Types of Dementia

There are well over 100 different types of dementias. This chart lists some of the most well-known.

Type of Dementia	Brain Changes	Average Age of Onset	Typical Symptoms	Average Progression
Neurodegenerative Diseases				
Alzheimers Disease (AD) Most common type of dementia https://www.nia.nih.gov/health/alzheimers-disease-fact-sheet	Deterioration of brain cells, associated with amyloid plaques and tau tangles Left brain hippocampus first affected, then spreads throughout brain	After age 65, most commonly around age 75 Can have a genetic component	Issues with recent memory, word-finding, complicated unfamiliar tasks, interpreting meaning behind words May develop insomnia, depression, motor issues, restlessness, agitation, or delusions Old memories, familiar patterns and tasks, and motor skills are usually not affected initially	8-10 years of life after symptom onset Usually a steady progression with some plateaus
Young Onset Alzheimers Disease https://www.mayoclinic.org/diseases-conditions/alzheimers-disease/in-depth/alzheimers/art-20048356	Deterioration of brain cells, associated with amyloid plaques and tau tangles Left brain hippocampus first affected, then spreads throughout brain	Between 29-60 years of age Can have a genetic component	Same as above, with more motor skill issues often noted	10 years of life after symptom onset, usually a rapid progression after diagnosis but may plateau towards the end of the disease
Early Onset Familial Alzheimers Disease (eFAD) https://www.alzforum.org/early-onset-familial-ad/overview/what-early-onset-familial-alzheimer-disease-efad	Deterioration of brain cells, associated with amyloid plaques and tau tangles Left brain hippocampus first affected, then spreads throughout brain	Usually under the age of 65, often as young as 30s or 40s Caused by a familial gene mutation	Same as above	Same as above
Frontal Variant Alzheimers Disease (fvAD) https://minerva-access.unimelb.edu.au/bitstream/handle/11343/32987/294699_Does%20executive%20impairment_Woodward.pdf?sequence=1&isAllowed=y	Deterioration of brain cells, associated with amyloid plaques and tau tangles Frontal lobes are affected much earlier in the disease than other forms of Alzheimers Disease	Usually over age 65, often mid 70s	Typical Alzheimer Disease symptoms, but demonstrate more severe early decline in cognitive, functional, neuropsychiatric, and global abilities	8-10 years of life after symptom onset, typical quicker initial cognitive decline than standard form of Alzheimers Disease

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Down Syndrome-associated Alzheimers Disease https://www.alz.org/alzheimers-dementia/what-is-dementia/types-of-dementia/down-syndrome	Individuals with Down syndrome carry an extra copy of chromosome 21, which carries the gene that produces a protein called amyloid precursor protein (APP). This often leads to amyloid plaque development in the brain by age 40	At conception Caused by an extra chromosome during genetic development	Down Syndrome causes cognitive delays, health issues, and physical abnormalities, but individuals are also at a greatly increased risk of developing dementia	Those with Down Syndrome currently live to an average of 60 years of age
Progressive Supranuclear Palsy (PSP) https://www.nia.nih.gov/health/types-frontotemporal-disorders.	Deterioration of brain cells, associated with amyloid plaques and tau protein deposits	Around age 60 Rarely a genetic component	Slow or uncoordinated movements are often the first symptoms Issues with eye movements, loss of facial expressions, stiffness of neck/trunk, and falls are common; issues with cognitive function, behavior, and memory may also develop	6-10 years of life after symptom onset
Limbic-Predominant Age-Related TDP-43 Encephalopathy (LATE) New form of dementia recognized in 2020 https://pubmed.ncbi.nlm.nih.gov/31039256	'Secondary dementia': caused by brain blood supply issues rather than a direct brain disease Associated with abnormal TDP-43 protein accumulation	85-100 years of age May account for 1 in 4 cases of dementia for those over 90 years of age	Symptoms, such as memory loss, may be similar to Alzheimers, but tends to progress slower	8-15 years of life after symptom onset, generally has a slower progression than other forms of dementia
Corticobasal Degeneration or Corticobasal Ganglionic Degeneration (CBD or CBGD) Sometimes considered to be a subtype of FTD https://memory.ucsf.edu/dementia/corticobasal-syndrome#:~:text=Corticobasal%20syndrome%20(CBS)%20is%20a,controlling%20their%20arm%20or%20leg.	Degeneration of the cortex of the brain, especially the fronto-parietal region, as well as parts of the basal ganglia Associated with tau protein accumulation or amyloid plaques	60 years of age Might possibly be a genetic component	Movement issues in one limb is often the first symptom, but typically quickly affects all limbs. Speech problems, cognitive function issues, and behavioral changes are also common Sometimes behavioral changes or speech problems occur before movement issues	6-8 years of life after symptom onset

