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A Systematic Review of Neurological and Psychiatric Disorders

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ABSTRACT
The structure and functions of the brain were questioned and studied since Ancient Greek times and led to the compilation of enormous information on the subject globally. With the advent of new technology, the researchers are able to discover the causes of brain diseases/disorders. Due to the stressful life, brain disorders are considered as a significant global healthcare problem. It has generated a great need for continuous research for understanding brain structure as well as functions in context to health and diseases. In the present review, we have compiled various diseases and disorders related to the brain, along with their symptoms and the treatment strategies.

Keywords: Nervous system, Part of brain, Diseases, Disorder, Neurodegeneration

I. INTRODUCTION
The brain, which is housed inside the skull and consists of a vast mass of nerve cells, is the command canter of the Central Nervous System (CNS) [1–3]. The brainstem, cerebellum, and cerebrum are its three principal components. It regulates the body's mental processes, such as organizing, integrating, and digesting the data from the senses. organs. It is a jelly-like mass of tissue with 86 billion nerve cells and a weight of around 1.4 kg [4–7]. The brainstem, which in turn leads to the spinal cord, is linked to the cerebrum. The midbrain, pons, and medulla oblongata are the three components that make up the brainstem. The thalamus, pineal gland, hypothalamus, pituitary gland, amygdala, and other brain regions are located beneath the cerebral cortex the hippocampus. Each brain hemisphere's cross-section reveals a ventricular chamber that produces and circulates cerebrospinal fluid. The septum pellucidum, a membrane that divides the lateral ventricles, is located underneath the corpus callosum [8, 9]. The greatest portion of the human brain is called the cerebrum. It is separated into two cerebral hemispheres, each of which has two thirds of the brain's total mass. Speech and language are controlled by one hemisphere, which is functionally dominant. Visual and spatial information is interpreted by the other hemisphere. The bundle of nerve fibers that connects the left and right hemispheres of the human brain is known as the corpus callosum., Each hemisphere is composed of four lobes: the frontal, temporal, parietal, and occipital lobes [10–13]. The frontal lobe regulates voluntary movements and cognitive processes, including language, problem-solving, emotional expression, and memory sexual habits and judgment [14]. The primary auditory cortex, which receives the primary auditory perceptions like hearing, is located in the temporal lobe. sensory data from the ears and secondary regions, and transform it into meaningful phrases that are conveyed through words and speech [15]. Temperature, taste, touch, and movement are all processed by the parietal lobe [14]. Vision is mostly controlled by the occipital lobe [16].

BRAIN CELLS
Neurons are brain cells, whereas glial cells are the supporting non-neuron cells. There are about 86 billion neurons in the average adult human brain. Numerous studies have proposed that the brain's correct operation depends on both neurons and glial cells. Electrical and metabolic signals are sent and received by neurons in the brain [17]. Three fundamental components make up a neuron: the axon, branching dendrites, and the cell body, or soma. They serve as the brain's building blocks and carry information to the body's muscles, tissues, and other neurons.

Thinking, feeling, moving, and understanding our surroundings are all made possible by neurons. Another crucial component of the neurological system are glia cells. The Latin word for "glue" is "glia" [18]. Glial cells are essential for the proper operation of neurons and actively participate in brain signaling. The brain has a wide variety of glial cells. Oligodendrocytes are one of the three major types of glial cells: They aid in axon insulation and let electrical signals to travel over great distances at a remarkable speed;

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Microglia:Also referred to as immune cells of the central nervous system, microglia travel throughout the brain and interact with other glia cells continuously; Astrocytes: These are star-shaped cells that sustain the blood-brain barrier (BBB), nourish neurons, heal nerve damage, and aid in neurotransmission [19–21].

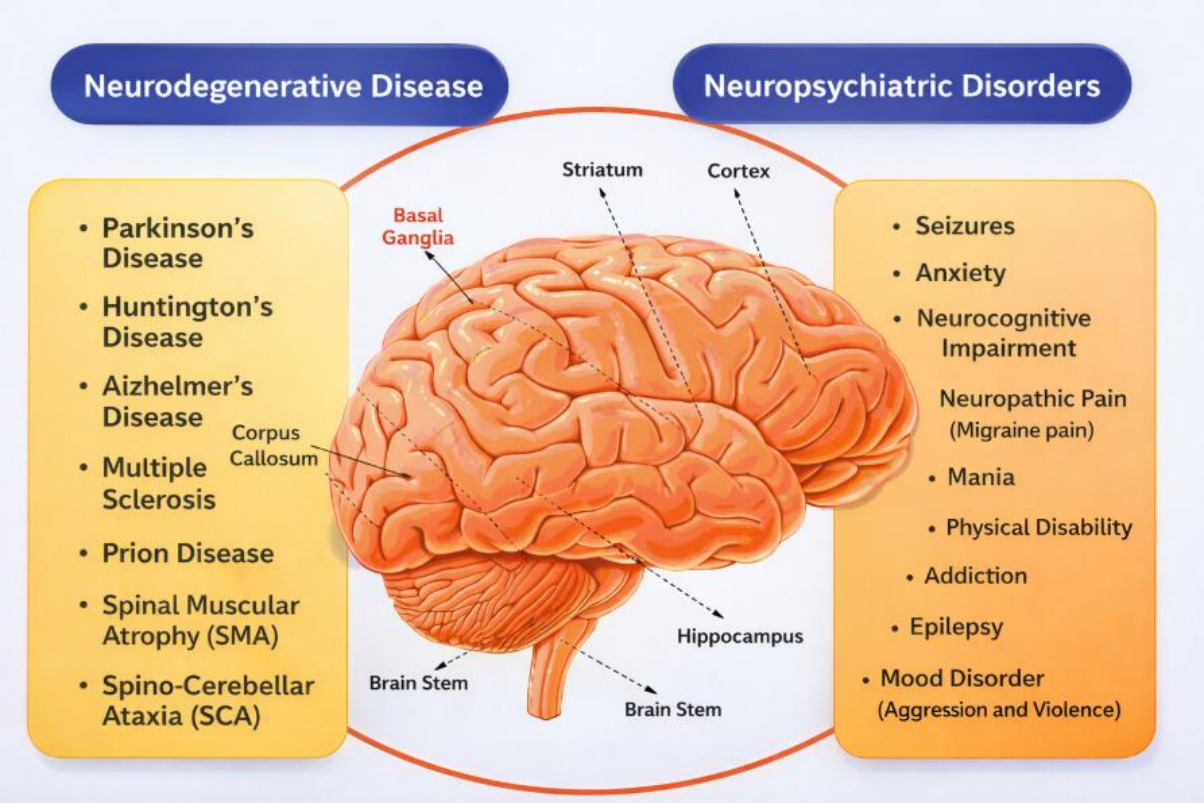

Figure1. significant neuropsychiatric conditions and neurodegenerative diseases in humans.

BLOOD BRAIN BARRIER (BBB)

Blood arteries are essential for supplying the body's tissues and organs with nutrients and oxygen. The CNS's distinct blood vessel system, which makes up the blood-brain barrier, enables these vessels to strictly control the flow of between the brain and the blood, including ions, chemicals, and cells [22]. The BBB's function is to shield the brain from viruses or circulating poisons while simultaneously permitting essential nutrients to enter the brain. The brain capillary endothelium forms the blood-brain barrier [23]. Additionally, it keeps the delicate balance of water, nutrients, and hormones in the brain's environment [24]. One of the biggest challenges in treating nearly every kind of brain condition is getting therapeutic medicines to the right parts of the brain. Many potentially significant therapeutic and diagnostic substances are unable to reach the brain due to the BBB. Antibodies and therapeutic compounds that could otherwise be useful in treatment do not sufficiently pass the blood-brain barrier [25].

Table. 1 Types of brain illnesses and diseases in humans, along with their signs and remedies

Category	Parts of the Brain affected	Type of Diseases/ Disorders	Treatment	Symptom	Reference
Autoimmune Diseases	Brain cells	Autoimmune encephalitis	Tumor removal and intravenous immunosuppressive treatment.	Aberrant movements, convulsions, memory and cognitive impairments, balance issues, speech or eyesight, psychosis, hostility, improper sexual behavior, panic attacks,	[28,29]

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				obsessive habits, euphoria, or terror.	
	Brain cells	Autoimmune epilepsy	Corticosteroids combined with plasma exchange or intravenous immunoglobulin	Uncontrollably frequent seizures.	[30,31]
	Inflammation of blood vessels in the Brain	Central nervous system vasculitis	cyclophosphamide with high-dose steroids like prednisone.	Headaches, strokes or brief ischemic episodes, disorientation or forgetfulness weakness, visual issues, convulsions, and encephalopathy (brain swelling).	[32,33]
	Venules with lymphocytic vasculitis and vasculature in the brain stem and a widespread gliosis affecting either white or gray matter	Hashimoto's encephalopathy	Corticosteroids	Changes in personality, hostility, delusional conduct, focus, and memory issues, unconsciousness, disorientation, headaches, muscle jerks, poor coordination, partial paralysis on the right side, psychosis, seizures (60% of cases), irregular sleep patterns, tremors, and speech issues.	[34,35]
	Myelin in the spinal cord and eye nerves	Neuromyelitis optical	Modest dosages of carbamazepine and intravenous corticosteroids.	Severe spasms, loss of feeling, weakness or paralysis in the arms or legs, and uncontrollable hiccups and vomiting, as well as bowel or bladder problems brought on by spinal cord injury.	[36,37]
	The optic nerve and its fatty covering (myelin) are irritated	Optic neuritis	vitamin B12 and intravenous immune globulin.	Vision becomes fuzzy or faint, and color vision is lost.	[38, 39]
	Cranial and facial nerves, hypothalamus	Neurosarcoidosis or neurosarcoid	Radiation, corticosteroids, immunosuppressive drugs, and painkillers therapy, physical therapy, and occupational therapy.	Bell's palsy, which causes hearing loss, double vision, and one-sided facial muscle weakness headaches, difficulty speaking, agitation, memory loss, mood swings, dementia, hallucinations, and seizures.	[40, 41]
	Ventral brain stem	Neuro-Behcet's disease	Corticosteroids with either methotrexate or azathioprine. For people who are at high risk, intravenous corticosteroids and cyclophosphamide.	Fever, headache, skin lesions, genital scarring, and ulcers.	[42,43]
	Brain cells	Cerebral lupus or Systemic Lupus	NSAIDs (nonsteroidal anti-inflammatory medications) like ibuprofen and naproxen	Fatigue, fever, swelling and stiffness in the joints, butterfly-shaped rash on the face, and	[44,45]

