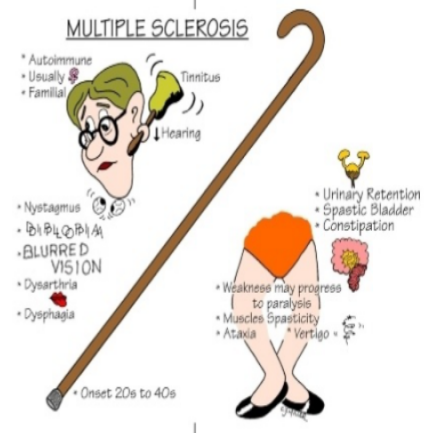
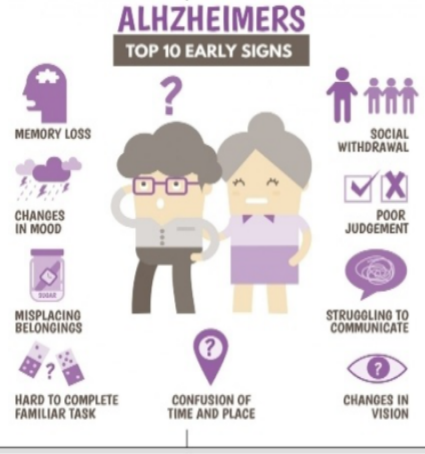
Disease	Feature	Cause	Sign & Symptoms	Histology & Microscopically	Other
Multiple Sclerosis (MS)	• Most common inflammatory demyelinating disease • Cause of **non trauma** related **neurologic disability** • Distinct **episodes** of neurologic deficits that are **separated in time** and are attributable to patchy white matter lesions that are **separated in space**. 	**Autoimmune** response directed against components of the myelin sheath.	• Progressive / chronic / attacks • Motor / Sensory / Visual • Course is variable, commonly multiple relapses followed by episodes of remission ; • recovery during remissions is not complete. • Patients present with one or more distinct episodes of CNS dysfunction • **Unilateral visual impairment** & optic neuritis due to ***optic nerve*** involvement • ***Brainstem*** involvement produces → cranial nerve signs ; ataxia & nystagmus • ***Spinal cord*** lesion give rise → to motor & sensory impairment. • Uhthoff phenomenon: heat and exercise **worsen** symptoms • CSF : presence of **oligoclonal IgG bands.**	• Lesions→ plaques: rounded, tan-gray and variably sized with a sharp demarcation from the surrounding brain tissue • ***Active plaques*** (ongoing myelin breakdown) 1. contain abundant macrophages 2. perivascular cuffs of Lymphocytes. • ***Inactive plaques*** (quiescent) 1. Inflammation disappears 2. leaving little to no myelin 3. Gliosis.	• Young adult • Diagnosis : 1. Clinical 2. MRI 3. CSF : presence of oligoclonal IgG bands. • Treatment : 1. High dose glucocorticoids 2. Monoclonal antibodies
Alzheimer Disease (AD)	• neurodegenerative diseases • **Progressive loss** of particular groups of **neurons**, which often have shared functions • The accumulation of **protein aggregates** 	**Aβ (amyloid β) and tau proteins accumulation** • amyloid β is derived from cleavage of Amyloid precursor protein (APP) by the enzymes β- and γ-secretase. • Defective clearance of Aβ results in its accumulation as amyloid fibrils. • Neurofibrillary tangles made from insoluble polymers of over-phosphorylated microtubule associated protein tau . • These deposits interfere with cellular functions by displacing organelles	• Insidious onset of impaired higher intellectual function • memory impairment • altered mood and behavior. • Over time ,patients come to require assistance with basic activities of daily living • The time from diagnosis to death varies from as little as 3 years to as long as 10 or more years	• **Aβ** is toxic to neurons it causes damages synapses, and kills neurons • **Tau proteins** impair the axonal transport thus affecting the nutrition of axon terminals and dendrites. 1. Cortical atrophy 2. widening of the cerebral sulci that is most pronounced in the **frontal, temporal, and parietal lobes.** 3. Amyloid plaques {Senile plaques}{ Alzheimer's plaques}(extracellular – accumulation of **Aβ**) , Neuritic plaques are focal, spherical collections of dilated, tortuous, processes of dystrophic neurites around a central amyloid (Aβ) core . 4. neurofibrillary tangles (intracellular– **Tau** accumulation) ,Tau containing bundles of filaments in neurons cytoplasm ,flame shapes	• Most common cause of dementia in older adults • Eventual feature of the cognitive impairment in **trisomy 21 individuals** (**Down syndrome**) • Aβ is a 36 to 43 amino acid peptide • Amyloid precursor protein (APP) is a transmembrane protein