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Neurodegenerative Diseases: Movement Disorders				
Dementia with Lewy Bodies (DLB) Third most common type of dementia https://www.medicalnewstoday.com/articles/314850.php#types	Deterioration of brain cells, associated with Lewy bodies, tiny spherical alpha-synuclein protein deposits that disrupt function of brain cells	After age 55 Rarely a genetic component	Decreased focus, hallucinations, delusional thinking, insomnia, nightmares, swallowing difficulties, intention tremors, declined motor skills, some memory loss May have toxic or opposite reactions to anti-anxiety, anti-depressant, and neuroleptic medications	7-9 years of life after symptom onset May be periods of near-normal cognition alternating with declined cognition
Dementia in Parkinson Disease (PDD) https://www.nia.nih.gov/health/parkinsons-disease#:~:text=Parkinson's%20disease%20is%20a%20brain,have%20difficulty%20walking%20and%20talking.	In Parkinson Disease, death of nerve cells in the brain result in a deficiency of the neurotransmitter dopamine, which causes motor and speech issues Dementia in Parkinson Disease: presence of Lewy bodies	Around age 60 Can have a genetic component	Motor symptoms nearly always precede cognitive impairment by several years A majority of those with Parkinson Disease do not develop dementia	10-20 years of life after Parkinson Disease symptom onset

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Neurodegenerative Diseases: Frontotemporal Dementias				
Frontotemporal Dementia (FTD; Pick's Disease) Fourth most common type of dementia https://www.mayoclinic.org/diseases-conditions/frontotemporal-dementia/symptoms-causes/syc-20354737#:~:text=Frontotemporal%20dementia%20is%20an%20umbrella,these%20lobes%20shrink%20(atrophy).	Deterioration of brain cells in the frontal and/or temporal parts of the brain Often associated with tau protein accumulation, but not amyloid plaques Several subtypes of FTD:	Varies based on subtype	Varies based on subtype	Varies based on subtype
Behavioral Variant FTD (bvFTD) Most common type of FTD https://www.nia.nih.gov/health/types-frontotemporal-disorders	Deterioration of cells in the frontal and/or temporal parts of the brain Often associated with tau protein accumulation, but not amyloid plaques	Early 50s Can have a genetic component	Issues with disinhibition, repetitive behaviors, lack of impulse control, egocentric behavior, depression, overeating, impaired judgement, and cognitive and executive functioning Later, issues with movement and language typically also occur, but memory is preserved for many	8-10 years of life after symptom onset, may be slightly shorter life expectancy than other forms of Frontotemporal Dementia
Frontotemporal Dementia with Parkinsonism linked to Chromosome 17 (FTDP-17) https://medlineplus.gov/genetics/condition/frontotemporal-dementia-with-parkinsonism-17/#:~:text=Frontotemporal%20dementia%20with%20parkinsonism%2D17%20(FTDP%2D17)%20is,the%20frontal%20and%20temporal%20lobes.	Deterioration of brain cells in the medial temporal lobe, as well as the frontal cortex and other brain areas Identified with autopsy posthumously Associated with tau protein accumulation	40-60 years of age Caused by a familial gene mutation	Frontotemporal symptoms associated with Parkinsonian features related to movement disorders such as rigidity, reduced speed, and uncontrolled movements and eye movements Early symptoms often include behavioral and personality changes	5-10 years of life after symptom onset
Dementia Lacking Distinctive Histology (DLDH; a type of FTD) https://n.neurology.org/content/40/2/251	Deterioration of brain cells in the left side of frontal and/or temporal parts of the brain Often associated with tau protein accumulation, but not usually amyloid plaques	50-60 years of age May have a genetic component	Issues with personality changes, speech, and swallowing Memory loss and other symptoms typically also develop	2-7 years of life after symptom onset