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		Erythematosus (SLE)	sodium (Aleve), Antimalarial medications include mycophenolate mofetil, methotrexate, rituxan, corticosteroids prednisone, immunosuppressants azathioprine (imuran, azasan), and hydroxychloroquine (plaquenil).	skin lesions, dry eyes, shortness of breath, chest pain, and fingers and toes turning white or blue in cold or stressful situations.	
	Hippocampus, amygdala, cerebral lobes, ventricles, and caudate nucleus.	Autism or Autism Spectrum Disorder (ASD)	Therapies for behavior and communication, such as play therapy, behavioral therapies, and sensory treatments, occupational therapy, speech therapy, and a single medication, risperidone (risperdal).	Adoption of odd speech patterns, like avoiding eye contact and speaking in a robotic tone with others, being unable to answer their name, having delayed speech development, unable to carry on a conversation, repeating words a lot, and seemingly having trouble comprehending and expressing their own emotions.	[46, 47]
Dementia	Degradation of the frontal and temporal lobes.	Fronto-temporal dementia	Antidepressant drugs and Antipsychotic drugs.	Lack of inhibition, socially unacceptable behavior, poor judgment, lack of empathy, and repetitious obsessive behavior, difficulty focusing or making plans, frequent, sudden mood swings, and trouble speaking	[48,49]
	Clumps of a protein in the cortex.	Dementia with Lewy bodies	Cholinesterase inhibitors, carbidopa and levodopa (sinemet, rytary, dopa).	Sleep disturbances, hallucination, imbalance, movement difficulties.	[50, 51]
	Blocking of blood vessels.	Vascular dementia	High blood pressure under control through exercise, diet and medication.	Problems with short-term memory, laughing or crying at inappropriate times, trouble in concentrating, planning, or trouble in managing money, inability to follow instructions, loss of bladder or bowel control and hallucinations or delusions.	[52, 53]
	Destroy the connections between neurons in sections of the brain and has an impact on the cerebral cortex (plaque deposition of improperly folded tau and amyloid β-protein in the brain).	Alzheimer's Disease (AD)	Rivastigmine (Exelon), Tacrine (Cognex), and Donepezil (Aricept)	Loss of energy, disinterest in social and professional interests, and memory loss, such as forgetting conversations and recent events, mood swings, despair, linguistic issues, and coordination issues.	[54, 55]
Brain Infections	Inflammation of the meninges, brain and spinal	Meningitis	Antibiotics administered intravenously, fluids administered intravenously	Reduced appetite, agitation, drowsiness, lethargy, fever, light sensitivity,	[56, 57]

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	cord.		to avoid dehydration, steroid medicine to lessen any swelling around the brain, and oxygen via a face mask if breathing becomes problematic.	drowsiness, dizziness, nausea, and vomiting.	
	Temporal lobe, frontal lobe	Encephalitis	Corticosteroids, ganciclovir (Cytovene), and acyclovir (Zovirax).	Headache, fever, joint or muscular aches, weakness or exhaustion, disorientation, agitation, or hallucinations, convulsions, paralysis or lack of feeling in specific facial or body parts, weakness in the muscles, difficulty speaking or hearing, and unconsciousness.	[58, 59]
	Fungal and viral infection in brain	Brain abscess	Antibiotic medications and surgery.	Differences in mental processes, such as increased confusion, decreased responsiveness, and irritability, speech, sensation, decreased movement due to the loss of muscle function, changes in vision, changes in personality or behavior, vomiting, fever, chills, neck stiffness, especially when it occurs with fever and chills, sensitivity to light.	[60, 61]
Movement Disorders	Cerebellum	Ataxia	Physical and mental exercise, occupational treatment, and speech and language therapy.	Inadequate coordination, a shaky gait and propensity for stumbling, challenges with fine motor skills, such as swallowing difficulties, involuntary back-and-forth eye motions (nystagmus), eating, writing, or buttoning a shirt.	[62, 63]
	Basal ganglia	Dystonia and Cervical dystonia	Procyclidine, hydrochloride, diazepam, lorazepam, clonazepam, levodopa, and Baclofen	Leg dragging, foot cramps, neck pulling that is uncontrollable, blinking that is out of control, and speech issues.	[64, 65]
	Basal ganglia	Chorea	Drugs such as Quetiapine, Haloperidol (Haldol), Olanzapine (Zyprexa), and Fluphenazine (Prolixin) (Seroquel), Risperidone (Risperdal).	Issues with posture, movement, speaking, and swallowing.	[66, 67]
	Nerve cells and basal ganglia	Huntington's Disease (HD)	Antipsychotic medications like haloperidol (Haldol), medications like tetrabenazine (Xenazine), and Chlorpromazinamantadine Clonazepam (Klonopin) and Levetiracetam (Keppra, etc.).	Uncontrollably jerking or writhing motions, muscular issues such stiffness or contracture, slow or irregular eye movements, poor balance, walking, and posture, and trouble speaking or swallowing.	[68, 69]

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	Cerebellum, basal ganglia and brainstem	Multiple System Atrophy (MSA)	Epyridostigmine (Mestinon), midodrine Levodopa, corticosteroid Fludrocortisone, and Carbidopa (Sinemet, Duopa).	Impaired coordination and mobility, including slurred speech, unsteady stride, and loss of balance visual abnormalities, difficulty chewing or swallowing (dysphagia), constipation, restless nights as a result of "acting out" dreams, uneven breathing, loss of libido, irregular heartbeat, and trouble regulating emotions.	[70, 71]
	Cerebral cortex, cerebellum and brainstem	Myoclonus	Anticonvulsants like levetiracetam (Keppra), tranquillizers like Clonazepam (Klonopin), Deep brain stimulation (DBS), piracetam onabotulinumtoxin A (Botox) injections, Roweepra, Spritam, Valproic acid (Depakene), and Primidone (Mysoline).	Shock that is abrupt, short, involuntary, varied in frequency and intensity, and restricted to a single area of the body or throughout the body, occasionally severe enough to make it difficult to walk, speak, or eat.	[72,73]
	Nerve cells, basal ganglia and substantia nigra	Parkinson's Disease (PD)	Levodopa, MAO-B, dopamine agonists, and the most efficient carbidopa-levodopa infusion inhibitor, anticholinergics, amantadine, DBS, and catechol O-methyltransferase (COMT) inhibitors.	Tremor, trembling hands or fingers, bradykinesia (slowed movement), stiff muscles, and poor posture and balance, lack of reflexive motions, alterations in speaking and writing.	[74, 75]
	Basal ganglia and the brain stem	Progressive supra nuclear palsy or Steele Richardson Olszewski syndrome	Occupational and physical therapy, drugs such as onabotulinum toxin type A (Botox), prism or bifocal spectacles.	Stiffness and unnatural motions, difficulties swallowing and speaking, sensitivity to light, insomnia, disinterest in enjoyable activities, impulsive conduct, which may include uncontrollably laughing or sobbing, memory, reasoning, problem-solving, and decision-making issues, sadness, and anxiety.	[76, 77]
	Cingulate cortex and cerebellum	Restless Legs Syndrome (RLS)	Rotigotine (Neupro), Pramipexole (Mirapex), Gabapentin (Neurontin, Gralise), Ropinirole (Requip), Muscle relaxant Gabapentin Enacarbil (Horizant), Pregabalin (Lyrica), Tramadol (Ultram, ConZip), Codeine, Oxycodone (Oxycontin, Roxicodone, and others), and Hydrocodone (Hysingla ER, Zohydro ER)	Leg twitching, sensations following rest, and symptoms getting worse at night and in the evening.	[78, 79]

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	Striatum and basal ganglia	Tardive dyskinesia	Drugs such as Valbenazine (Ingrezza), Deutetrabenazine (Austedo), Vitamin B6, vitamin E, and melatonin	lip puckering, lip smacking, lip pursing, tongue movements, and excessive eye Blinking	[80,81]
	Basal ganglia	Tourette syndrome	Drugs that reduce or block dopamine, injections of botulinum (Botox), drugs for ADHD, Deep brain stimulation, behavior therapy, psychotherapy, antidepressants, central adrenergic inhibitors, and anti-seizure drugs.	Blinking of the eyes, jerking of the head, repetition of motions seen, shrugging of the shoulders, stepping in a specific pattern, eye flashing, obscene gestures, lip motions, jumping, nose twitching, and bending or twisting.	[82, 83]
	Brain and spinal cord	Brain and spinal cord	Drugs such as T-Zinc acetate (Galzin), Trientine (Syprine), and Penicillamine (Cuprimine, Depen) as well as surgery.	Exhaustion, appetite loss or stomach discomfort, skin discoloration, and white eyes (jaundice), golden-brown eye discoloration (Kayser-Fleischer rings), fluid accumulation in the legs or belly, issues with swallowing, speaking, or physical coordination, as well as uncontrollably moving or rigid muscles.	[84, 85]
Neuromuscular Diseases	Degeneration of the spinal cord's nerve cells and the brain.	Amyotrophic Lateral Sclerosis (ALS)	Respiratory, speech, occupational, physical, and nutritional treatments.	Muscle twitching and cramping, impairment, and loss of hand and arm motor control in the use of the arms and legs, tripping and falling, dropping objects, chronic exhaustion, uncontrollably laughing or sobbing, thick or slurred speech, and difficulty projecting the voice.	[86, 87]
	Axon (myelin sheath)	Charcot-Marie-Tooth disease	Orthopedic devices, occupational therapy, physical therapy, and painkillers.	Foot abnormalities, weakness in the lower leg and foot muscles, and trouble lifting the foot while walking, numbness, tingling, burning, or loss of temperature sensation in the hands and feet, as well as pain or discomfort in these areas.	[88, 89]
	Brain, spinal cord and nerve cells	Multiple sclerosis	Exercise regimens that improve muscle control, strength, and endurance.	Red-green color distortion, blurred or double vision, discomfort, and vision loss due to swelling of the optic nerve (optic neuritis), difficulty walking, numbness, prickling, or pins and needles (paresthesia), arm and leg muscle weakness, difficulty coordinating, exhaustion, loss of feeling,	[90, 91]

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				speech issues, tremors, lightheadedness, hearing loss, bowel and bladder issues, and depression.	
	Lack of the protein dystrophin in neurons	Muscular dystrophy	Prednisone, surgery, and physical therapy and exercise.	Progressive atrophy and weakness of the muscles, a waddling gait, and trouble climbing stairs, frequent falls, spinal curvature, thigh muscle atrophy, aberrant calf enlargement, breathing and swallowing difficulties, and heart enlargement.	[92, 93]
	Neurons	Myasthenia gravis	Steroids, anti-cholinesterase medications, or immune-suppressive medications reaction, thymectomy, and plasmapheresis	Visual issues such as double vision (diplopia) and drooping eyelids (ptosis), muscle weakness and exhaustion, as well as limb or neck weakness.	[94, 95]
	Brain bubbles	Myositis, encompassing dermatomyositis and polymyositis	Physical therapy and exercise, steroids, intravenous immunoglobulin therapy, Injection of rituximab	Weakness in the muscles, soreness in the muscles, intense fatigue, and overall sick, dyspnea.	[96, 97]
	Nerves in the brain and brainstem	Peripheral neuropathy	Antidepressants, topical medicines, painkillers, anti-seizure drugs, therapies, transcutaneous electrical nerve stimulation (TENS).	Numbness that develops gradually, jabbing, searing or throbbing discomfort, lack of coordination, weakening of the muscles and paralysis if the motor neurons are damaged.	[98, 99]
	Spinal cord and brain stem	Spinal muscular atrophy	Drugs such as spinraza and zolgensma.	Weakness and twitching of the muscles, breathing and swallowing difficulties, and morphological alterations of the limbs, spine, and chest as a result of weak muscles, making it difficult to stand and walk.	[100, 101]
Seizure Disorders	Brainstem, basal ganglia, cerebellum, and thalamus.	Tonic-clonic seizures or Grand- mal seizure	Medications such as valproic acid, phenytoin (Dilantin, Phenytek), and carbamazepine (Carbatrol, Tegretol) (Depakene), oxcarbazepine (Oxtellar, Trileptal), Lamotrigine (Lamictal), Gabapentin (Gralise, Neurontin), Topiramate (Topamax), Phenobarbital, and Zonisamide (Zonegran)	Bewilderment, exhaustion, loss of control over one's bowels and bladder, and unresponsiveness following convulsions with a bad headache.	[102, 103]
	Alterations in brain function	Atonic seizures	Ethosuximide (Zarontin) and Valproic acid (Depakene)	When a person suddenly loses muscle strength, goes limp, and falls to the ground while seated, their	[104, 105]

