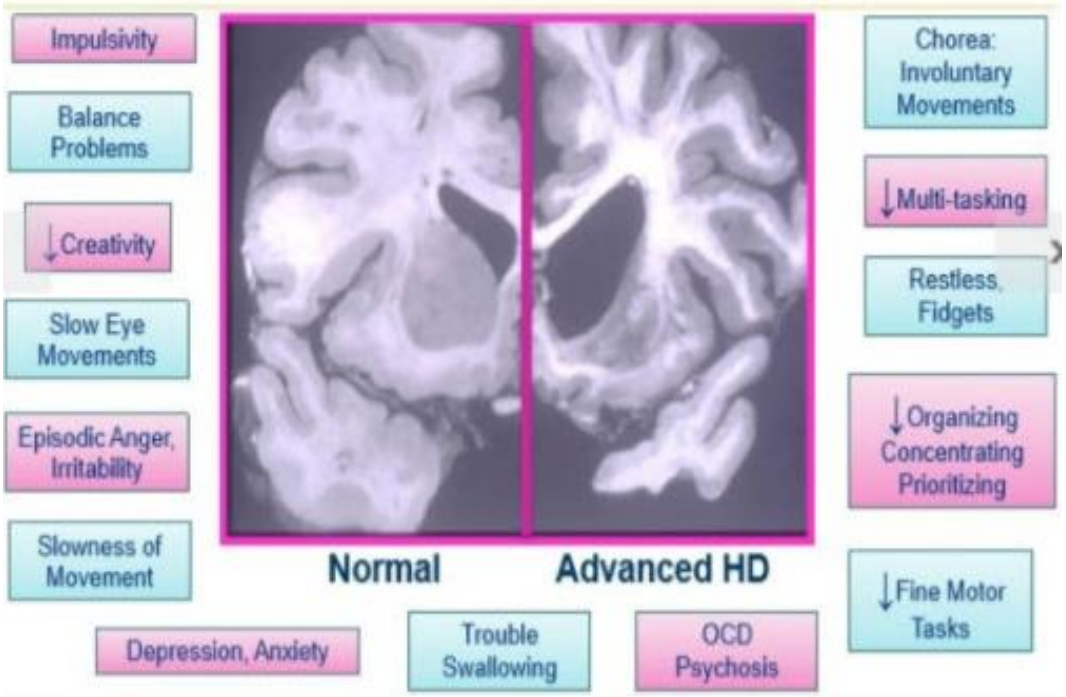


HD is known as the quintessential family disease because every child of a parent with HD has a 50/50 chance of inheriting the faulty gene.

Huntington's disease HD

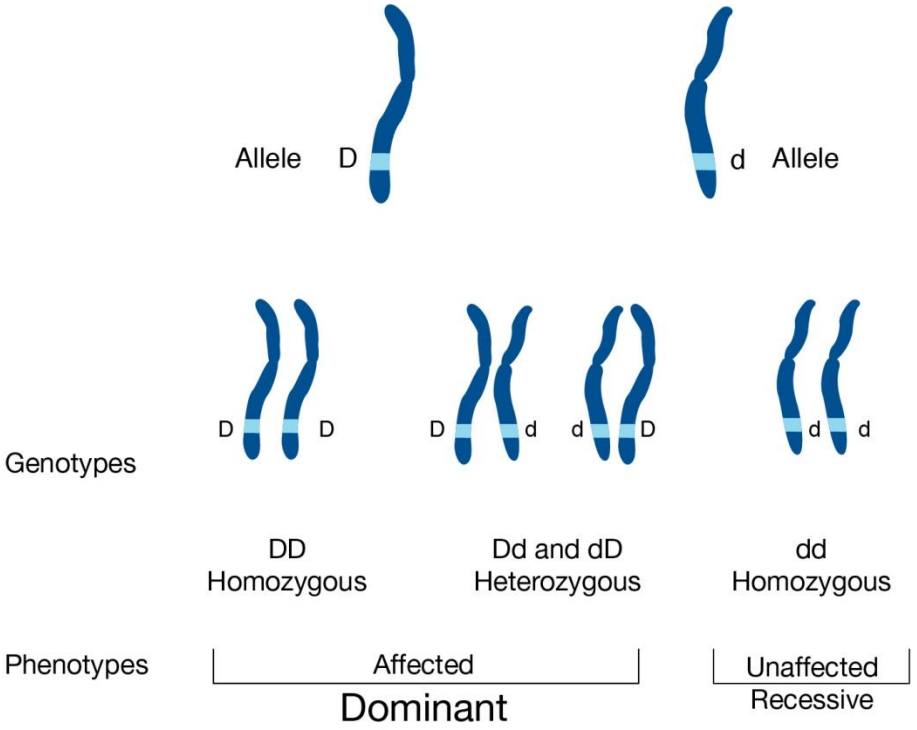
The symptoms of HD are described as having ALS, Parkinson's and Alzheimer's – simultaneously (between the ages of 30 to 50, and worsen over a 10 to 25-year period)

Patient's succumbs to pneumonia, heart failure or other complications.



Over time, HD affects the individual's ability to reason, walk and speak

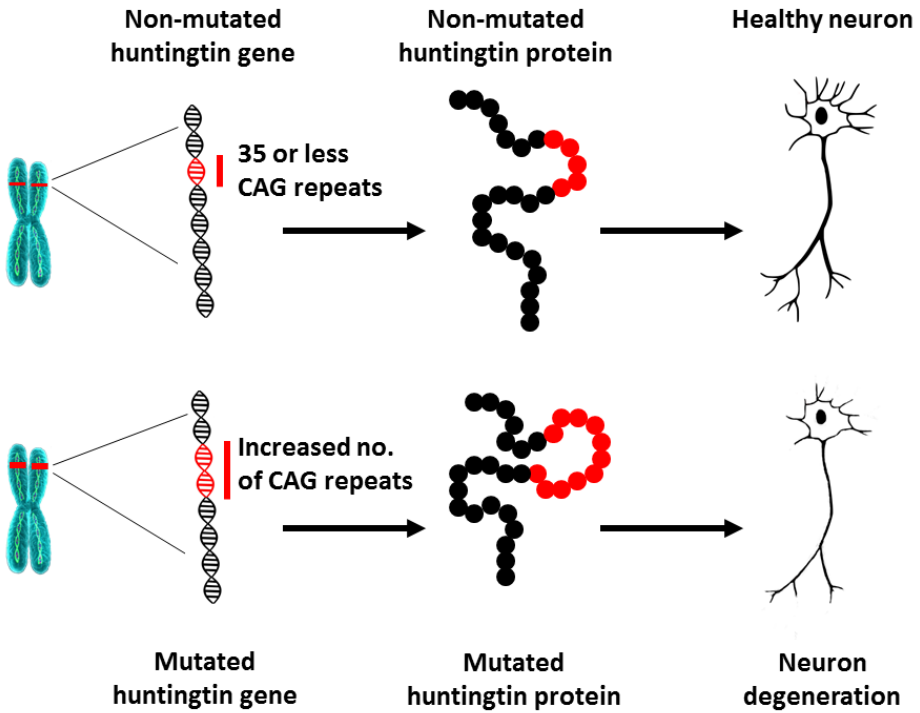
Huntington's disease HD hereditary



Everyone has the gene that causes HD, but only those that inherit the expansion of the gene will develop HD and perhaps pass it on to each of their children. Every person who inherits the expanded HD gene will eventually develop the disease.

Huntington's disease HD mechanism

Neurodegenerative disorder caused by an expansion of a repeating CAG triplet series in the huntingtin gene on chromosome 4, which results in a protein with an abnormally long polyglutamine sequence. The HD gene was identified in 1993.