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Neurodegenerative Diseases: Primary Progressive Aphasia				
Primary Progressive Aphasia (PPA) Subtype of FTD with two main types: Semantic Variant PPA and Nonfluent Variant PPA https://www.mayoclinic.org/diseases-conditions/primary-progressive-aphasia/symptoms-causes/syc-20350499	Deterioration of brain cells in the left side of frontal and/or temporal parts of the brain Often associated with tau protein accumulation, but not amyloid plaques	Most often between age 50 and 75 Can have a genetic component	Slowly lose the ability to speak, write, read, and comprehend language Over time, issues with memory and executive functioning often occur, Behavioral changes may also occur	3-12 years of life after symptom onset Patients often experience language issues for an average of four years before going to the doctor
Semantic Variant Primary Progressive Aphasia (SV-PPA) https://www.theaftd.org/what-is-ftd/primary-progressive-aphasia/semantic-variant-ppa-svppa/	Deterioration of brain cells in the left side of frontal and/or temporal lobes Often associated with tau protein accumulation, but not amyloid plaques	Most often between age 50 and 75 Can have a genetic component	In this subtype of PPA, the ability to comprehend words is affected Speech is typically not initially affected	3-12 years of life after symptom onset
Progressive Nonfluent/ Agrammatic Aphasia (NFV-PPA) https://www.theaftd.org/what-is-ftd/primary-progressive-aphasia/nonfluent-agrammatic-ppa-nfvppa/	Deterioration of brain cells in the left side of frontal and/or temporal lobes Often associated with tau protein accumulation, but not usually amyloid plaques	Most often between age 50 and 75 Can have a genetic component	In this subtype of PPA, the ability to form speech is affected Comprehension is typically not initially affected	3-12 years of life after symptom onset
Logopenic Aphasia (also called **Progressive Fluent Aphasia**) https://rarediseases.info.nih.gov/diseases/10791/logopenic-progressive-aphasia#:~:text=Logopenic%20progressive%20aphasia%20(LPA)%20is,primary%20progressive%20aphasia%20(PPA).	Deterioration of brain cells, associated with a decrease in tau proteins rather than an accumulation	50-75 years of age Can have a genetic component	In this subtype of PPA, the ability to find the right words while speaking is affected Comprehension is typically not initially affected	3-12 years of life after symptom onset