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				Head nods, jerking motions, drooping eyelids, and an abrupt drop in head height will all occur while the person is still conscious.	
	Temporal lobe	Myoclonic seizures	Anti-seizure medication, nerve stimulation, dietary therapy or surgery.	Abrupt spasms, rapid increases in muscle tone, and occasionally dozing off.	[106]
	Thalamus	Absence seizures or Petit mal seizures	Ethosuximide (Zarontin), Valproic acid (Depakene) and Lamotrigine (Lamictal).	abrupt halt without falling, lip-smacking, fluttering eyelids, biting, finger rubbing and little hand movements.	[107,108]
Stroke Diseases	Alterations in brain functioning.	Trauma	Prompt medical attention, medication, surgery, and rehabilitation.	loss of consciousness for a few seconds to many minutes, a state of confusion or disorientation, headache, nausea or vomiting, exhaustion or sleepiness, speech issues, trouble falling asleep, lightheadedness, and loss of balance.	[109, 110]
	Brain cells	Tumors	Radiation, minimally invasive brain surgery that leaves no scars, medication, surgery, and rehabilitation treatment, targeted medication therapy, and chemotherapy.	Headache, nausea, vomiting, and visual issues such double vision, blurred vision, or lack of peripheral vision, hearing issues, speech challenges, and a progressive loss of feeling or movement in an arm or leg.	[111, 112]
Mental Disorders	Amygdala	Anxiety disorders, including panic disorder, obsessive compulsive disorder, and phobias	Antidepressants, buspirone, and benzodiazepines are among the medications used in psychotherapy.	Exhaustion, difficulty falling asleep, tense or aching muscles, shaking, twitchiness, anxiety, perspiration, nausea, diarrhea, and agitation.	[113, 114]
	Cingulate cortex and frontal gyrus	Post-traumatic stress disorder	Psychotherapy, cognitive therapy, eye movement desensitization and reprocessing (EMDR). Medications like anti-depressants and anti-anxiety (Prazosin).	Negative thoughts, memory problems, difficulty in maintaining close relationships and emotionally numb.	[115,116]
	Medial frontal lobe	Psychotic disorders, including schizophrenia	Cornerstone, antipsychotic drugs, asenapine (saphris), aripiprazole (abilify), and brexpiprazole (rexulti), ziprasidone (geodon), risperidone (risperdal), quetiapine (seroquel), paliperidone (invega), and long-acting injectable antipsychotics.	delusions, hallucinations, disordered speech or thought patterns, severely chaotic or aberrant motor behavior, unpleasant symptoms, insomnia, irritation or depression, and a lack of drive.	[117, 118]
	Hippocampus	Depression, bipolar disorder, and	Mood stabilizers, anti-psychotics, anti-depressants, antipsychotic	Exaggerated feelings of well-being and self-confidence, as well as increased activity,	[119, 120]

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		other mood disorders	and anti-anxiety medications.	energy, or agitation (euphoria), irregular sleep patterns, excessive chatter, or making poor financial decisions.	
	Brain cells	Eating disorders	Cognitive behavioral therapy, family-based therapy (FBT), psychotherapy, and healthy eating (CBT), antidepressants, and anxiety drugs.	Abnormally low body weight and a severe, persistent fear of gaining weight regurgitating food after consumption.	[121, 122]
	Amygdala, pre frontal cortex	Personality disorders	Psychotherapy	Aggression toward humans and animals, property damage, dishonesty, stealing, severe breaking the law, taking advantage of people, having unhealthy or abusive relationships, and acting carelessly all the time.	[123, 124]

BRAIN DISORDERS

The entire body is impacted by any abnormalities, illnesses, or dysfunctions in the brain. Neurons and tissue infections can occur in the brain. Trauma (a mental illness) or a stroke (a lack of blood supply due to environmental or unintentional circumstances) can cause damage. Degeneration of brain cells happens with brain damage [26, 27]. Numerous internal and external elements are involved. While neurotoxicity refers to chemically generated neuronal damage, trauma-related brain damage is caused by individual variables or psychologically unstable conditions [26]. In general, there are two types of brain problems in humans: neuropsychiatric disorders and neurodegenerative diseases. (Figure 1). Both are difficult to comprehend and incurable, although they can be treated or their symptoms suppressed using medications, surgery, and physical therapy (Table 1). [28–124].

NEURODEGENERATIVE DISEASES

The progressive loss of neurons in neurodegenerative diseases is a composite type of conditions that impair the Central Nervous System's (CNS) ability to operate as well as the PNS, or peripheral nervous system. These illnesses demonstrate their effects on the human body's mental and physical functions, including speech, movement, and coordination, intelligence, and memory. Because the symptoms of these illnesses are similar, their causes are not specific. Because the symptoms of various illnesses are identical, it is impossible to pinpoint their causes. Alzheimer's disease (AD), Parkinson's disease (PD), prion disease, Huntington's disease (HD), and spino-cerebellar ataxia (SCA) are a few common neurodegenerative illnesses and SMA, or spinal muscular atrophy [125]. The brain's hippocampus, cortex, and amygdala have all been shown to experience neuronal death in AD [126], whereas substantia nigra pars in Parkinson's disease compacta exhibits neuronal loss, which results in a dopamine shortage [127].

The most common condition, Alzheimer's disease (AD), primarily affects those over 60. German physician Alois Alzheimer initially identified it in 1906 based on a patient named Auguste D. who had signs of memory loss as well as some psychological alterations including mood swings and unresponsive conduct. Emil Kraepelin, a colleague of German physician Alois Alzheimer, gave AD its name. He stated in 1910 that This illness was initially referred to as "Alzheimer's disease" in his medical book "Psychiatrie" [128, 129]. Emil Kraepelin, a colleague of German physician Alois Alzheimer, gave AD its name. He stated in 1910 that. `This illness was initially referred to as "Alzheimer's disease" in his medical book "Psychiatrie" [128, 129]. Progressive memory loss and cognitive deficits are its hallmarks. Extracellular amyloid β plaques and intracellular neurofibrillary tangles are two of AD's pathological

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hallmarks to brain atrophy, neuronal loss, and synaptic dysfunction [130].

After AD, Parkinson's disease (PD) is the second most prevalent neurodegenerative illness. It is a degenerative CNS condition that mostly affects how the body moves. Both motor and non-motor functions are impacted. Although the exact causes of Parkinson's disease (PD) are still unknown, both hereditary and environmental factors are thought to be involved for the development of the illness. Dr. James Parkinson (1817) wrote about it in his monograph "An Essay on the Shaking Palsy" [131, 132]. Lewy bodies build up in the substantia nigra pars compacta of the midbrain, and dopaminergic neurons are lost. Rigidity, akinesia, tremors, postural instability, and non-motor symptoms are the most common clinical symptoms [133].

Prion illness was identified as a neurological condition in humans by neurologists Hans Gerhard Creutzfeldt and Alfons Maria Jakob in 1920. Transmissible Spongiform Encephalopathies (TSEs), another name for prion disease, are uncommon progressive neurodegenerative conditions which impact both people and animals [134]. This illness causes a normal cell surface glycoprotein (PrPC) to change into a conformational altered isoform, or PrPSc. PrPSC is responsible for the neurological disorder [135]. Creutzfeldt-Jakob disease (CJD) and bovine spongiform encephalopathy (BSE, sometimes known as "mad cow" disease) are the two most prevalent prion diseases that infect humans. Prions are misfolded proteins with the ability to spread [136, 137].

Huntington's chorea is another name for Huntington's disease (HD). It is caused by the brain's neuronal cells gradually deteriorating. In 1872, George Huntington identified it as a genetic neurodegenerative illness [138]. The unstable trinucleotide repeat of cytosine-adenine causes this autosomal-dominant neurodegenerative illness Guanine (CAG). Involuntary motions, cognitive deterioration, and behavioral abnormalities are its clinical characteristics [139].

DISORDERS AND CONDITIONS OF NEUROPSYCHIATRY

A type of disorder or conditions known as neuropsychiatry deals with mental disruption brought on by abnormal brain activity. It is described as mental illnesses or brain disorders [140]. Berrios and Markova claim that neuropsychiatry explains how brain injuries can cause mental disorders. [141]. Neuropsychiatric illnesses have a detrimental effect on an individual's overall health and well-being. It impairs learning in childhood and makes it difficult to concentrate or focus at work in adulthood [141, 142]. Due to their comparable symptoms, they are complicated and difficult to comprehend. Seizures, attention or cognitive deficiency disorders, uncontrollable anger, migraine headaches, addictions, and eating disorders are a few typical neuropsychiatric conditions anxiety and depression. A person with neuropsychiatric problems exhibits altered conduct, such as criminal activity, hostility, violence, and antisocial personality disorder, episodic dyscontrol, impulse regulation problems, and psychopathy [143, 144]. Although the exact origins of brain illnesses are yet unknown, some environmental and genetic variables contribute to the illness. These conditions are quite common and have early onset (autism in childhood and schizophrenia in maturity) [145, 146]. The various types of brain illnesses, together with their signs and remedies, are compiled in Table 1.

NEUROPSYCHIATRY DISORDERS AND NEURODEGENERATIVE DISEASES CAUSES

It is impossible to overlook how genes and environmental factors contribute to the development of neurological diseases and illnesses. Any harm to the central nervous system causes cell death, which results in a loss of function [147]. The brain's regular functioning was impeded by the illnesses, which resulted in the gradual deterioration or abrupt loss of brain functions (sensory, motor, and cognitive) [148]. Tau protein and β-amyloid are two examples of neurodegenerative illnesses that are marked by aberrant protein accumulation in the brain tissue (plaque buildup in the form of neurofibrillary tangles) in AD, misfolded Huntingtin protein in HD, ubiquitinated protein aggregation in ALS, α-synuclein accumulation in PD, and cell surface glycoprotein accumulation in prion disease [149–152]. According to certain research, a gene mutation causes an accumulation of misfolded proteins.Physical injury to the brain may lead to synaptic insufficiency, massive cell death and inflammation that may lead to temporary or permanent loss of various bodily actions like coordination in the movement (ataxias) and different cognitive functions like memory, learning, decision-making skills, talking and dementia. Neurodegenerative disorders have no long-term cure. DBS and cell transplantation treatments are utilized to regulate or lessen the physiological and

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cognitive symptoms in advanced stages of the disorder's inadequacies [153–155].

β-amyloid protein aggregation in AD speeds up the development of neurofibrillary tangles, which cause synaptophysin in the form of glial inflammation and neurodegeneration cell loss in the brain's cingulate gyrus, temporal lobes, parietal lobes, cerebral cortex, and subcortical areas [156]. In Parkinson's disease (PD), intracellular protein buildup speeds up the loss of dopamine-producing neurons in the substantia nigra. The ubiquitin complex is linked to α-synuclein. Lewy bodies, which are cytoplasmic inclusions created by these protein aggregates, are important in both familial and occasional PD cases [133]. Huntingtin protein buildup inside cells is also the cause of DeCell in the brain's striatum area die as a result of the huntingtin gene mutation [138].One glial condition is multiple sclerosis (MS).It involves severe autoimmune damage to myelinated axons, which results in axonal damage and additional loss of neuronal transmission, primarily in the white the brain stem, the basal ganglia, and matter tracts [157].Likewise, it falls between 5 and 10% for PD [156]. However, HD is thought to be a purely hereditary condition brought on by expansions of tri-nucleotide repeats nucleotides (CAG) [138].The complete genome sequence may aid scientists and medical professionals in comprehending the genetic elements that are crucial to health, illness, as well as medication reaction [158].It is challenging to comprehend how hereditary and environmental variables interact to impair brain function [159].Research has shown that hereditary factors significantly contribute to the development of bipolar disorder and schizophrenia.It falls between 70 and 80 percent in a twin study.In a similar vein, genetic variables significantly increased the risk of depression by 38% to 75% [160, 161].One in four adults over 55 who have dementia have a family history of the condition [162].