The normal function of huntingtin is not known, but the expanded polyglutamine sequence in the huntingtin protein is toxic to brain cells. Atrophy is most marked in the corpus striatum of the basal ganglia, including the caudate and putamen.

In later phases of the disease, other regions of the brain are also affected.

Neurodegenerative diseases

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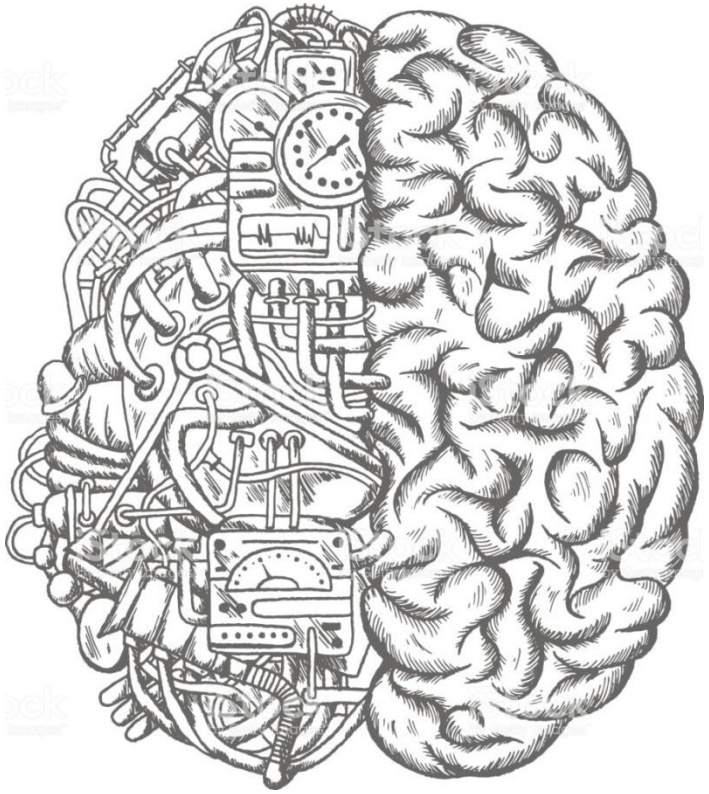
- Motor neurone diseases MND



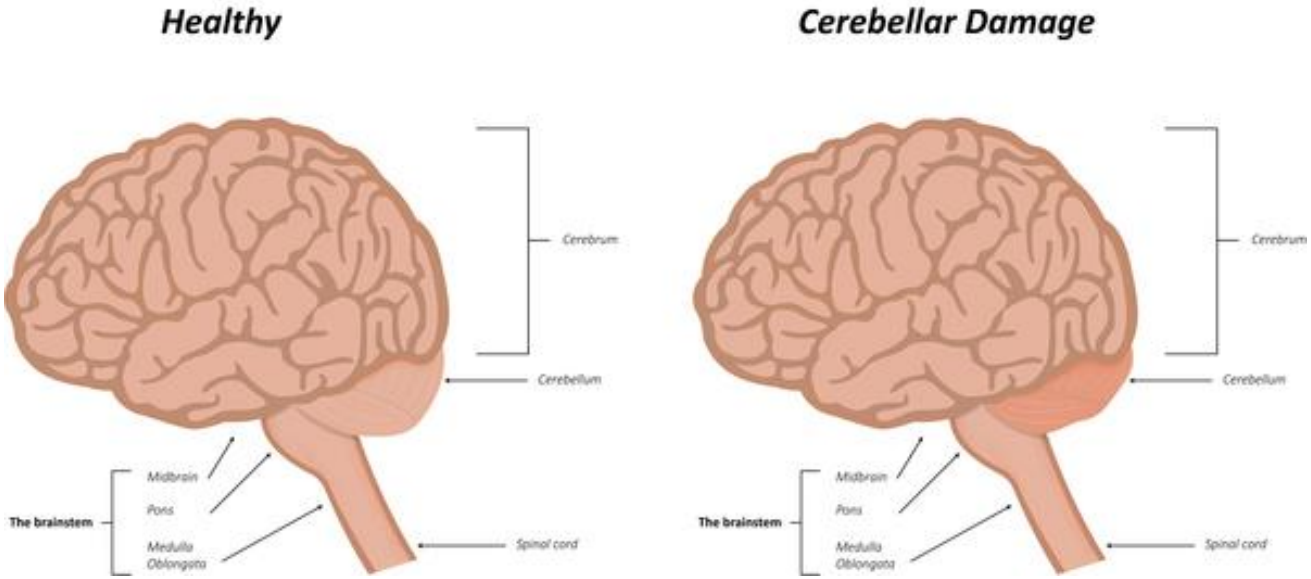
Spinocerebellar ataxia SCA

Group of hereditary ataxias that are characterized by degenerative changes in the part of the brain related to the movement control (cerebellum), and in the spinal cord.

There are many different types of SCA, and they are classified according to the mutated (altered) gene responsible for the specific type of SCA.

The types are described using "SCA" followed by a number, according to their order of identification: SCA1 through SCA40.

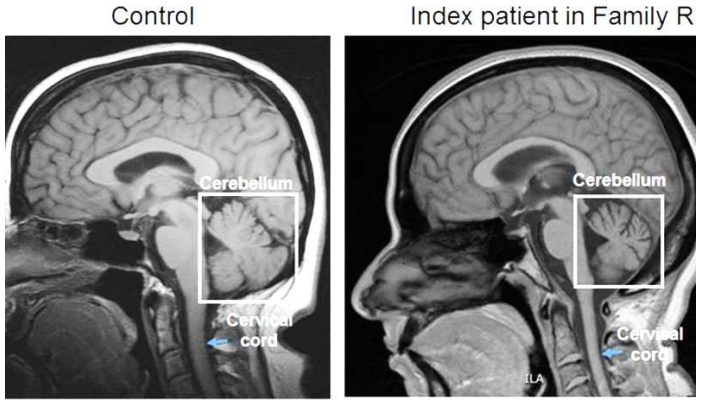
SCA is inherited in an autosomal dominant manner.



Spinocerebellar ataxia SCA

The signs and symptoms may vary by type but are similar, and may include an uncoordinated walk (gait), poor hand-eye coordination, and abnormal speech (dysarthria).

- Problems with coordination and balance (ataxia)
- Uncoordinated walk
- Poor hand-eye coordination
- Abnormal speech (dysarthria)
- Involuntary eye movement
- Vision problems
- Difficulty processing, learning, and remembering information



People affected by one of these types of SCA usually require a wheelchair by 10-15 years after the onset of symptoms.