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Diseases Associated with Ataxia				
Huntington's Disease Also referred to as Huntington's Chorea https://www.alz.org/alzheimers-dementia/what-is-dementia/types-of-dementia/huntington-s-disease#:~:text=Huntington's%20disease%20(HD)%20is%20a,movement%2C%20mood%20and%20thinking%20skills.	Associated with an abnormal accumulation of the protein huntingtin, causing degeneration of the central area of the brain	30-50 years of age Caused by a genetic mutation	Uncontrolled muscle movement is typically the first symptom Behavioral and personality changes, cognitive function issues, irritability, depression, and obsessive-compulsive behaviors are also common	5-8 years of life after symptom onset
Friedreich's Ataxia (FRDA or FA) https://www.ninds.nih.gov/Disorders/Patient-Caregiver-Education/Fact-Sheets/Friedreichs-Ataxia-Fact-Sheet	Caused by degenerative changes in the sensory nerve fibers of the spinal cord, resulting in reduced signals to the cerebellum of the brain	10-15 years of age Caused by an inherited recessive gene mutation	Lack of coordination or control of movement (ataxia) during walking is typically the first symptom Slurred speech, cardiac disease, scoliosis of the spine, foot deformities, and diabetes often develop Cognitive function is not usually affected	Varies based on severity Patients are usually wheelchair-dependent within 10-20 years from symptom onset
Autosomal Dominant Cerebellar Ataxia (ADCA) https://rarediseases.org/rare-diseases/autosomal-dominant-hereditary-ataxia/	Degeneration of the brain cerebellum Includes many different subtypes	Varies based on subtype, but most common onset is in early adulthood Caused by an inherited dominant gene mutation	Lack of coordination or control of movement (ataxia) during walking is typically the first symptom Slurred speech, cardiac disease, scoliosis of the spine, foot deformities, vision disorders, sensory loss, neuropathy, and diabetes may develop	Varies based on subtype

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Cerebrovascular Disease Dementias				
Vascular Dementia Second most common type of dementia https://www.mayoclinic.org/diseases-conditions/vascular-dementia/symptoms-causes/syc-20378793#:~:text=Vascular%20dementia%20is%20a%20general,t%20always%20cause%20vascular%20dementia.	A disease of the small blood vessels of the brain, which causes small strokes and areas of bleeding Hypertension, high cholesterol, diabetes, and smoking are the main risk factors	Typically over 40 years of age, risk increases with age Rarely a genetic component	Issues with problem solving, concentration, and planning; depressed or angry moods, daily fluctuations in ability, apathy	Length of life after symptom onset varies widely from 3-30 years Step-wise progression that is often unpredictable and spotty
Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts & Leukoencephalopathy (CADASIL) https://www.ninds.nih.gov/Disorders/All-Disorders/CADASIL-Information-Page	Caused by widespread areas of damage to the deep layers of white matter in the brain. This results in narrowing of the arteries that supply blood to the brain Hypertension, high cholesterol, diabetes, and smoking are the main risk factors, most individuals have a history of strokes	Early 50s Caused by an inherited genetic mutation	Often the initial symptom is migraine with aura episodes Focal neurologic symptoms are common (deficit affecting one particular part of the body), as are seizures, vision issues, and psychiatric problems (e.g., severe depression or personality changes) May also be at increased risk for heart attacks	15-20 years of life after patient's first stroke Step-wise progression that is often unpredictable and spotty
Normal Pressure Hydrocephalus (NPH) https://www.ninds.nih.gov/Disorders/All-Disorders/Normal-Pressure-Hydrocephalus-Information-Page	Caused by excess cerebrospinal fluid accumulation in the brain, causing brain ventricle enlargement and damage of surrounding cells	Over 60 years of age	Difficulty walking, loss of bladder control, cognitive function issues	Can be treated surgically with a shunt, good prognosis for recovery if treated in a timely manner
Binswanger Disease Also known as **White Matter Disease** or Subcortical Vascual (Vascular?) Dementia https://www.ninds.nih.gov/Disorders/All-Disorders/Binswangers-Disease-Information-Page	Is actually a syndrome with multiple causes	50 years of age The risk factors have a genetic component	Issues with depression, apathy, inactivity, and difficulty in decision-making and judgement are often the first signs. Later, issues with speech, swallowing, incontinence, and movement typically occur.	Length of life after symptom onset varies widely from 3-15 years Step-wise progression that is often unpredictable and spotty