A collection of symptoms associated with neurodegenerative disorders are collectively referred to as dementia.Significant cognitive dysfunctions that are sufficient to interfere with day-to-day functioning are caused by neurodegenerative diseases in their advanced stages.Dementia symptoms include loss of attentive function, visual impairment, memory and learning difficulties, and behavioural abnormalities [162].Additionally, epigenetic variables contribute significantly to the exacerbation or enhancement of disease-like symptoms.Certain metals, such as lead (Pb), mercury (Hg), arsenic (As), cadmium (Cd), and aluminium, have been linked to the development of AD and PD [163, [164].Neurological conditions including Paraquat (PQ) and 1-methyl l-4 phenyl l-1, 2, 3, 6-tetrahydropyridine are also significantly impacted by pesticides. (MPTP) [165].Similar to hazardous nanoparticles and trichloroethylene (TCE), rotenone has also been demonstrated to induce damage to neuronal cells and inappropriate CNS operation [166, 167].

NEURODEGENERATIVE DISEASE DATABASE

Building a database for neurodegenerative disorders is urgently needed due to the complicated pathophysiology and overlap of symptoms in different neurodegenerative diseases.An online database has been created by researchers (DND:More than 100 neuro-related illness ideas are included in the database of neurodegenerative disorders, which includes details about all linked genes, their products, and pathological routes and methods of therapy [168].In order to better understand the molecular and genetic processes involved in the disease's course and therapy, it offers a wealth of information on nearly every facet of neurodegenerative illnesses [168].With the assistance of a group of Penn researchers, University of Pennsylvania researchers have created a unique Integrated Neurodegenerative Disease. Database (INDD) for Fronto-temporal lobar degeneration, AD, PD, and ALS [169].Multiple database tables can be reliably and precisely queried from a single console using this database (Penn INDD).Comparative research of different neurodegenerative disorders can also benefit from it [169]. Kandale and associates.18 disorders have been added to the Integrated Database of Neurodegenerative disorders (IDND) by [170].Three different databases were used to prepare IDND: UniProt kB (protein information), kEGG (Pathway), and PubMed (disease papers).

Another specialized online resource that thoroughly gathers and meta-analyses all published research in the relevant topic is called PD Gene [171].Researchers can better understand the genetic architecture behind Parkinson's disease susceptibility with the aid of this database.ITGA8 was discovered as a novel putative PD risk locus with the aid of this database [171].

Alz Data is associated with Alzheimer's disease, the most common and quickly growing neurological illness. This database includes:(i) High throughput omic data, such as transcriptomes, proteomics, functional genomics, and genomics (GWAS and

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whole exome sequencing);(ii) High-confidence functional data, such as transgenic mice, population-based longitudinal studies, and neuroimaging screening phenotyping [172].

One of the prevalent mental illnesses with an 80% inheritance rate is schizophrenia [173].The SZDB database contains the results of genetic and molecular research on schizophrenia.SZDB 2.0 (www.szdb.org), a comprehensive database for schizophrenic research, was just released [174].Genomes Wide Association Study (GWAS), a polygenic risk score calculator, and genetic and gene expression data are all included in the updated version. research, copy number variations, transcript QTL, methylation QTL, gene expression Quantitative Trait Loci (eQTL), and information on protein-protein interactions [174].This database will undoubtedly offer a solid foundation for improving schizophrenia research.

Another database called BD Gene was created to investigate the genetic complexity of bipolar disorder (BD) and how it overlaps with major depressive disorder and schizophrenia MDD disorder [173].The researchers have free access to it.It offers a thorough analysis of the literature as well as information on high-confidence candidate genes and pathways for a deeper comprehension of the pathology of the illness [173].

BRAIN DISORDER STUDY MODELS

Numerous animal models are employed, including Caenorhabditis elegans, Drosophila melanogaster (fruitfully), Musca domestica (house fly), Danio rerio (zebra fish), pigs, and monkeys for comprehending the molecular mechanisms behind a variety of brain diseases and disorders [175].Additionally, cell lines are employed to investigate the molecular mechanisms behind the development of brain illnesses.Recent research has shown that the anatomy and organization of the human and monkey brains are remarkably similar.It is anticipated that it will greatly aid in comprehending the human brain illnesses or conditions [176].The type of biological questions that need to be addressed, however, determines which model is best [176].Because of the conserved nature of C. elegans, it has been utilized as a model organism to research several aspects of neurodegenerative illnesses such as PD [177], AD [178], and HD [179],C. elegans counterparts.Thanks to advancements in transgenic technology, Drosophila are now used as models for a variety of neurodegenerative diseases, including AD, taupathies, PD, different polyglutamine disorders, inherited spastic paraplegia, and amyotrophic sclerosis [180–182].Genes from zebra fish and their human counterparts have conserved roles in the genesis of neurodegenerative disorders, such as AD, HD, and PD [183].Zebrafish larvae exhibit measurable and human-like behavioral and neuropathological traits [184].To better understand the pathophysiology of autism, transgenic mice and rats have been created by genetic alteration techniques. Fragile X syndrome (FXS) and other neuropsychiatric conditions [185, 187].It is anticipated that the pig's cerebral cortex will have a high translational value since, in contrast to the cerebral cortex of mice or rats, it possesses cerebral convolution (gyri and sulci) similar to the human neocortex [188].Since the pig's cerebral cortex has cerebral convolution (gyri and sulci) similar to the human neocortex, it is expected to have a high translational value compared to the cerebral cortex of mice or rats [188].Because pigs' anatomy, physiology, and genomes are more comparable to those of humans than those of mice, genetically altered pigs are also being used to examine a variety of neurological conditions. [190].Transgenic monkeys have also been used to study neurological problems [191].Monkeys are favored for comprehending the mechanisms underlying the development of Parkinson's disease [191], microcephaly [192], Sleep problems [194] and AD [193].

Because brain neurons are incapable of regenerating, aging-related degeneration causes serious brain dysfunctions.Early on, neurodegenerative illnesses and disorders are characterized by a sluggish rate of progression.Elderly people are particularly affected by these illnesses in developed nations with long life expectancies [195].Parkinson's disease (PD), progressive supranuclear palsy (PSP), multi-system atrophy (MSA), Fronto-temporal dementia (FTD), dementia with Lewy bodies (DLB), and Alzheimer's disease (AD).Parkinson's disease (PD) is a progressive neurological disease that causes the body to become rigid and move slowly.Neuronal loss in the substantia nigra and other brain regions is one of its hallmarks.It is linked to the development of intracellular protein inclusions in neurons called Lewy bodies (LBs) [196, 197].Drugs can now be transferred across the blood-brain barrier thanks to nanotechnology.To get across the BBB, researchers are attempting to create liposomes that are filled with nanoparticles [198, 199].To find effective ways to help people with brain problems, more research is needed.One of the most potential uses of

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nanotechnology in clinical neuroscience is the delivery of medications across the blood-brain barrier.Drug distribution across the blood-brain barrier may benefit from nanoparticles' ability to perform several functions in a predetermined order [200–204].

II. CONCLUSION

Research has concentrated more on the study of epigenetic variables because non-genetic factors play a role in the development of human brain problems.In this regard, the GWAS data and the databases created for neurodegenerative illnesses have been a huge help in the region.Even if there are a number of models for studying neurodegenerative diseases, further specialized methods are still required, particularly for neuropsychiatric disorders as a result of symptom overlap.The BD gene has made an effort to address both the overlapping symptoms of bipolar disorder and its hereditary complexity. major depressive disorders (MDD) and schizophrenia.

REFERENCES

[1]. Nieuwenhuys R, Ten Donkelaar HJ, Nicholson C. The meaning of it [1] all.The central nervous system of vertebrates. Berlin, Heidelberg: Springer 1998; pp. 2135-95. [http://dx.doi.org/10.1007/978-3-642-18262-4_24]

[2]. Safi K, Seid MA, Dechmann DK. Bigger is not always better: when brains get smaller. Biol Lett 2005; 1(3): 283-6. [http://dx.doi.org/10.1098/rsbl.2005.0333] [PMID: 17148188]

[3]. Yuste R, Church GM. The new century of the brain. Sci Am 2014; [http://dx.doi.org/10.1038/scientificamerican0314-38] [PMID: 24660326]

[4]. Roth G, Dicke U. Evolution of the brain and intelligence. Trends CognSci (Regul Ed) 2005; 9(5): 250-7. [http://dx.doi.org/10.1016/j.tics.2005.03.005] [PMID: 15866152]

[5]. Jones EG, Mendell LM. Assessing the decade of the brain. Science 1999; 284(5415): 739. [http://dx.doi.org/10.1126/science.284.5415.739] [PMID: 10336393]

[6]. Striedter GF. Précis of principles of brain evolution. Behav Brain Sci 2006; 29(1): 1-12. [http://dx.doi.org/10.1017/S0140525X06009010] [PMID: 16542524]

[7]. Dhanwate AD. Brainstem death: A comprehensive review in Indian perspective. Indian J Crit Care Med peer-reviewed, official publication of Indian Society of Critical Care Medicine 2014; 18(9): 596. [http://dx.doi.org/10.4103/0972-5229.140151]

[8]. Butler AB. Chordate evolution and the origin of craniates: an old brain in a new head. Anat Rec 2000; 261(3): 111-25. [http://dx.doi.org/10.1002/1097-0185(20000615)261:33.0.CO;2-F] [PMID: 10867629]

[9]. Jarvis ED, Güntürkün O, Bruce L, et al. Avian Brain NomenclatureConsortium. Avian brains and a new understanding of vertebrate brain evolution. Nat Rev Neurosci 2005; 6(2): 151-9. [http://dx.doi.org/10.1038/nrn1606] [PMID: 15685220]

[10]. Chiel HJ, Beer RD. The brain has a body: Adaptive behavior emerges from interactions of nervous system, body and environment. Trends Neurosci 1997; 20(12): 553-7. [http://dx.doi.org/10.1016/S0166-2236(97)01149-1] [PMID: 9416664]

[11]. Glees P. The human brain. Cambridge University Press 2005.

[12]. Cosgrove KP, Mazure CM, Staley JK. Evolving knowledge of sex differences in brain structure, function, and chemistry. Biol Psychiatry 2007; 62(8): 847-55. [http://dx.doi.org/10.1016/j.biopsych.2007.03.001] [PMID: 17544382]

[13]. Frigeri T, Paglioli E, de Oliveira E, Rhoton AL Jr. Microsurgical anatomy of the central lobe. J Neurosurg 2015; 122(3): 483-98. [http://dx.doi.org/10.3171/2014.11.JNS14315] [PMID: 25555079]

[14]. Müller WE, Eckert GP, Scheuer K, Cairns NJ, Maras A, Gattaz WF. Effects of β-amyloid peptides on the fluidity of membranes from frontal and parietal lobes of human brain. High potencies of A β 1-42 and A β 1-43. Amyloid 1998; 5(1): 10-5. [http://dx.doi.org/10.3109/13506129809007284] [PMID: 9547000]

[15]. Cowell PE, Turetsky BI, Gur RC, Grossman RI, Shtasel DL, Gur RE. Sex differences in aging of the human frontal and temporal lobes. J Neurosci 1994; 14(8): 4748-55. [http://dx.doi.org/10.1523/JNEUROSCI.14-08-04748.1994] [PMID: 8046448]

[16]. Frackowiak RS. Human brain function. Elsevier 2004.