Many will eventually need assistance to perform daily tasks

Neurodegenerative diseases

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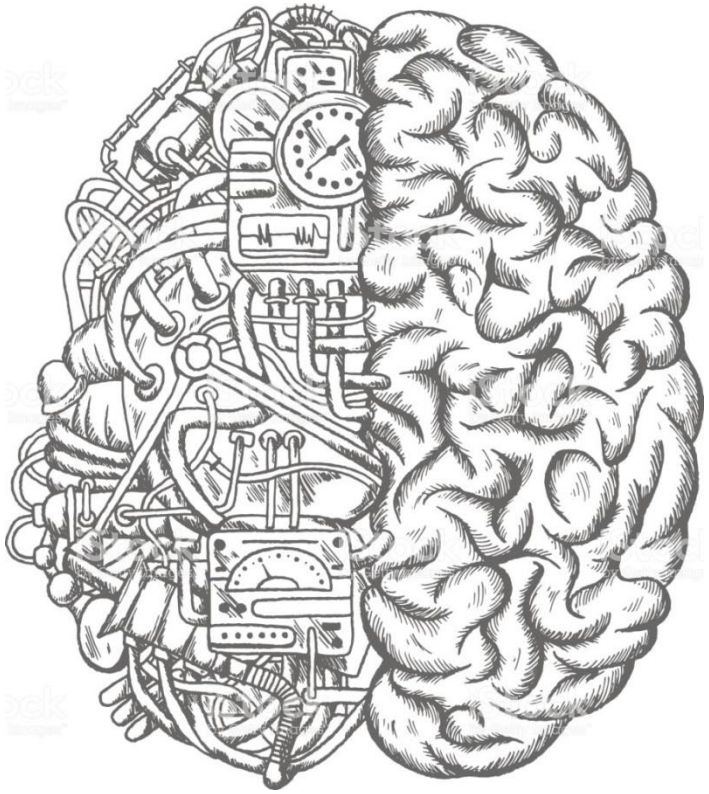
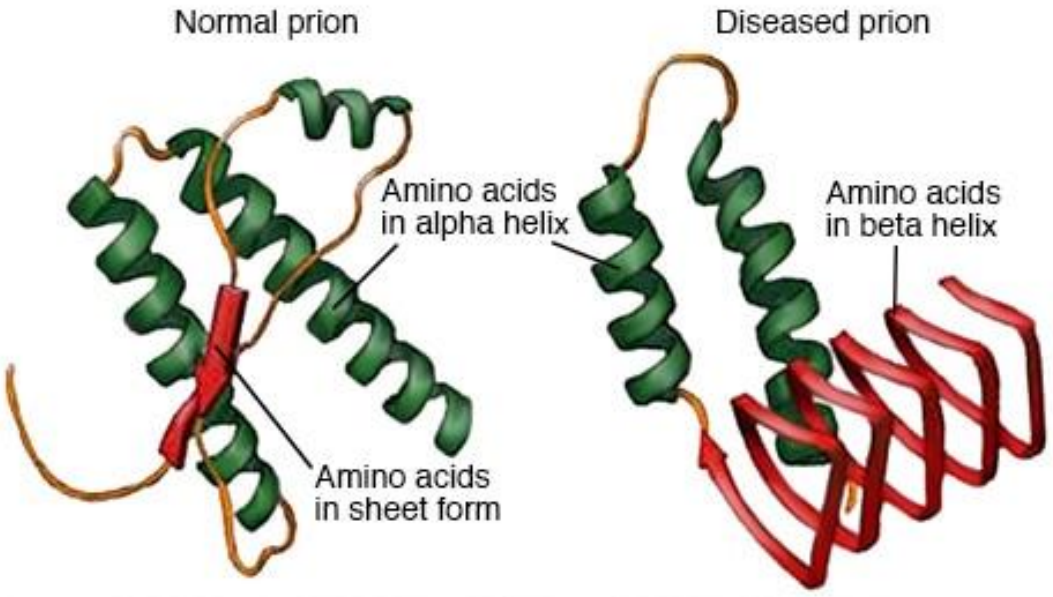


Prion disease

Prion diseases occur when normal prion protein, found on the surface of many cells, becomes abnormal and clump in the brain, causing brain damage.

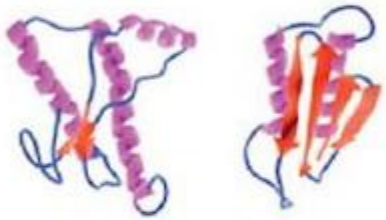
This abnormal accumulation of protein in the brain can cause memory impairment, personality changes, and difficulties with movement.



How Creutzfeldt-Jakob disease works

CAUSE

Creutzfeldt-Jakob disease is caused by abnormal proteins called prions that are not killed by standard methods for sterilizing surgical equipment.



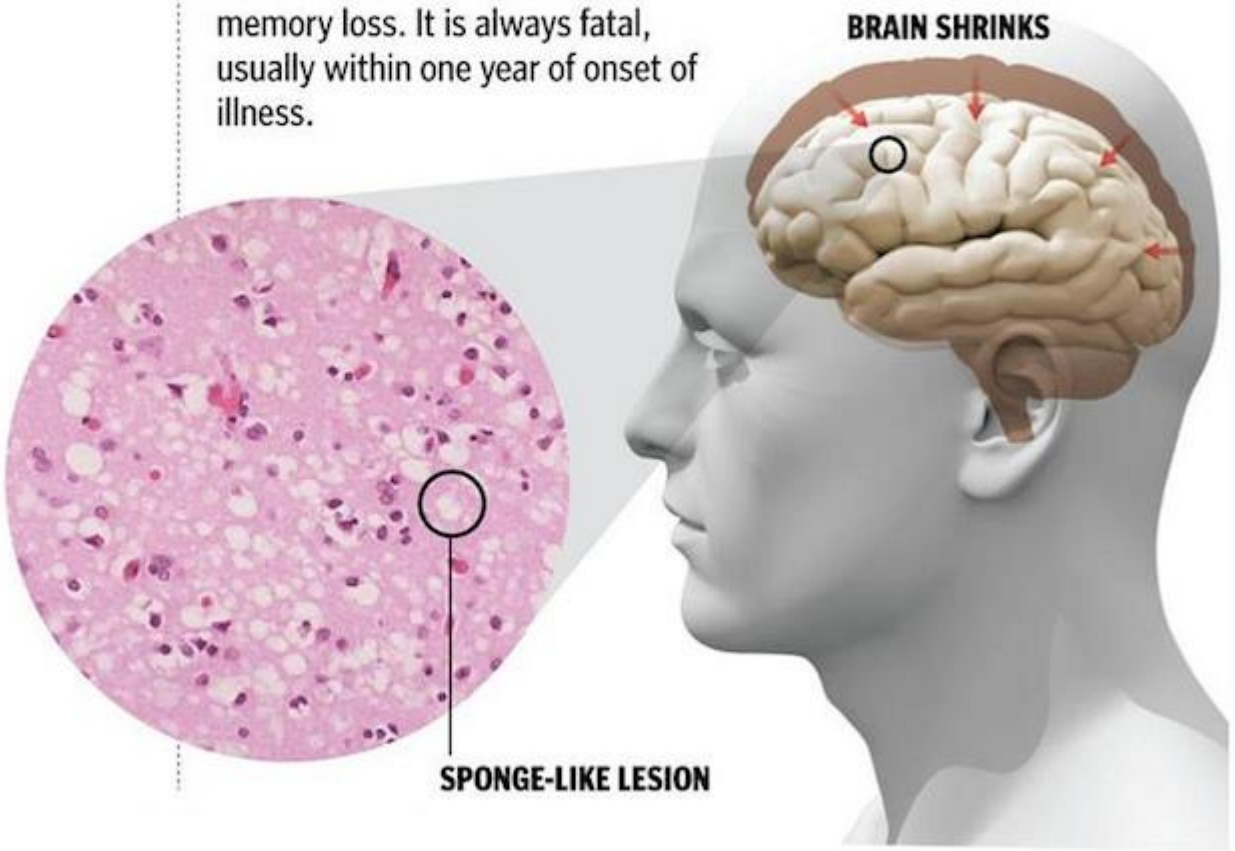
NORMAL HUMAN PROTEIN

DISEASE-CAUSING PRION

As prions build up in cells, the brain slowly shrinks and the tissue fills with holes until it resembles a sponge.

CONSEQUENCES

Those affected lose the ability to think and to move properly and suffer from memory loss. It is always fatal, usually within one year of onset of illness.