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Infectious and Prion Diseases				
Creutzfeldt-Jakob Disease (CJD) https://www.cdc.gov/prions/cjd/index.html	Triggered by a prion, a type of protein that can cause disease	Varies based on subtype	Sudden issues with depression, agitation, apathy, mood swings, confusion, disorientation, memory, vision, hallucinations, muscle stiffness, judgement and planning, and involuntary, jerky movements	Varies based on subtype Typically: sudden onset of symptoms and fast progression of disease
Sporadic Creutzfeldt-Jakob Disease https://pubmed.ncbi.nlm.nih.gov/29887134/	Triggered by a prion, a type of protein that can cause disease	65 years of age Cause is unknown, disease appears without warning	Sudden issues with depression, agitation, apathy, mood swings, confusion, disorientation, memory, vision, hallucinations, muscle stiffness, judgement and planning, and involuntary, jerky movements	Length of life after symptom onset is typically less than one year
Familial or Genetic or Hereditary Creutzfeldt-Jakob Disease https://www.alzheimer-europe.org/Dementia/Other-forms-of-dementia/Infectious-diseases/Human-Prion-Diseases/Familial-CJD	Triggered by a prion, a type of protein that can cause disease: Caused by an inherited genetic mutation on a prion-related protein gene, so abnormal prions accumulate in the brain	50 years of age	Sudden issues with depression, agitation, apathy, mood swings, confusion, disorientation, memory, hallucinations, vision, muscle stiffness, judgement and planning, and involuntary, jerky movements	Length of life after symptom onset typically less than one year Extremely rapid progression from symptom onset to dementia symptoms
Acquired Creutzfeldt-Jakob Disease https://case.edu/medicine/pathology/divisions/national-prion-disease-pathology-surveillance-center/human-prion-disease/acquired-cjd	Triggered by a prion, a type of protein that can cause disease One type, Variant CJD (vCJD), develops from eating beef infected with Bovine Spongiform Encephalopathy (BSE), known as “mad cow disease” Acquired CJD can also occur via blood transfusion	30 years of age No genetic component	Very rapid development of issues with memory and behavior	An average of 18 months of life after symptom onset

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Fatal Familial Insomnia (FFI) https://rarediseases.org/rare-diseases/fatal-familial-insomnia/	Triggered by a prion, a type of protein that can cause disease Caused by an inherited mutation in a prion-related protein gene, so abnormal prions accumulate in the thalamus of the brain	45-50 years of age Inherited mutation in a prion-related protein gene	Insomnia is typically the first symptom, mild initially then progressively worsens Dementia, movement issues, anxiety, sexual dysfunction, depression, autonomic dysfunction, and often occur Sometimes dementia is the first symptom	1-6 years of life after symptom onset
Sporadic Fatal Insomnia (sFI) https://www.merckmanuals.com/professional/neurologic-disorders/prion-diseases/fatal-insomnia	Triggered by a prion, a type of protein that can cause disease Is not caused by a gene mutation	50-55 years of age No genetic component	Cognitive decline and movement issues are typically the first symptoms Insomnia is not usually a main symptom but can be observed by a sleep study and can be used as a diagnostic tool	1-8 years of life after symptom onset
Variably Protease-Sensitive Prionopathy (VPSPr) https://www.merckmanuals.com/professional/neurologic-disorders/prion-diseases/variably-protease-sensitive-prionopathy-vpspr	Triggered by a prion, a type of protein that can cause disease May have a genetic component, but no specific gene mutation has been identified	70 years of age May have a genetic component	Psychiatric symptoms, speech issues, and cognitive impairment are initial symptoms Movement issues often develop later	An average of two years of life after symptom onset
Gerstmann-Straussler-Scheinker Disease (GSS) https://www.ninds.nih.gov/Disorders/All-Disorders/Gerstmann-Straussler-Scheinker-Disease-Information-Page	Triggered by a prion, a type of protein that can cause disease Caused by an inherited mutation in a prion-related protein gene, so abnormal prions accumulate in the brain	45 years of age Inherited mutation in a prion-related protein gene	Movement issues such as unsteadiness and shakiness are often initial symptom Dementia typically occurs later	An average of five years of life after symptom onset
Post-Encephalitic Parkinsonism (PEP) https://www.alzheimer-europe.org/Dementia/Other-forms-of-dementia/Infectious-diseases/Postencephalitic-Parkinsonism-PEP	A complication of an encephalitic viral infection that was seen after World War I, triggered degeneration of nerve cells in the substantia nigra of brain	Dependent upon age of encephalitic infection No genetic component	Movement issues, gait instability, swallowing issues, cognitive impairment	9-24 years of life after symptom onset