[17]. [Aitken JT, Bridger JE. Neuron size and neuron population density in the lumbosacral region of the cat's spinal cord. J Anat 1961; 95(1): 38-53. [PMID: 13681841]
[18]. Jäkel S, Dimou L. Glial cells and their function in the adult brain: A journey through the history of their ablation. Front Cell Neurosci 2017; 11: 24. [http://dx.doi.org/10.3389/fncel.2017.00024] [PMID: 28243193]
[19]. Yajima K, Suzuki K. Demyelination and remyelination in the rat central nervous system following ethidium bromide injection. Lab Invest 1979; 41(5): 385-92. [PMID: 502470]
[20]. Amat JA, Ishiguro H, Nakamura K, Norton WT. Phenotypic diversity and kinetics of proliferating microglia and astrocytes following cortical stab wounds. Glia 1996; 16(4): 368-82. [http://dx.doi.org/10.1002/(SICI)1098-1136(199604)16:43.0.CO;2-W] [PMID: 8721677]
[21]. Zheng D, Purves D. Effects of increased neural activity on brain growth. Proc Natl Acad Sci USA 1995; 92(6): 1802-6. [http://dx.doi.org/10.1073/pnas.92.6.1802] [PMID: 7892181]
[22]. Daneman R, Zhou L, Kebede AA, Barres BA. Pericytes are required for blood-brain barrier integrity during embryogenesis. Nature 2010; 468(7323): 562-6. [http://dx.doi.org/10.1038/nature09513] [PMID: 20944625]
[23]. Daneman R, Prat A. The blood-brain barrier. Cold Spring Harb PerspectBiol 2015; 7(1)a020412 [http://dx.doi.org/10.1101/cshperspect.a020412] [PMID: 25561720]
[24]. Tsuji A. Small molecular drug transfer across the blood-brain barrier via carrier-mediated transport systems. NeuroRx 2005; 2(1): 54-62. [http://dx.doi.org/10.1602/neurorx.2.1.54] [PMID: 15717057]
[25]. Ballabh P, Braun A, Nedergaard M. The blood-brain barrier: An overview: Structure, regulation, and clinical implications. Neurobiol Dis 2004; 16(1): 1-13. [http://dx.doi.org/10.1016/j.nbd.2003.12.016] [PMID: 15207256]
[26]. Clarke PGH, Oppenheim RW. Neuron death in vertebrate development: in vitro methods. Methods Cell Biol 1995; 46: 277-321. [http://dx.doi.org/10.1016/S0091-679X(08)61933-0] [PMID: 7609654]
[27]. Finch CE, Day JR. Molecular biology of aging in the nervous system: a synopsis of the levels of mechanisms. Neurodegener Dis 1994; 33-50.
[28]. Lancaster E. The diagnosis and treatment of autoimmune encephalitis. J Clin Neurol 2016; 12(1): 1-13. [http://dx.doi.org/10.3988/jcn.2016.12.1.1] [PMID: 26754777]
[29]. Venkatesan A. Epidemiology and outcomes of acute encephalitis. Curr Opin Neurol 2015; 28(3): 277-82. [http://dx.doi.org/10.1097/WCO.0000000000000199] [PMID: 25887770]
[30]. Levite M. Autoimmune epilepsy. Nat Immunol 2002; 3(6): 500-0. [http://dx.doi.org/10.1038/ni0602-500] [PMID: 12032558]
[31]. Britton J. Autoimmune epilepsy. Handb Clin Neurol 2016; 133: 219-45. [http://dx.doi.org/10.1016/B978-0-444-63432-0.00013-X] [PMID: 27112680]
[32]. Byram K, Hajj-Ali RA, Calabrese L. CNS vasculitis: An approach to differential diagnosis and management. Curr Rheumatol Rep 2018; 20(7): 37. [http://dx.doi.org/10.1007/s11926-018-0747-z] [PMID: 29846828]
[33]. John S, Hajj-Ali RA. CNS vasculitis. Semin Neurol 2014; 34(4): 405-12. [http://dx.doi.org/10.1055/s-0034-1390389] [PMID: 25369436]
[34]. Mocellin R, Walterfang M, Velakoulis D. Hashimoto's encephalopathy: Epidemiology, pathogenesis and management. CNS Drugs 2007; 21(10): 799-811. [http://dx.doi.org/10.2165/00023210-200721100-00002] [PMID: 17850170]
[35]. Pinedo-Torres I, Paz-Ibarra JL. Current knowledge on Hashimoto's encephalopathy: A literature review. Medwave 2018; 18(6): e7298. [http://dx.doi.org/10.5867/medwave.2018.06.7298] [PMID: 30451215]
[36]. Weinshenker BG, Wingerchuk DM. Neuromyelitis spectrum disorders. Mayo Clin Proc 2017; 92(4): 663-79. [http://dx.doi.org/10.1016/j.mayocp.2016.12.014] [PMID: 28385199]
[37]. Huda S, Whittam D, Bhojak M, Chamberlain J, Noonan C, Jacob A. Neuromyelitis optica spectrum disorders. Clin Med (Lond) 2019;

19(2): 169-76. [http://dx.doi.org/10.7861/clinmedicine.19-2-169] [PMID: 30872305]

[38]. Wilhelm H, Schabet M. The diagnosis and treatment of optic neuritis. DtschArztebl Int 2015; 112(37): 616-25. [http://dx.doi.org/10.3238/arztebl.2015.0616] [PMID: 26396053]

[39]. Jenkins TM, Toosy AT. Optic neuritis: The eye as a window to the brain. Curr Opin Neurol 2017; 30(1): 61-6. [http://dx.doi.org/10.1097/WCO.0000000000000414] [PMID: 27906756]

[40]. Burns TM. Neurosarcoidosis. Arch Neurol 2003; 60(8): 1166-8. [http://dx.doi.org/10.1001/archneur.60.8.1166] [PMID: 12925378]

[41]. Bargagli E, Prasse A. Sarcoidosis: A review for the internist. Intern Emerg Med 2018; 13(3): 325-31. [PMID: 29299831]

[42]. Ferreira BFA, Rodriguez EEC, Prado LLD, Gonçalves CR, Hirata CE, Yamamoto JH. Frosted branch angiitis and cerebral venous sinus thrombosis as an initial onset of neuro-Behçet's disease: A case report and review of the literature. J Med Case Reports 2017; 11(1): 104. [http://dx.doi.org/10.1186/s13256-017-1261-z] [PMID: 28410605]

[43]. Kalra S, Silman A, Akman-Demir G, et al. Diagnosis and management of Neuro-Behçet's disease: International consensus recommendations. J Neurol 2014; 261(9): 1662-76. [http://dx.doi.org/10.1007/s00415-013-7209-3] [PMID: 24366648]

[44]. Rahman A, Isenberg DA. Systemic lupus erythematosus. N Engl J Med 2008; 358(9): 929-39. [http://dx.doi.org/10.1056/NEJMra071297] [PMID: 18305268]

[45]. Bărbulescu AL, Sandu RE, Vreju AF, et al. Neuroinflammation in systemic lupus erythematosus - A review. Rom J MorpholEmbryol 2019; 60(3): 781-6. [PMID: 31912087]

[46]. Miller-Kuhaneck H, Watling R. Autism: A comprehensive occupational therapy approach. 2010.

[47]. Patel VB, Preedy VR, Martin CR, Eds. Comprehensive guide to autism. Springer New York 2014. [http://dx.doi.org/10.1007/978-1-4614-4788-7]

[48]. Finger EC. Frontotemporal Dementias Continuum (Minneap Minn). 2016; 22: pp. (2 Dementia)464-89. [http://dx.doi.org/10.1212/CON.0000000000000300]

[49]. Convery R, Mead S, Rohrer JD. Review: Clinical, genetic and neuroimaging features of frontotemporal dementia. Neuropathol Appl Neurobiol 2019; 45(1): 6-18. [http://dx.doi.org/10.1111/nan.12535] [PMID: 30582889]

[50]. Cummings JL. The Neuropsychiatric Inventory: Assessing psychopathology in dementia patients. Neurology 1997; 48(5)(Suppl. 6): S10-6. [http://dx.doi.org/10.1212/WNL.48.5_Suppl_6.10S] [PMID: 9153155]

[51]. Gomperts SN. Lewy body dementias: Dementia with Lewy bodies and Parkinson disease dementia. Continuum (Minneap Minn) 2016; 22(2 Dementia): 435-63. [http://dx.doi.org/10.1212/CON.0000000000000309] [PMID: 27042903]

[52]. Hachinski V. Vascular dementia: A radical redefinition. Dementia 1994; 5(3-4): 130-2. [PMID: 8087166]

[53]. Brust JC. Vascular dementia is overdiagnosed. Arch Neurol 1988; 45(7): 799-801. [http://dx.doi.org/10.1001/archneur.1988.00520310117026] [PMID: 3291834]

[54]. Mantzavinos V, Alexiou A. Biomarkers for Alzheimer's DiseaseDiagnosis. Curr Alzheimer Res 2017; 14(11): 1149-54. [http://dx.doi.org/10.2174/1567205014666170203125942] [PMID: 28164766]

[55]. Ballard C, Gauthier S, Corbett A, Brayne C, Aarsland D, Jones E. Alzheimer's disease. Lancet 2011; 377(9770): 1019-31. [http://dx.doi.org/10.1016/S0140-6736(10)61349-9] [PMID: 21371747]

[56]. Sakushima K, Hayashino Y, Kawaguchi T, Jackson JL, Fukuhara S.Diagnostic accuracy of cerebrospinal fluid lactate for differentiating bacterial meningitis from aseptic meningitis: A meta-analysis. J Infect 2011; 62(4): 255-62. [http://dx.doi.org/10.1016/j.jinf.2011.02.010] [PMID: 21382412]

[57]. Spach DH, Jackson LA. Bacterial meningitis. Neurol Clin 1999; 17(4): 711-35. [http://dx.doi.org/10.1016/S0733-8619(05)70163-8] [PMID: 10517925]