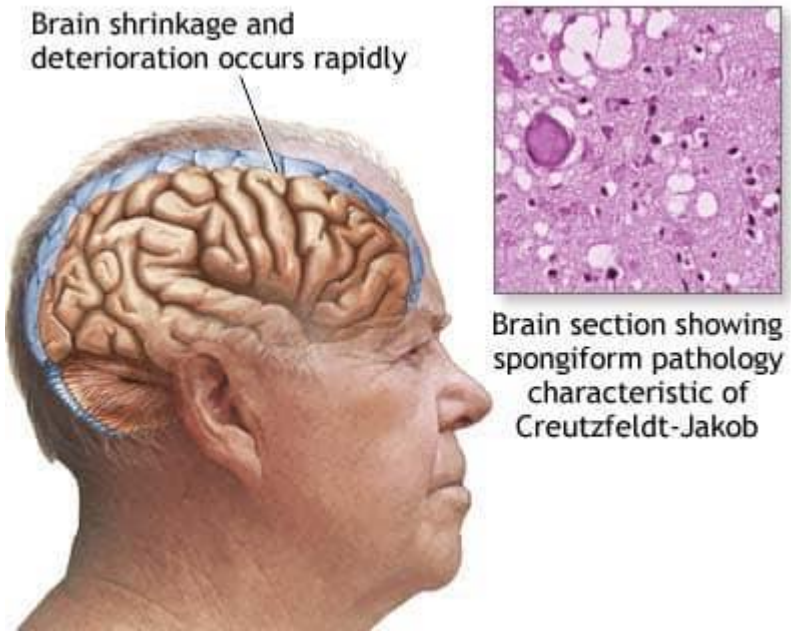
Prion diseases

- Sporadic CJD - on the other hand, develops suddenly without any known risk factors. Most cases of CJD are sporadic and tend to strike people around age 60. Acquired CJD is caused by exposure to infected tissue during a medical procedure, such as a cornea transplant. Symptoms of CJD quickly lead to severe disability and death. In most cases, death occurs within a year.
- Variant CJD - This is an infectious type of the disease that is related to “mad cow disease.” Eating diseased meat may cause the disease in humans. The meat may cause normal human prion protein to develop abnormally. This type of the disease usually affects younger people.
- Variably protease-sensitive prionopathy (VPSPr) - extremely rare. It is more likely to strike people around age 70 who have a family history of dementia.
- Gerstmann-Sträussler-Scheinker disease (GSS) - occurs at an earlier age, typically around age 40.
- Kuru - caused by eating human brain tissue contaminated with infectious prions. (seen in New Guinea)
- Fatal insomnia (FI) - rare hereditary disorder causing difficulty sleeping. There is also a sporadic form of the disease that is not inherited.
- Scrapie - infects sheeps and goats

Prion disease

Symptoms of prion diseases include:

- Rapidly developing dementia
- Difficulty walking and changes in gait
- Hallucinations
- Muscle stiffness
- Confusion
- Fatigue
- Difficulty speaking



Neurodegenerative diseases

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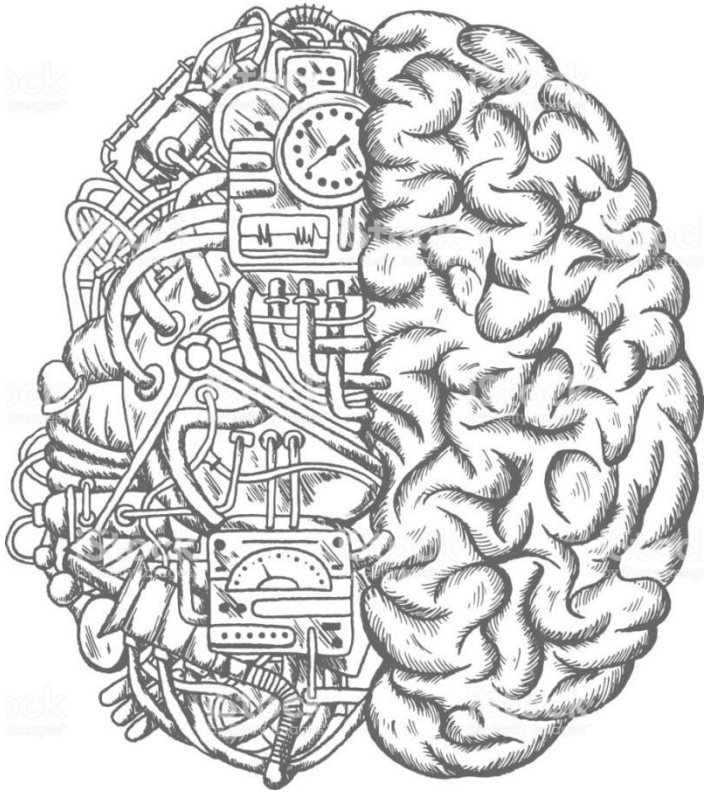
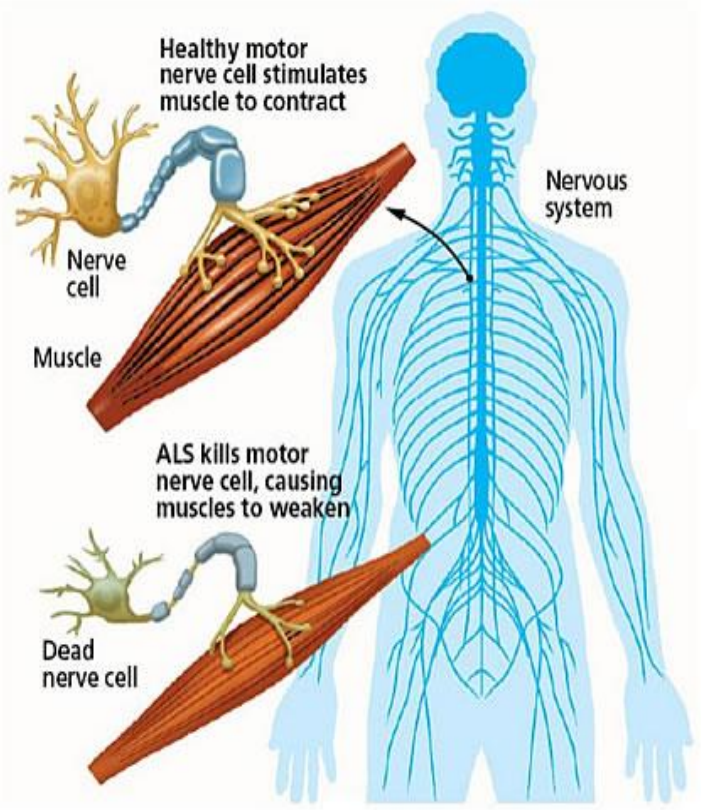


Motor neuron disease (MND) Amyotrophic lateral sclerosis (ALS)

The motor neuron diseases (MNDs) are a group of progressive neurological disorders that destroy motor neurons, the cells that control skeletal muscle activity such as walking, breathing, speaking, and swallowing;

- amyotrophic lateral sclerosis,
- progressive bulbar palsy,
- primary lateral sclerosis,
- progressive muscular atrophy,
- spinal muscular atrophy,
- Kennedy's disease,
- post-polio syndrome.



Amyotrophic lateral sclerosis (ALS)

Motor neurone disease, also known as amyotrophic lateral sclerosis (ALS), occurs when specialist nerve cells in the brain and spinal cord called motor neurones stop working properly.