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HIV-Associated Dementia (HAD) Previously named AIDS Dementia Complex https://www.hopkinsmedicine.org/health/conditions-and-diseases/hiv-and-aids/hiv-and-dementia	Majority of people with HIV do not develop dementia, but HAD can occur if the HIV infection affects subcortical brain neurons	60-70 years of age No genetic component	Memory problems, movement issues, anxiety, cognitive impairment, speech problems, and mood swings	Length of life after symptom onset is an average of six months, some treatments may prolong life
General Paresis or Dementia Paralytica or **Late Neurosyphilis Dementia** https://medlineplus.gov/ency/article/000748.htm	Triggered by a syphilis bacterial infection	Dependent on age of syphilis infection No genetic component	Psychiatric symptoms most common	Rapidly progressive course of disease, dementia can occur 10-30 years after initial syphilis infection

Metabolic Diseases				
Batten Disease Common name for several forms of disorders known as neuronal ceroid lipofuscinoses (NCLs) https://www.ninds.nih.gov/Disorders/Patient-Caregiver-Education/Fact-Sheets/Batten-Disease-Fact-Sheet	A gene defect affects the cells' ability to recycle certain molecules, resulting in an abnormal buildup in the cells that impairs function	Varies based on subtype, but most begin in early childhood Caused by an inherited gene mutation	Vision loss, seizures, developmental delay, loss of previously acquired skills, abnormal movements, dementia, personality changes, insomnia, speech issues	Varies based on subtype
Gaucher Disease https://www.alzheimer-europe.org/Dementia/Other-forms-of-dementia/Metabolic-diseases/Cerebral-Lipidoses/Gaucher-disease	Triggered by a metabolic disorder enzyme deficiency, which can result in accumulation of glucocerebroside in the brain	Varies depending on subtype, from infancy to older adults Caused by an inherited gene mutation	Abdominal complaints, skeletal abnormalities, and blood disorders are common Dementia, seizures, and vision issues may also occur	Varies depending on subtype

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Traumatic Diseases				
Chronic Traumatic Encephalopathy (CTE) Previously referred to as Dementia Pugilistica https://www.nhs.uk/conditions/chronic-traumatic-encephalopathy/#:~:text=Chronic%20traumatic%20encephalopathy%20(CTE)%20is,are%20based%20on%20ex%2Dathletes.	Damage and shrinkage of brain nerve cells in the cerebral cortex and basal ganglia, triggered by traumatic injury	Can occur many years after brain trauma	Movement issues, cognitive impairment, aggression, and personality changes are common	Varies depending on extent of trauma

Diseases Related to Toxic Substances				
Wernicke-Korsakoff Syndrome (WKS) Also known as alcohol-related dementia	Triggered by a deficiency of Vitamin B1 (thiamine), which is most common in those who chronically abuse alcohol or have malabsorption disorders	Varies depending on age of onset of alcohol abuse No genetic component	Impaired memory, including long-term memory gaps, which the person may try to fill in with incorrect versions of what they think happened (confabulation) Also can cause lack of coordination, vision issues, and personality changes	Varies depending on severity of the thiamine deficiency
Methamphetamine-Induced Neurodegeneration https://www.sciencedirect.com/science/article/pii/S0074774209880057	Triggered by methamphetamine abuse, which is toxic to the brain and damages cells	Varies depending on age of onset of drug abuse No genetic component	Memory problems, cognitive impairment, movement issues, personality changes	Varies depending on the severity of the damage

Other Dementias				
Mixed Picture Dementia	A combination of more than one type of dementia	Varies depending on the types of dementia	Varies depending on the types of dementia	Varies depending on the types of dementia