[58]. Ellul M, Solomon T. Acute encephalitis - diagnosis and management. Clin Med (Lond) 2018; 18(2): 155-9. [http://dx.doi.org/10.7861/clinmedicine.18-2-155] [PMID: 29626021]
[59]. Roos KL, Tyler KL. Meningitis, Encephalitis, Brain Abscess, and Empyema Harrison's Principles of Internal Medicine. 19th ed. New York, NY: McGraw-Hill Education 2015.
[60]. Brook I. Microbiology and treatment of brain abscess. J Clin Neurosci [60] 2017; 38: 8-12. [http://dx.doi.org/10.1016/j.jocn.2016.12.035] [PMID: 28089421]
[61]. Brouwer MC, Coutinho JM, van de Beek D. Clinical characteristics and outcome of brain abscess: Systematic review and meta-analysis. Neurology 2014; 82(9): 806-13. [http://dx.doi.org/10.1212/WNL.0000000000000172] [PMID: 24477107]
[62]. Jones TM, Shaw JD, Sullivan K, Zesiewicz TA. Treatment of cerebellar ataxia. Neurodegener Dis Manag 2014; 4(5): 379-92. [http://dx.doi.org/10.2217/nmt.14.27] [PMID: 25405651]
[63]. Morton SM, Bastian AJ. Can rehabilitation help ataxia? Neurology 2009; 73(22): 1818-9. [http://dx.doi.org/10.1212/WNL.0b013e3181c33b21] [PMID: 19864635]
[64]. Crowner BE. Cervical dystonia: Disease profile and clinical management. Phys Ther 2007; 87(11): 1511-26. [http://dx.doi.org/10.2522/ptj.20060272] [PMID: 17878433]
[65]. Shaikh AG, Zee DS, Crawford JD, Jinnah HA. Cervical dystonia: A neural integrator disorder. Brain 2016; 139(Pt 10): 2590-9. [http://dx.doi.org/10.1093/brain/aww141] [PMID: 27324878]
[66]. Bashir H, Jankovic J. Treatment options for chorea. Expert Rev Neurother 2018; 18(1): 51-63. [http://dx.doi.org/10.1080/14737175.2018.1403899] [PMID: 29120264]
[67]. Hermann A, Walker RH. Diagnosis and treatment of choreasyndromes. Curr Neurol Neurosci Rep 2015; 15(2): 514. [http://dx.doi.org/10.1007/s11910-014-0514-0] [PMID: 25620691]
[68]. Bates G, Tabrizi S, Jones L, Eds. Huntington's disease (No 64). Oxford Monographs on Medical G. 2014. Myers RH. Huntington's disease genetics. NeuroRx 2004; 1(2):
[69]. [69] 255-62. [http://dx.doi.org/10.1602/neurorx.1.2.255] [PMID: 15717026]
[70]. Wyant KJ, Ridder AJ, Dayalu P. Huntington's Disease-Update on Treatments. Curr Neurol Neurosci Rep 2017; 17(4): 33. [http://dx.doi.org/10.1007/s11910-017-0739-9] [PMID: 28324302]
[71]. Swan L, Dupont J. Multiple system atrophy. Phys Ther 1999; 79(5): 488-94. [http://dx.doi.org/10.1093/ptj/79.5.488] [PMID: 10331752]
[72]. Palma JA, Norcliffe-Kaufmann L, Kaufmann H. Diagnosis of multiple system atrophy. Auton Neurosci 2018; 211: 15-25. [http://dx.doi.org/10.1016/j.autneu.2017.10.007] [PMID: 29111419]
[73]. Lance JW. Action myoclonus, Ramsay Hunt syndrome, and other cerebellar myoclonic syndromes. Adv Neurol 1986; 43: 33-55. [PMID: 3080851]
[74]. Chokroverty S. Propriospinal myoclonus. Clin Neurosci 1995-1996; [74] 3(4): 219-22. [PMID: 8891395]
[75]. Shulman JM, De Jager PL, Feany MB. Parkinson's disease: Genetics and pathogenesis. Annu Rev Pathol 2011; 6: 193-222. [http://dx.doi.org/10.1146/annurev-pathol-011110-130242] [PMID: 21034221]
[76]. Hasnain M, Vieweg WV, Baron MS, Beatty-Brooks M, Fernandez A, Pandurangi AK. Pharmacological management of psychosis in elderly patients with parkinsonism. Am J Med 2009; 122(7): 614-22. [http://dx.doi.org/10.1016/j.amjmed.2009.01.025] [PMID: 19559160]
[77]. Armstrong MJ. Progressive Supranuclear Palsy: an Update. Curr Neurol Neurosci Rep 2018; 18(3): 12. [http://dx.doi.org/10.1007/s11910-018-0819-5]
[78]. Barsottini OG, Felício AC, Aquino CC, Pedroso JL. Progressive supranuclear palsy: New concepts. ArqNeuropsiquiatr 2010; 68(6): 938-46. [http://dx.doi.org/10.1590/S0004-282X2010000600020] [PMID: 21243256]
[79]. Japaridze G, Kasradze S, Maisuradze L, Popp R, Wetter T. The Restless Legs Syndrome. Georgian Med News 2018; (285): 74-81. [PMID: 30702074]
[80]. Venkateshiah SB, Ioachimescu OC. Restless legs syndrome. Crit Care Clin 2015; 31(3): 459-72.

[http://dx.doi.org/10.1016/j.ccc.2015.03.003] [PMID: 26118915]
[81]. Vijayakumar D, Jankovic J. Drug-Induced Dyskinesia, Part 2: Treatment of Tardive Dyskinesia. Drugs 2016; 76(7): 779-87. [http://dx.doi.org/10.1007/s40265-016-0568-1] [PMID: 27091214]
[82]. Fernandez HH, Friedman JH. Classification and treatment of tardive syndromes. Neurologist 2003; 9(1): 16-27. [http://dx.doi.org/10.1097/01.nrl.0000038585.58012.97] [PMID: 12801428]
[83]. Efron D, Dale RC. Tics and Tourette syndrome. J Paediatr Child Health 2018; 54(10): 1148-53. [http://dx.doi.org/10.1111/jpc.14165] [PMID: 30294996]
[84]. Leckman JF. Tourette's syndrome. Lancet 2002; 360(9345): 1577-86. [http://dx.doi.org/10.1016/S0140-6736(02)11526-1] [PMID: 12443611]
[85]. Ferenci P. Diagnosis of Wilson disease. Handb Clin Neurol 2017; 142: 171-80. [http://dx.doi.org/10.1016/B978-0-444-63625-6.00014-8] [PMID: 28433100]
[86]. Singh S, Behari M. Wilson's disease. J Assoc Physicians India 2003; 51: 183-90. [PMID: 12725264]
[87]. Hardiman O, Al-Chalabi A, Chio A, et al. Amyotrophic lateral sclerosis. Nat Rev Dis Primers 2017; 3: 17071. [http://dx.doi.org/10.1038/nrdp.2017.71] [PMID: 28980624]
[88]. Brown RH, Al-Chalabi A. Amyotrophic Lateral Sclerosis. N Engl J Med 2017; 377(2): 162-72. [http://dx.doi.org/10.1056/NEJMra1603471] [PMID: 28700839]
[89]. Ramchandren S. Charcot-Marie-Tooth Disease and Other Genetic Polyneuropathies. Continuum (Minneap Minn) (5,Peripheral Nerve and Motor Neuron Disorders) 2017; 23: 1360-77. [http://dx.doi.org/10.1212/CON.0000000000000529] [PMID: 28968366]
[90]. Sman AD, Hackett D, Fiatarone Singh M, Fornusek C, Menezes MP,Burns J. Systematic review of exercise for Charcot-Marie-Tooth disease. J PeripherNervSyst 2015; 20(4): 347-62. [http://dx.doi.org/10.1111/jns.12116] [PMID: 26010435]
[91]. Koch MW, Metz LM, Agrawal SM, Yong VW. Environmental factors and their regulation of immunity in multiple sclerosis. J Neurol Sci 2013; 324(1-2): 10-6. [http://dx.doi.org/10.1016/j.jns.2012.10.021] [PMID: 23154080]
[92]. Martinelli V. Trauma, stress and multiple sclerosis. Neurol Sci 2000; 21(4)(Suppl. 2): S849-52. [http://dx.doi.org/10.1007/s100720070024] [PMID: 11205361]
[93]. Carter JC, Sheehan DW, Prochoroff A, Birnkrant DJ. Muscular Dystrophies. Clin Chest Med 2018; 39(2): 377-89. [http://dx.doi.org/10.1016/j.ccm.2018.01.004] [PMID: 29779596]
[94]. Turner C, Hilton-Jones D. Myotonic dystrophy: diagnosis, management and new therapies. Curr Opin Neurol 2014; 27(5): 599-606. [http://dx.doi.org/10.1097/WCO.0000000000000128] [PMID: 25121518]
[95]. Gilhus NE. Myasthenia Gravis. N Engl J Med 2016; 375(26): 2570-81. [http://dx.doi.org/10.1056/NEJMra1602678] [PMID: 28029925]
[96]. Sieb JP. Myasthenia gravis: An update for the clinician. Clin Exp Immunol 2014; 175(3): 408-18. [http://dx.doi.org/10.1111/cei.12217] [PMID: 24117026]
[97]. Miller ML, Shefner JM, Callen J. Diagnosis and differential diagnosis of dermatomyositis and polymyositis in adults www. uptodate. com2017.
[98]. Bohan A, Peter JB. Polymyositis and dermatomyositis (second of two parts). N Engl J Med 1975; 292(8): 403-7. [http://dx.doi.org/10.1056/NEJM197502202920807] [PMID: 1089199]
[99]. Watson JC, Dyck PJ. Peripheral neuropathy: A practical approach to diagnosis and symptom management. Mayo Clin Proc 2015; 90(7): 940-51. [http://dx.doi.org/10.1016/j.mayocp.2015.05.004] [PMID: 26141332]
[100]. Hughes RAC. Peripheral neuropathy. BMJ 2002; 324(7335): 466-9. [http://dx.doi.org/10.1136/bmj.324.7335.466] [PMID: 11859051]
[101]. Dubowitz V. Ramblings in the history of spinal muscular atrophy. NeuromusculDisord 2009; 19(1): 69-73. [http://dx.doi.org/10.1016/j.nmd.2008.10.004] [PMID: 18951794]
[102]. Nicole S, Diaz CC, Frugier T, Melki J. Spinal muscular atrophy: Recent advances and

future prospects. Muscle Nerve 2002; 26(1): 4-13. [http://dx.doi.org/10.1002/mus.10110]
[103]. Abou-Khalil BW, Gallagher MJ, Macdonald RL. Epilepsies.Bradley's Neurology in Clinical Practice. 6th ed. Philadelphia, PA: Elsevier 2012; pp. 1583-633. [http://dx.doi.org/10.1016/B978-1-4377-0434-1.00092-X]
[104]. Fisher RS, Schachter SC. The postictal state: A neglected entity in the management of epilepsy. Epilepsy Behav 2000; 1(1): 52-9. [http://dx.doi.org/10.1006/ebeh.2000.0023] [PMID: 12609127]
[105]. Asadi-Pooya AA. Psychogenic nonepileptic seizures: A concise review. Neurol Sci 2017; 38(6): 935-40. [http://dx.doi.org/10.1007/s10072-017-2887-8] [PMID: 28275874]
[106]. De Clerck L, Nica A, Biraben A. Clinical aspects of seizures in the elderly. GeriatrPsycholNeuropsychiatrVieil 2019; 17(S1): 7-12. [http://dx.doi.org/10.1684/pnv.2019.0790] [PMID: 30916651]
[107]. Devinsky O, Schein A, Najjar S. Epilepsy associated with systemic autoimmune disorders. Epilepsy Curr 2013; 13(2): 62-8. [http://dx.doi.org/10.5698/1535-7597-13.2.62] [PMID: 23646005]
[108]. Brigo F, Trinka E, Lattanzi S, Bragazzi NL, Nardone R, Martini M. A brief history of typical absence seizures - Petit mal revisited. Epilepsy Behav 2018; 80: 346-53. [http://dx.doi.org/10.1016/j.yebeh.2018.01.007] [PMID: 29402631]
[109]. Pack AM. Epilepsy Overview and Revised Classification of Seizures and Epilepsies. Continuum (Minneap Minn) 2019; 25(2): 306-21. [http://dx.doi.org/10.1212/CON.0000000000000707] [PMID: 30921011]
[110]. Wakai A, McCabe A, Roberts I, Schierhout G. Mannitol for acute traumatic brain injury. Cochrane Database Syst Rev 2013; 8(8)CD001049 [http://dx.doi.org/10.1002/14651858.CD001049.pub5] [PMID: 23918314]
[111]. Burgess S, Abu-Laban RB, Slavik RS, Vu EN, Zed PJ. A systematic review of randomized controlled trials comparing hypertonic sodium solutions and mannitol for traumatic brain injury: Implications for emergency department management. Ann Pharmacother 2016; 50(4): 291-300. [http://dx.doi.org/10.1177/1060028016628893] [PMID: 26825644]
[112]. McNeill KA. Epidemiology of brain tumors. Neurol Clin 2016; 34(4): 981-98. [http://dx.doi.org/10.1016/j.ncl.2016.06.014] [PMID: 27720005]
[113]. Hadidchi S, Surento W, Lerner A, et al. Headache and brain tumor. Neuroimaging Clin N Am 2019; 29(2): 291-300. [http://dx.doi.org/10.1016/j.nic.2019.01.008] [PMID: 30926118]
[114]. Schnurr PP, Friedman MJ, Engel CC, et al. Cognitive behavioral therapy for posttraumatic stress disorder in women: A randomized controlled trial. JAMA 2007; 297(8): 820-30. [http://dx.doi.org/10.1001/jama.297.8.820] [PMID: 17327524]
[115]. Whitfield C. Psychiatric drugs as agents of Trauma. Int J Risk Saf Med 2010; 22(4): 195-207. [http://dx.doi.org/10.3233/JRS-2010-0508]
[116]. National Collaborating Centre for Mental Health (UK). PostTraumatic Stress Disorder: The management of PTSD in adults and children in primary and secondary care. UK: National Collaborating Centre for Mental Health 2005. [PMID: 21834189]
[117]. Jonas DE, Cusack K, Forneris CA, et al. Psychological and pharmacological treatments for adults with posttraumatic stress disorder (PTSD). AHRQ ComparEffectiv Rev 2013; p. 92. [PMID: 23658937] [http://dx.doi.org/10.1037/e553842013-001]
[118]. Tharoor H, Kumar TR. Psychotic disorders including schizo-phrenia.Emergencies in psychiatry in low-and middle-income countries. 2017; pp. 25-35.
[119]. Tsuang MT, Van Os J, Tandon R, et al. Attenuated psychosis syndrome in DSM-5. Schizophr Res 2013; 150(1): 31-5. [http://dx.doi.org/10.1016/j.schres.2013.05.004] [PMID: 23773295]
[120]. Rodrigues MF, Nardi AE, Levitan M. Mindfulness in mood and anxiety disorders: A review of the literature. Trends Psychiatry Psychother 2017; 39(3): 207-15. [http://dx.doi.org/10.1590/2237-6089-2016-0051] [PMID: 28767927]
[121]. Anthes E. Depression: A change of mind. Nature 2014; 515(7526): 185-7. [http://dx.doi.org/10.1038/515185a] [PMID: 25391944]