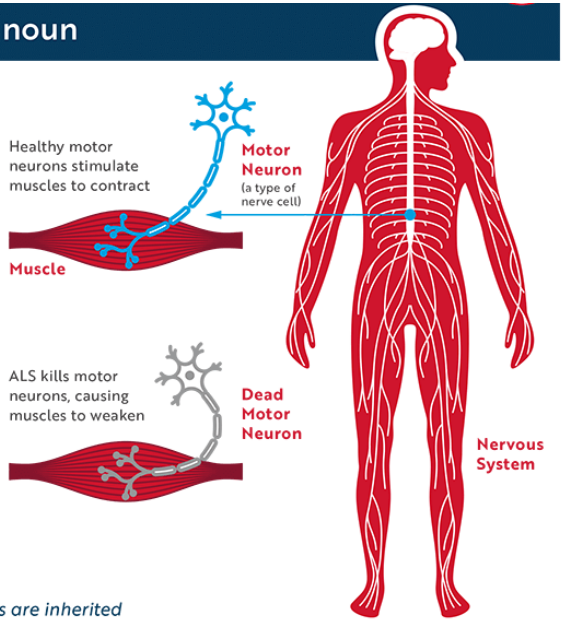
This leads to muscle weakness, often with visible wasting.

a·myo·tro·phic lateral sclerosis | noun

ALS IS A PROGRESSIVE NEURODEGENERATIVE DISEASE THAT AFFECTS NERVE CELLS IN THE BRAIN AND SPINAL CORD.

ALS usually strikes people between the ages of 40 and 70, and approximately 20,000 people in the U.S. have the disease at any given time.

Although there is not yet a cure or treatment that halts ALS, scientists we fund through our global research program have made significant progress in understanding what causes ALS. But their work is not done. Together, we work toward a cure.



FACTS ABOUT ALS

5,000+ *people are diagnosed per year*

10% *10% of cases are inherited through a mutated gene*

2-5 YEARS *is the average life expectancy*

90% *of cases occur without family history*

Every **90 MINUTES** *someone is diagnosed and someone passes away*

$250,000 *is the estimated out-of-pocket cost for caring for a person with ALS*

Only **3 DRUGS** *are currently approved by the U.S. FDA to treat ALS (Riluzole, Nuedexta, and Radicava)*

$2 BILLION *is the estimated cost to develop a drug to slow or stop the progression of ALS*

There is **NO CURE** *for ALS*

SYMPTOMS

Progressive loss of muscle control

ALS gradually prohibits the ability to:

- Speak
- Swallow
- Walk
- Grasp objects
- Move
- Breathe

DIAGNOSIS

Difficult to diagnose

- ALS is often diagnosed by ruling out other diseases, which may take months or years

MILITARY

Veterans are 2x as likely to get ALS

- ALS affects veterans who

Amyotrophic lateral sclerosis (ALS)

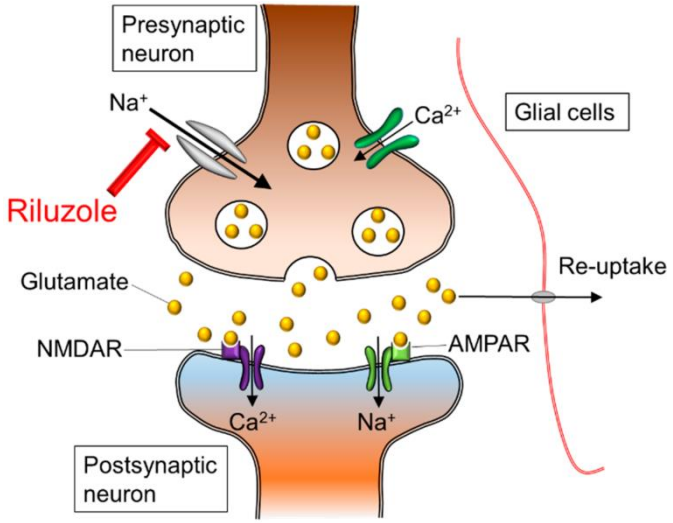
Early symptoms can include:

- weakness in ankle or leg – trip, or find it harder to climb stairs
- slurred speech, which may develop into difficulty swallowing some foods
- a weak grip –drop things, or find it hard to open jars or do up buttons
- muscle cramps and twitches
- weight loss –arms or leg muscles may have become thinner over time
- difficulty stopping oneself crying or laughing in inappropriate situations

Amyotrophic lateral sclerosis (ALS)

Treatments:

- highly specialised clinics, typically involving a specialist nurse and occupational therapy to help make everyday tasks easier
- physiotherapy and exercises to maintain strength and reduce stiffness
- advice from a speech and language therapist
- advice from a dietitian about diet and eating
- a medicine called riluzole that can slightly slow down the progression medicines to relieve muscle stiffness and help with saliva problems
- emotional support

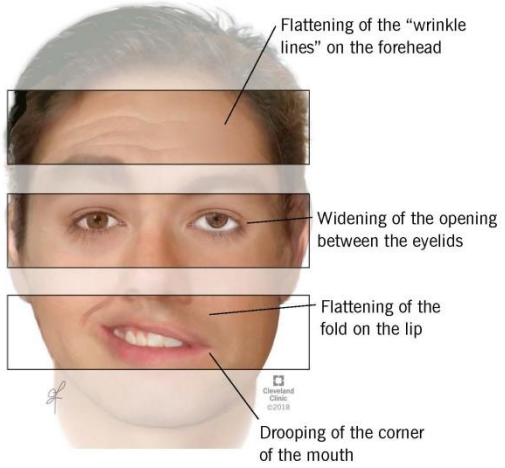


Progressive bulbar palsy

The brain stem is the part of the brain needed for swallowing, speaking, chewing, and other functions.

Signs and symptoms of progressive bulbar palsy include difficulty swallowing, weak jaw and facial muscles, progressive loss of speech, and weakening of the tongue.

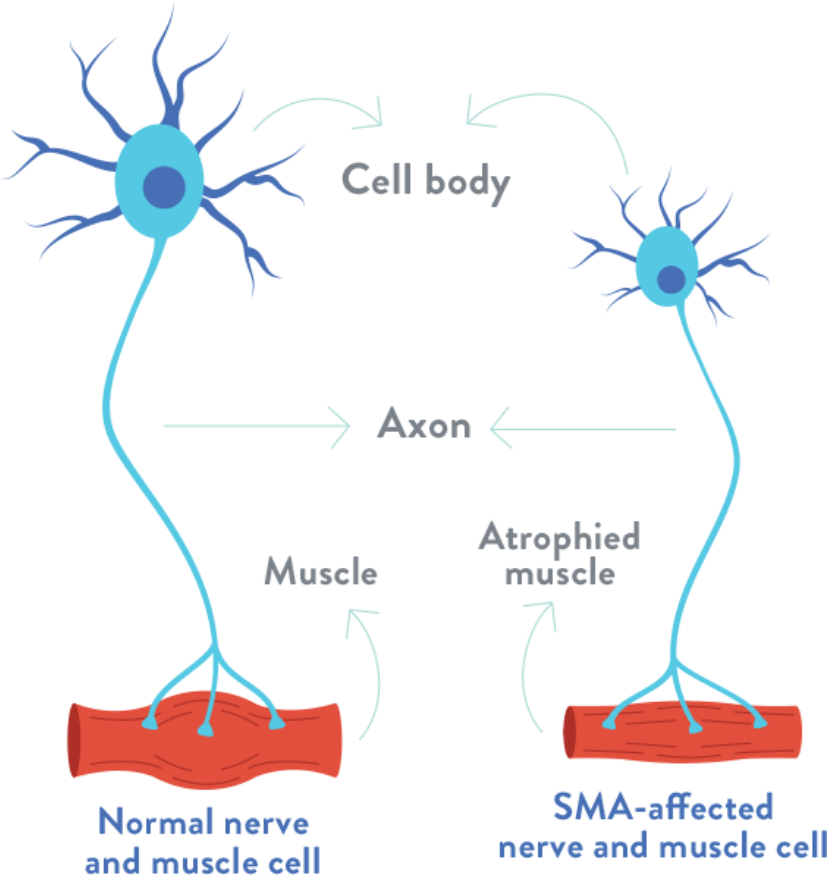
Additional symptoms include less prominent weakness in the arms and legs, and outbursts of laughing or crying (called emotional lability).



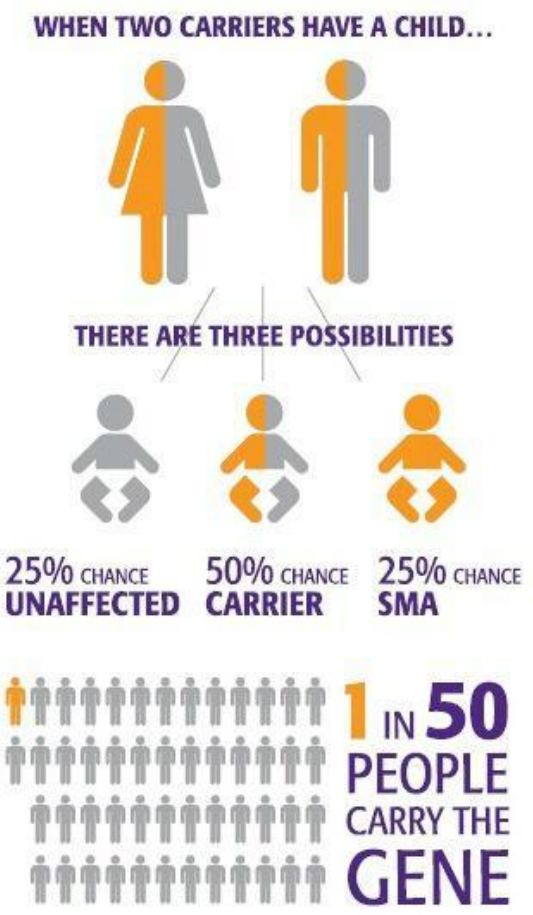
Progressive bulbar palsy is considered a variant form of amyotrophic lateral sclerosis (ALS). Many people with progressive bulbar palsy later develop ALS.

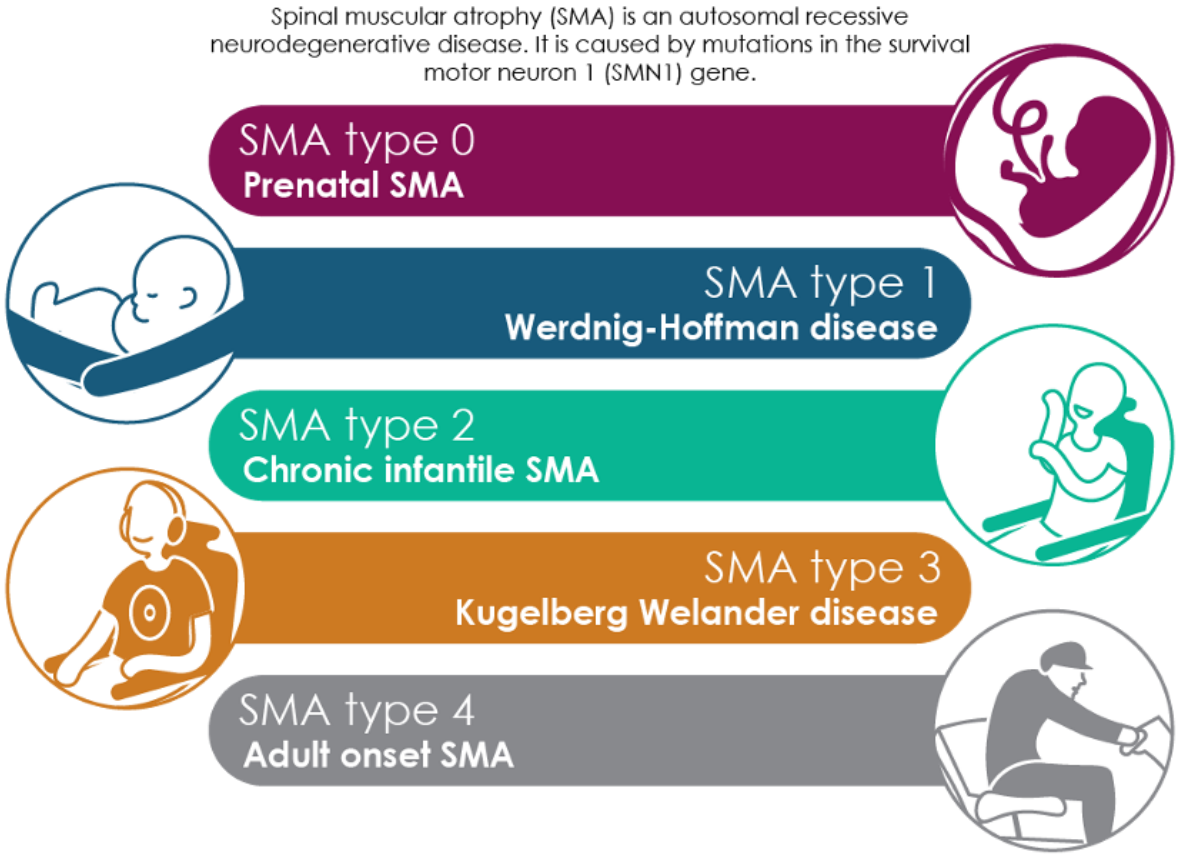
Spinal muscular atrophy SMA

Spinal muscular atrophy is a genetic disorder characterized by weakness and wasting (atrophy) in muscles used for movement (skeletal muscles). It is caused by a loss of specialized nerve cells, called motor neurons that control muscle movement.