[122]. Uher R, Treasure J. Brain lesions and eating disorders. J Neurol Neurosurg Psychiatry 2005; 76(6): 852-7. [http://dx.doi.org/10.1136/jnnp.2004.048819] [PMID: 15897510]
[123]. Romanos GE, Javed F, Romanos EB, Williams RC. Oro-facial manifestations in patients with eating disorders. Appetite 2012; 59(2): 499-504. [http://dx.doi.org/10.1016/j.appet.2012.06.016] [PMID: 22750232]
[124]. Tyrer P, Reed GM, Crawford MJ. Classification, assessment, prevalence, and effect of personality disorder. Lancet 2015; 385(9969): 717-26. [http://dx.doi.org/10.1016/S0140-6736(14)61995-4] [PMID: 25706217]
[125]. Magnavita JJ. EdHandbook of personality disorders: Theory and practice. John Wiley and Sons 2004.
[126]. Başar E, Güntekin B. A review of brain oscillations in cognitive disorders and the role of neurotransmitters. Brain Res 2008; 1235: 172-93. [http://dx.doi.org/10.1016/j.brainres.2008.06.103] [PMID: 18640103]
[127]. Hardy JA, Higgins GA. Alzheimer's disease: The amyloid cascade hypothesis. Science 1992; 256(5054): 184-5. [http://dx.doi.org/10.1126/science.1566067] [PMID: 1566067]
[128]. Cipriani G, Dolciotti C, Picchi L, Bonuccelli U. Alzheimer and his disease: A brief history. Neurol Sci 2011; 32(2): 275-9. [http://dx.doi.org/10.1007/s10072-010-0454-7] [PMID: 21153601]
[129]. Jankovic J. Parkinson's disease: Clinical features and diagnosis. JNeurol Neurosurg Psychiatry 2008; 79(4): 368-76. [http://dx.doi.org/10.1136/jnnp.2007.131045] [PMID: 18344392]
[130]. Alzheimer's Association. Alzheimer's disease facts and figures. Alzheimers Dement 2017; 13(4): 325-73. [http://dx.doi.org/10.1016/j.jalz.2017.02.001]
[131]. Dubois B, Hampel H, Feldman HH, et al. Proceedings of the meetingof the International Working Group (IWG) and the American Alzheimer's Association on "The Preclinical State of AD"; July 23, 2015; Washington DC, USA. Preclinical Alzheimer's disease: Definition, natural history, and diagnostic criteria. Alzheimers Dement 2016; 12(3): 292-323. [http://dx.doi.org/10.1016/j.jalz.2016.02.002] [PMID: 27012484]
[132]. Jost BC, Grossberg GT. The evolution of psychiatric symptoms in Alzheimer's disease: A natural history study. J Am Geriatr Soc 1996; 44(9): 1078-81. [http://dx.doi.org/10.1111/j.1532-5415.1996.tb02942.x] [PMID: 8790235]
[133]. Calne DB, Langston JW. Aetiology of Parkinson's disease. Lancet 1983; 2(8365-66): 1457-9. [http://dx.doi.org/10.1016/S0140-6736(83)90802-4]
[134]. McCall B. Young-onset Parkinson's disease: A guide to care and support. Nurs Times 2003; 99(30): 28-31. [PMID: 12961940]
[135]. Sveinbjornsdottir S. The clinical symptoms of Parkinson's disease. J Neurochem 2016; 139(Suppl. 1): 318-24. [http://dx.doi.org/10.1111/jnc.13691] [PMID: 27401947]
[136]. Chesebro B. Introduction to the transmissible spongiform encephal-opathies or prion diseases. Br Med Bull 2003; 66: 1-20. [http://dx.doi.org/10.1093/bmb/66.1.1] [PMID: 14522845]
[137]. Goldmann W. PrP genetics in ruminant transmissible spongiform encephalopathies. Vet Res 2008; 39(4): 30. [http://dx.doi.org/10.1051/vetres:2008010] [PMID: 18284908]
[138]. Castle AR, Gill AC. Physiological functions of the cellular prion protein. Front Mol Biosci 2017; 4: 19. [http://dx.doi.org/10.3389/fmolb.2017.00019] [PMID: 28428956]
[139]. Brown P. The risk of bovine spongiform encephalopathy ('mad cow disease') to human health. JAMA 1997; 278(12): 1008-11. [http://dx.doi.org/10.1001/jama.1997.03550120068035] [PMID: 9307349]
[140]. Vonsattel JP, DiFiglia M. Huntington disease. J Neuropathol Exp Neurol 1998; 57(5): 369-84. [http://dx.doi.org/10.1097/00005072-199805000-00001] [PMID: 9596408]
[141]. Andrew SE, Goldberg YP, Kremer B, et al. Huntington disease without CAG expansion: phenocopies or errors in assignment? Am J Hum Genet 1994; 54(5): 852-63. [PMID: 8178825]
[142]. Brower MC, Price BH. Neuropsychiatry of frontal lobe dysfunction in violent and criminal behaviour: A critical review. J

Neurol Neurosurg Psychiatry 2001; 71(6): 720-6. [http://dx.doi.org/10.1136/jnnp.71.6.720] [PMID: 11723190]

[143]. Berrios GE, Marková IS. The concept of neuropsychiatry: A historical overview. J Psychosom Res 2002; 53(2): 629-38. [http://dx.doi.org/10.1016/S0022-3999(02)00427-0] [PMID: 12169337]

[144]. Kas MJ, Penninx B, Sommer B, Serretti A, Arango C, Marston H. Aquantitative approach to neuropsychiatry: The why and the how. NeurosciBiobehav Rev 2019; 97: 3-9. [http://dx.doi.org/10.1016/j.neubiorev.2017.12.008] [PMID: 29246661]

[145]. Autry AE, Monteggia LM. Brain-derived neurotrophic factor and neuropsychiatric disorders. Pharmacol Rev 2012; 64(2): 238-58. [http://dx.doi.org/10.1124/pr.111.005108] [PMID: 22407616]

[146]. Cummings JL, Mega MS. Neuropsychiatry and behavioral neuroscience. Oxford University Press 2003.

[147]. Rapoport J, Chavez A, Greenstein D, Addington A, Gogtay N. Autism spectrum disorders and childhood-onset schizophrenia: clinical and biological contributions to a relation revisited. J Am Acad Child Adolesc Psychiatry 2009; 48(1): 10-8. [http://dx.doi.org/10.1097/CHI.0b013e31818b1c63] [PMID: 19218893]

[148]. Cornblatt BA, Keilp JG. Impaired attention, genetics, and the pathophysiology of schizophrenia. Schizophr Bull 1994; 20(1): 31-46. [http://dx.doi.org/10.1093/schbul/20.1.31] [PMID: 8197420]

[149]. Uhl GR, Grow RW. The burden of complex genetics in brain disorders. Arch Gen Psychiatry 2004; 61(3): 223-9. [http://dx.doi.org/10.1001/archpsyc.61.3.223] [PMID: 14993109]

[150]. Cannon JR, Greenamyre JT. The role of environmental exposures in neurodegeneration and neurodegenerative diseases. Toxicol Sci 2011; 124(2): 225-50. [http://dx.doi.org/10.1093/toxsci/kfr239] [PMID: 21914720]

[151]. Hussain R, Zubair H, Pursell S, Shahab M. Neurodegenerative Diseases: Regenerative Mechanisms and Novel Therapeutic Approaches. Brain Sci 2018; 8(9): 177. [http://dx.doi.org/10.3390/brainsci8090177] [PMID: 30223579]

[152]. Prusiner SB. Shattuck lecture--neurodegenerative diseases and prions. N Engl J Med 2001; 344(20): 1516-26. [http://dx.doi.org/10.1056/NEJM200105173442006] [PMID: 11357156]

[153]. Goedert M. Alpha-synuclein and neurodegenerative diseases. Nat Rev Neurosci 2001; 2(7): 492-501. [http://dx.doi.org/10.1038/35081564] [PMID: 11433374]

[154]. Beal MF. Mitochondrial dysfunction in neurodegenerative diseases. BiochimBiophys Acta 1998; 1366(1-2): 211-23. [http://dx.doi.org/10.1016/S0005-2728(98)00114-5] [PMID: 9714810]

[155]. Taber KH, Hurley RA, Yudofsky SC. Diagnosis and treatment of neuropsychiatric disorders. Annu Rev Med 2010; 61: 121-33. [http://dx.doi.org/10.1146/annurev.med.051408.105018] [PMID: 19824816]

[156]. Vadakkan KI. Neurodegenerative disorders share common features of “loss of function” states of a proposed mechanism of nervous system functions. Biomed Pharmacother 2016; 83: 412-30. [http://dx.doi.org/10.1016/j.biopha.2016.06.042] [PMID: 27424323]

[157]. Hervás R, Oroz J, Galera-Prat A, et al. Common features at the start of the neurodegeneration cascade. PLoS Biol 2012; 10(5)e1001335 [http://dx.doi.org/10.1371/journal.pbio.1001335] [PMID: 22666178]

[158]. Brookmeyer R, Johnson E, Ziegler-Graham K, Arrighi HM.Forecasting the global burden of Alzheimer’s disease. Alzheimers Dement 2007; 3(3): 186-91. [http://dx.doi.org/10.1016/j.jalz.2007.04.381] [PMID: 19595937]

[159]. Datta G, Colasanti A, Rabiner EA, et al. Neuroinflammation and its relationship to changes in brain volume and white matter lesions in multiple sclerosis. Brain 2017; 140(11): 2927-38. [http://dx.doi.org/10.1093/brain/awx228] [PMID: 29053775]

[160]. Brown RC, Lockwood AH, Sonawane BR. Neurodegenerative diseases: An overview of environmental risk factors. Environ Health Perspect 2005; 113(9): 1250-6. [http://dx.doi.org/10.1289/ehp.7567] [PMID: 16140637]