Spinal muscular atrophy SMA





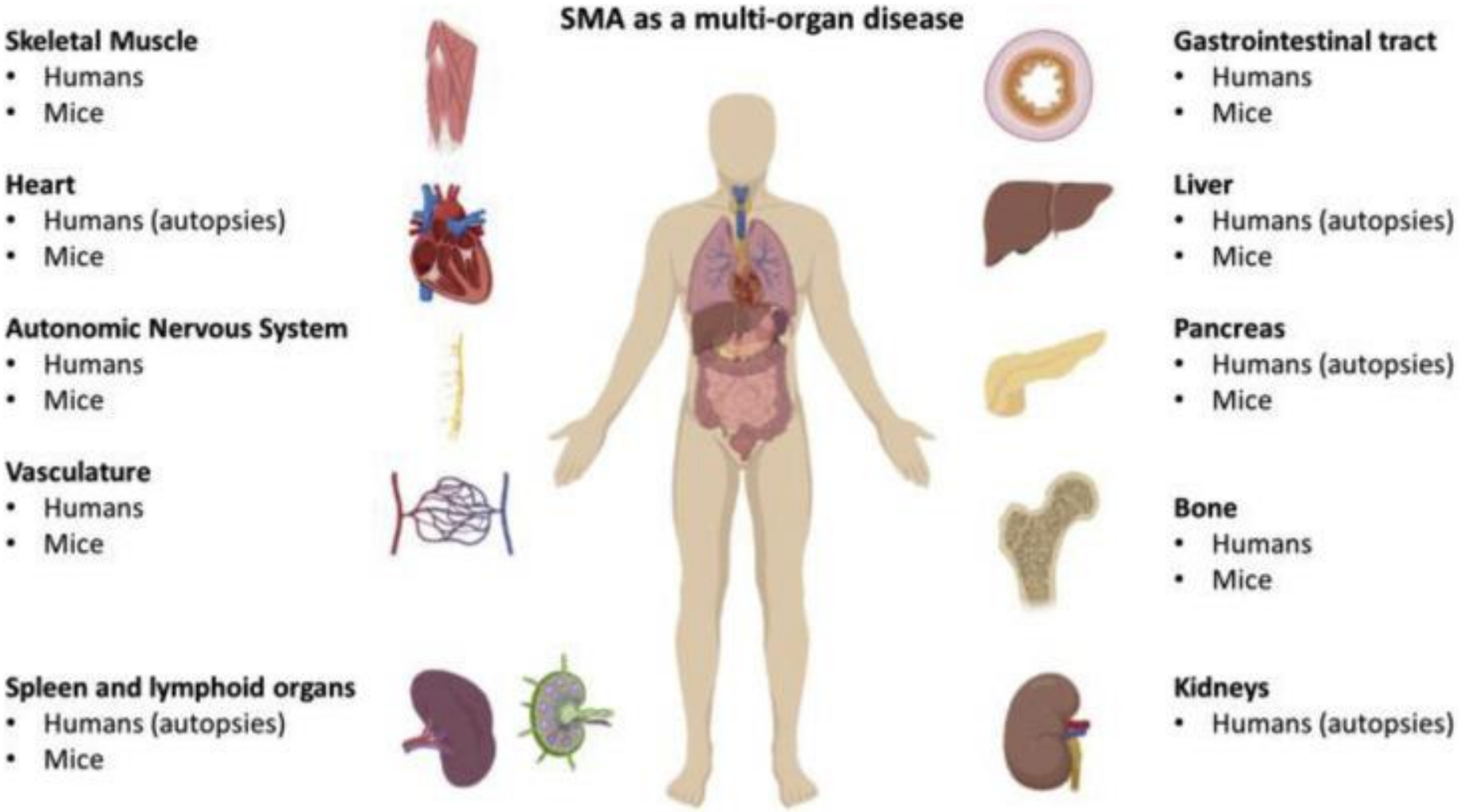
2 cases on 100 000

Spinal muscular atrophy SMA

There are many types of spinal muscular atrophy that are caused by changes in the same genes:

- type 0 is evident before birth and is the rarest and most severe form of the condition. Affected infants move less in the womb, and as a result they are often born with joint deformities (contractures).
- type I (also called Werdnig-Hoffmann disease) is the most common form of the condition. It is a severe form of the disorder with muscle weakness evident at birth or within the first few months of life.
- type II (also called Dubowitz disease) is characterized by muscle weakness that develops in children between ages 6 and 12 months. Children with this type can sit without support, although they may need help getting to a seated position.
- type III (also called Kugelberg-Welander disease) typically causes muscle weakness after early childhood. Many affected individuals require wheelchair assistance later in life. People with spinal muscular atrophy type III typically have a normal life expectancy.
- Spinal muscular atrophy type IV is rare and often begins in early adulthood.

Spinal muscular atrophy SMA



Primary lateral sclerosis (PLS)

- PLS affects the upper motor neurons (also called corticospinal neurons) in the arms, legs, and face.
- It occurs when nerve cells in the motor regions of the cerebral cortex.
- The disorder often affects the legs first, followed by the body, trunk, arms and hands, and, finally the bulbar muscles (muscles that control speech, swallowing, and chewing)
- PLS is not fatal.

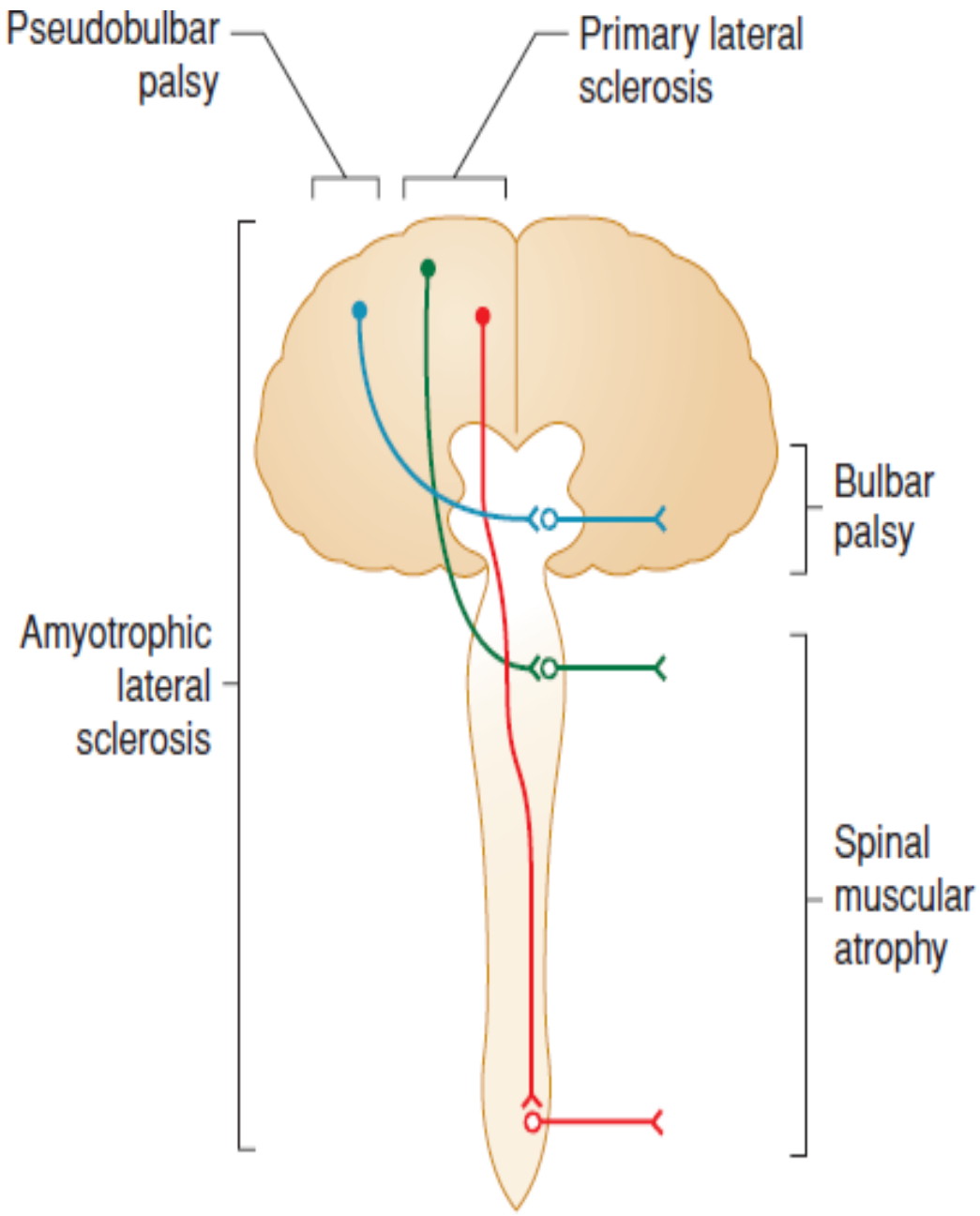
Primary lateral sclerosis (PLS)

Symptoms include weakness, muscle stiffness and spasticity, clumsiness, slowing of movement, and problems with balance and speech. PLS is more common in men than in women

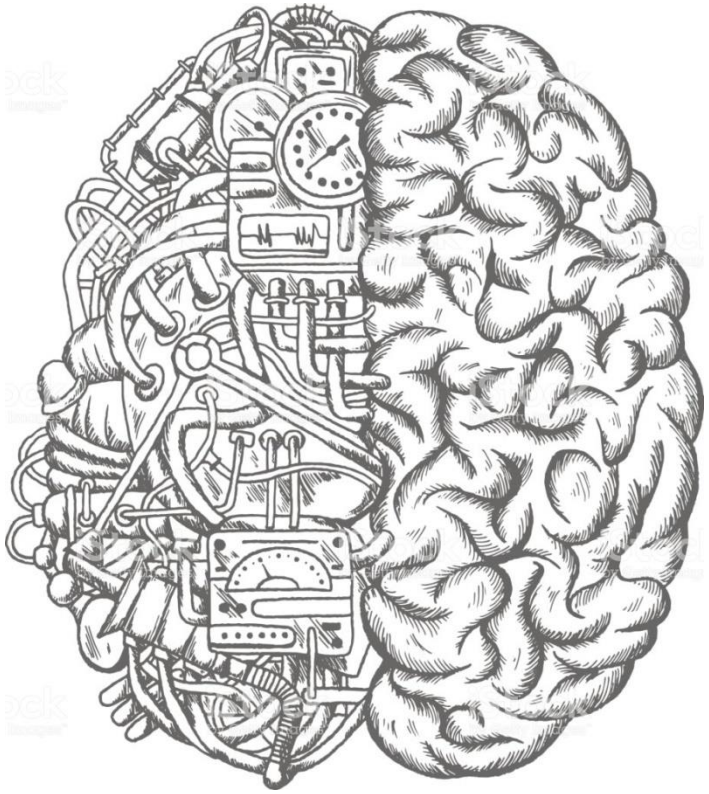
Treatment for individuals with PLS is symptomatic. Muscle relaxants such as baclofen, tizanidine, and the benzodiazepines may reduce spasticity.

Other drugs may relieve pain and antidepressants can help treat depression.

Physical therapy, occupational therapy, and rehabilitation may prevent joint immobility and slow muscle weakness and atrophy.


Pseudobulbar palsy
Primary lateral sclerosis
Bulbar palsy
Amyotrophic lateral sclerosis
Spinal muscular atrophy

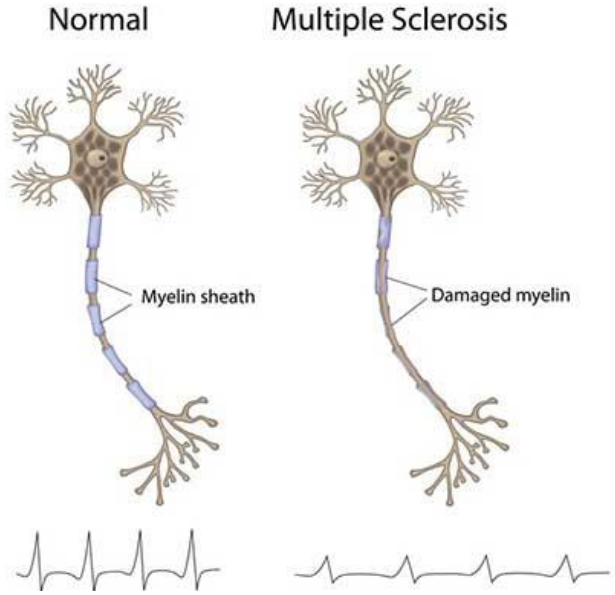
- Alzheimer’s AD
- Parkinson’s PD
- Huntington’s HD
- Spinocerebellar ataxia SCA
- Prion disease
- Motor neurone diseases MND



and multiple sclerosis

Multiple sclerosis MS

An unpredictable disease of the central nervous system, multiple sclerosis (MS) can range from relatively benign to somewhat disabling to devastating, as communication between the brain and other parts of the body is disrupted.



Autoimmune disease -- one in which the body, through its immune system, launches a defensive attack against its own tissues.

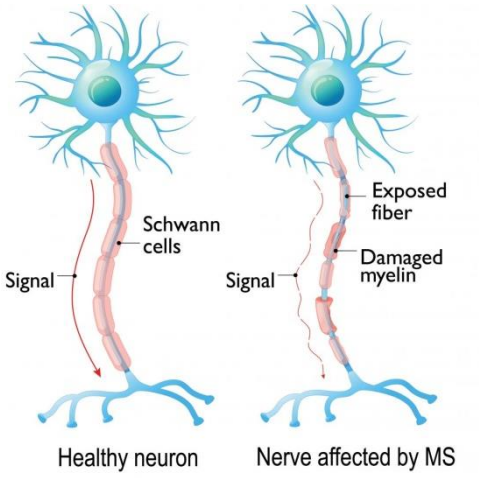
Nerve-insulating myelin that comes under assault.

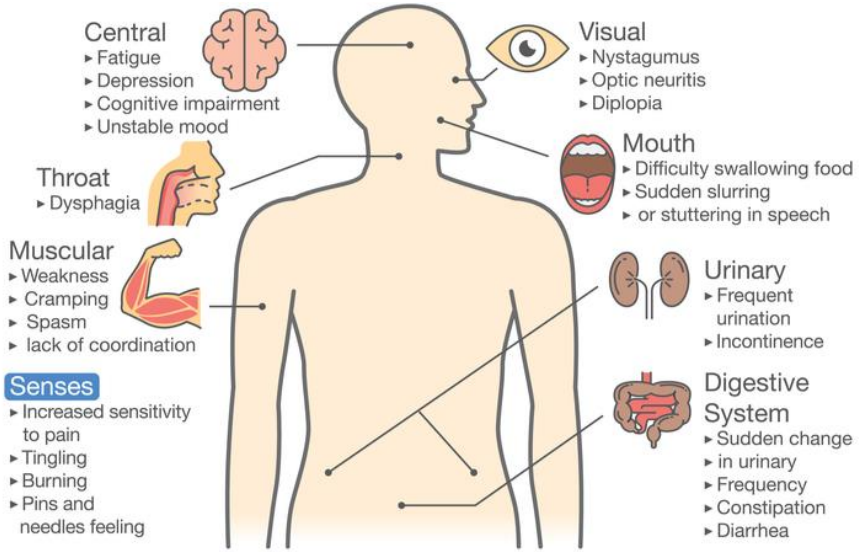
Multiple sclerosis MS

First symptoms of MS between the ages of 20 and 40

Initial symptom of MS is often blurred or double vision, red-green color distortion, or even blindness in one eye

Most MS patients experience muscle weakness in their extremities and difficulty with coordination and balance.

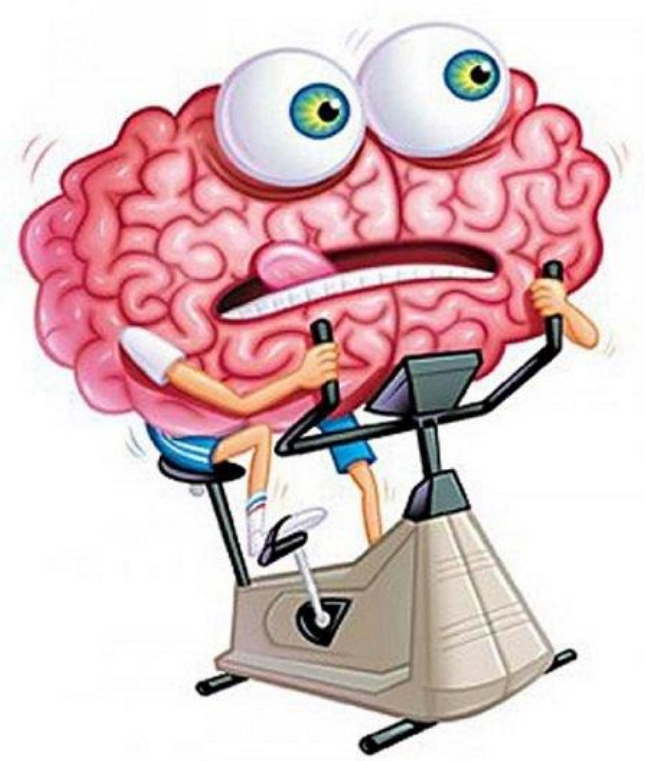




MS is a disease with a natural tendency to remit spontaneously, for which there is no universally effective treatment (steroids, retrovirus therapy).

It's about 2 to 3 times more common in women than men.

Remember to exercise your brain



Thank you