[161]. Chin-Chan M, Navarro-Yepes J, Quintanilla-Vega B. Environmental pollutants as risk factors for neurodegenerative disorders: Alzheimer and Parkinson diseases. Front Cell Neurosci 2015; 9: 124. [http://dx.doi.org/10.3389/fncel.2015.00124] [PMID: 25914621]
[162]. Uher R. Gene-environment interactions in severe mental illness. Front Psychiatry 2014; 5: 48. [http://dx.doi.org/10.3389/fpsyt.2014.00048] [PMID: 24860514]
[163]. Faraone SV, Larsson H. Genetics of attention deficit hyperactivity disorder. Mol Psychiatry 2019; 24(4): 562-75. [http://dx.doi.org/10.1038/s41380-018-0070-0] [PMID: 29892054]
[164]. Franke B, Faraone SV, Asherson P, et al. International Multicentre persistent ADHD CollaboraTion. The genetics of attention deficit/hyperactivity disorder in adults, a review. Mol Psychiatry 2012; 17(10): 960-87. [http://dx.doi.org/10.1038/mp.2011.138] [PMID: 22105624]
[165]. Kozlowski H, Luczkowski M, Remelli M, Valensin D. Copper, zinc and iron in neurodegenerative diseases (Alzheimer's, Parkinson's and prion diseases). Coord Chem Rev 2012; 256(19-20): 2129-41. [http://dx.doi.org/10.1016/j.ccr.2012.03.013]
[166]. Barnham KJ, Bush AI. Metals in Alzheimer's and Parkinson's diseases. Curr Opin Chem Biol 2008; 12(2): 222-8. [http://dx.doi.org/10.1016/j.cbpa.2008.02.019] [PMID: 18342639]
[167]. Jokanović M. Neurotoxic effects of organophosphorus pesticides and possible association with neurodegenerative diseases in man: A review. Toxicology 2018; 410: 125-31. [http://dx.doi.org/10.1016/j.tox.2018.09.009] [PMID: 30266654]
[168]. Uversky VN. Neurotoxicant-induced animal models of Parkinson's disease: understanding the role of rotenone, maneb and paraquat in neurodegeneration. Cell Tissue Res 2004; 318(1): 225-41. [http://dx.doi.org/10.1007/s00441-004-0937-z] [PMID: 15258850]
[169]. Caudle WM, Guillot TS, Lazo CR, Miller GW. Industrial toxicants and Parkinson's disease. Neurotoxicology 2012; 33(2): 178-88. [http://dx.doi.org/10.1016/j.neuro.2012.01.010] [PMID: 22309908]
[170]. Gowthaman R, Gowthaman N, Rajangam MK, Srinivasan K. Database of neurodegenerative disorders. Bioinformation 2007; 2(4): 153-4. [http://dx.doi.org/10.6026/97320630002153] [PMID: 21670793]
[171]. Xie SX, Baek Y, Grossman M, et al. Building an integrated neurodegenerative disease database at an academic health center. Alzheimers Dement 2011; 7(4): e84-93. [http://dx.doi.org/10.1016/j.jalz.2010.08.233] [PMID: 21784346]
[172]. Kandale VV, Mujawar SN, Welasly PJ, et al. Development of integrated database of neurodegenerative diseases (IDND). Rev Res 2013; 2(9): 1-5.
[173]. Lill CM, Roehr JT, McQueen MB, et al. Comprehensive research synopsis and systematic meta-analyses in Parkinson's disease genetics: The PDGene database. PLoS Genet 2012; 8(3)e1002548 [http://dx.doi.org/10.1371/journal.pgen.1002548] [PMID: 22438815]
[174]. Zhang DF, Fan Y, Xu M, et al. Complement C7 is a novel risk gene for Alzheimer's disease in Han Chinese. Natl Sci Rev 2019; 6(2): 257-74. [http://dx.doi.org/10.1093/nsr/nwy127] [PMID: 31032141]
[175]. Chang SH, Gao L, Li Z, Zhang WN, Du Y, Wang J. BDgene: A genetic database for bipolar disorder and its overlap with schizophrenia and major depressive disorder. Biol Psychiatry 2013; 74(10): 727-33. [http://dx.doi.org/10.1016/j.biopsych.2013.04.016] [PMID: 23764453]
[176]. Wu Y, Li X, Liu J, Luo XJ, Yao YG. SZDB2.0: An updated comprehensive resource for schizophrenia research. Hum Genet 2020. [http://dx.doi.org/10.1007/s00439-020-02171-1] [PMID: 32385526]
[177]. Jennings CG, Landman R, Zhou Y, et al. Opportunities and challenges in modeling human brain disorders in transgenic primates. Nat Neurosci 2016; 19(9): 1123-30. [http://dx.doi.org/10.1038/nn.4362] [PMID: 27571191]
[178]. Buffalo EA, Movshon JA, Wurtz RH. From basic brain research to treating human brain disorders. Proc Natl Acad Sci USA 2019; 116(52): 26167-72.

[http://dx.doi.org/10.1073/pnas.1919895116] [PMID: 31871205]
[179]. Harrington AJ, Hamamichi S, Caldwell GA, et al. C. elegans as a model organism to investigate molecular pathways involved with Parkinson's disease. Dev Dyn 2010; 239(5): 1282-95.
[180]. Alexander AG, Marfil V, Li C. Use of Caenorhabditis elegans as a model to study Alzheimer's disease and other neurodegenerative diseases. Front Genet 2014; 5: 279. [http://dx.doi.org/10.3389/fgene.2014.00279] [PMID: 25250042]
[181]. Voisine C, Hart AC. Caenorhabditis elegans as a model system for triplet repeat diseases. Trinucleotide Repeat Protocols. Humana Press 2004; pp. 141-60. [http://dx.doi.org/10.1385/1-59259-804-8:141] [PMID: 15201454]
[182]. Cauchi RJ, van den Heuvel M. The fly as a model for neurodegenerative diseases: Is it worth the jump? Neurodegener Dis 2006; 3(6): 338-56. [http://dx.doi.org/10.1159/000097303] [PMID: 17192723]
[183]. Sang TK, Jackson GR. Drosophila models of neurodegenerative disease. NeuroRx 2005; 2(3): 438-46. [http://dx.doi.org/10.1602/neurorx.2.3.438] [PMID: 16389307]
[184]. Bilen J, Bonini NM. Drosophila as a model for human neurodegenerative disease. Annu Rev Genet 2005; 39: 153-71. [http://dx.doi.org/10.1146/annurev.genet.39.110304.095804] [PMID: 16285856]
[185]. Xi Y, Noble S, Ekker M. Modeling neurodegeneration in zebrafish. Curr Neurol Neurosci Rep 2011; 11(3): 274-82. [http://dx.doi.org/10.1007/s11910-011-0182-2] [PMID: 21271309]
[186]. Best JD, Alderton WK. Zebrafish: An in vivo model for the study of neurological diseases. Neuropsychiatr Dis Treat 2008; 4(3): 567-76. [http://dx.doi.org/10.2147/NDT.S2056] [PMID: 18830398]
[187]. Leung C, Jia Z. Mouse genetic models of human brain disorders. Front Gene 2016; 7(40) [http://dx.doi.org/10.3389/fgene.2016.00040]
[188]. Tayebati SK, Tomassoni D, Amenta F. Spontaneously hypertensive rat as a model of vascular brain disorder: Microanatomy, neurochemistry and behavior. J Neurol Sci 2012; 322(1-2): 241-9. [http://dx.doi.org/10.1016/j.jns.2012.05.047] [PMID: 22726353]
[189]. Lo Bianco C, Ridet JL, Schneider BL, Deglon N, Aebischer P. α Synucleinopathy and selective dopaminergic neuron loss in a rat lentiviral-based model of Parkinson's disease. Proc Natl Acad Sci USA 2002; 99(16): 10813-8.[http://dx.doi.org/10.1073/pnas.152339799] [PMID: 12122208]
[190]. Gieling ET, Schuurman T, Nordquist RE, et al. The pig as a model animal for studying cognition and neurobehavioral disorders.Mol Func Mod Neuropsych. Berlin, Heidelberg: Springer 2011; pp. 359-83. [http://dx.doi.org/10.1007/7854_2010_112] [PMID: 21287323]
[191]. Lind NM, Moustgaard A, Jelsing J, Vajta G, Cumming P, Hansen AK. The use of pigs in neuroscience: Modeling brain disorders. NeurosciBiobehav Rev 2007; 31(5): 728-51. [http://dx.doi.org/10.1016/j.neubiorev.2007.02.003] [PMID: 17445892]
[192]. Fan N, Lai L. Genetically modified pig models for human diseases. J Genet Genomics 2013; 40(2): 67-73. [http://dx.doi.org/10.1016/j.jgg.2012.07.014] [PMID: 23439405]
[193]. Cai DC, Wang Z, Bo T, et al. MECP2 duplication causes aberrant GABA pathways, circuits and behaviors in transgenic monkeys: Neural mappings to patients with autism. J Neurosci 2020; 40(19): 3799-814. [http://dx.doi.org/10.1523/JNEUROSCI.2727-19.2020] [PMID: 32269107]
[194]. Li X, Yang W, Li X, et al. Alpha-synuclein oligomerization and dopaminergic degeneration occur synchronously in the brain and colon of MPTP-intoxicated parkinsonian monkeys. Neurosci Lett 2020; 716: 134640.
[195]. Ke Q, Li W, Lai X, et al. TALEN-based generation of a cynomolgus monkey disease model for human microcephaly. Cell Res 2016; 26(9): 1048-61. [http://dx.doi.org/10.1038/cr.2016.93] [PMID: 27502025]
[196]. Elfenbein HA, Rosen RF, Stephens SL, et al. Cerebral ß-amyloid angiopathy in aged squirrel monkeys. HistoHistopath 2007. [http://dx.doi.org/10.14670/HH-22.155] [PMID: 17149688]

[197].]Barraud Q, Lambrecq V, Forni C, et al. Sleep disorders in Parkinson's disease: The contribution of the MPTP non-human primate model. Exp Neurol 2009; 219(2): 574-82. [http://dx.doi.org/10.1016/j.expneurol.2009.07.019] [PMID: 19635479]
[198]. Berg D. Biomarkers for the early detection of Parkinson's and Alzheimer's disease. Neurodegener Dis 2008; 5(3-4): 133-6. [http://dx.doi.org/10.1159/000113682] [PMID: 18322370]
[199]. Shulman JM, De Jager PL. Evidence for a common pathway linking neurodegenerative diseases. Nat Genet 2009; 41(12): 1261-2. [http://dx.doi.org/10.1038/ng1209-1261] [PMID: 19935760]
[200]. Kauwe JS, Cruchaga C, Mayo K, et al. Variation in MAPT is associated with cerebrospinal fluid tau levels in the presence of amyloid-beta deposition. Proc Natl Acad Sci USA 2008; 105(23): 8050-4. [http://dx.doi.org/10.1073/pnas.0801227105] [PMID: 18541914]
[201]. Silva GA. Nanotechnology approaches to crossing the blood-brain barrier and drug delivery to the CNS. BMC Neurosci 2008; 9(Suppl. 3): S4. [http://dx.doi.org/10.1186/1471-2202-9-S3-S4] [PMID: 19091001]
[202]. Krol S, Macrez R, Docagne F, et al. Therapeutic benefits from nanoparticles: the potential significance of nanoscience in diseases with compromise to the blood brain barrier. Chem Rev 2013; 113(3): 1877-903. [http://dx.doi.org/10.1021/cr200472g] [PMID: 23157552]
[203]. Brigger I, Morizet J, Aubert G, et al. Poly(ethylene glycol)-coated hexadecylcyanoacrylate nanospheres display a combined effect for brain tumor targeting. J Pharmacol Exp Ther 2002; 303(3): 928-36. [http://dx.doi.org/10.1124/jpet.102.039669] [PMID: 12438511]
[204]. Kreuter J. Drug delivery to the central nervous system by polymeric nanoparticles: What do we know? Adv Drug Deliv Rev 2014; 71: 2-14. [http://dx.doi.org/10.1016/j.addr.2013.08.008] [PMID: 23981489