**Interpreting Health Trends: Epidemiology's Contributions to Understanding Diseases**

**Sushant Wankhade1, Isha Wankhade1, Gurukiran P.2, Gurdeep Rattu3**

**1** Department of Biotechnology, Reva University, Rukmini Knowledge Park, Srinivasa Nagar, Bengaluru, Karnataka 560064, India

2 National Horticulture Research and Development Foundation (NHRDF), Nashik-Aurangabad Road, Nashik-422003, Maharashtra, India

**Authors email:**

[**wankhadesushant5@gmail.com**](mailto:wankhadesushant5@gmail.com)

[**isha27700@gmail.com**](mailto:isha27700@gmail.com)

[**gurukiranpjadav@gmail.com**](mailto:gurukiranpjadav@gmail.com)

[**gurdeep.r147@gmail.com**](mailto:gurdeep.r147@gmail.com) **(Corresponding author)**

Editor Id **“IIPER1680036292"**

**Abstract**

**This abstract provides an overview of the epidemiological trends and patterns of three distinct but significant diseases: Respiratory tract infections such as common cold, sinus infections, tonsils and laryngitis, Neurological diseases like Alzheimer disease and Parkinson’s disease, and endocrine diseases like Diabetes mellitus and Osteoporosis. Understanding the prevalence, risk factors, and distribution of these conditions is crucial for informing public health strategies and clinical interventions. The term "respiratory tract infections" refers to a wide variety of diseases, from minor colds to serious pneumonia. Globally, these infections are an important cause of sickness and death. Immunocompromised people, children, and the elderly are particularly at risk. Neurodegenerative conditions like Alzheimer's and Parkinson's disease primarily impact the elderly. The prevalence of Alzheimer's disease has been rising rapidly, and more people throughout the world are becoming affected. Age, genetics, and lifestyle choices all have significant consequences on how it develops.** **The prevalence of Parkinson's disease, which is marked by motor dysfunction, is also rising.** **Type 1 and type 2 diabetes create serious problems for the general population's health. Because of variables like obesity, sedentary lifestyles, and genetic susceptibility, the prevalence of diabetes has increased over the past few decades. Osteoporosis, which is characterized by decreased bone density and an elevated risk of fracture, primarily affects the elderly, especially postmenopausal women. Its widespread prevalence around the world raises serious public health issues.**

**Keywords:** Epidemiology, Disease, Pathogenicity, Symptoms, Prevention

1. **Introduction**

The study of the causes, symptoms, and distribution of health and disease in a given population is known as epidemiology. The word "epidemiology" derived from the Greek word "epi," which meaning "upon" or "among," as well as "demos," which means "people. “In essence, epidemiology is the study of how illnesses affect populations and spread, with the ultimate goal of enhancing community health and wellbeing. Epidemiologists study the occurrence of illnesses and other health-related occurrences in an effort to better understand the intricate interactions between a variety of variables, including genetics, environment, behaviour, and socioeconomic determinants.

The Spanish physician Joaqun de Villalba coined the term "epidemiology" in his 1802 book Epidemiologa Espaola to refer to the study of epidemics. The epidemiologic triad (also known as the epidemiologic triangle) is another often used model that looks at the interaction between host, agent, and environment factors in determining the likelihood of disease. The host is either the disease's actual or potential victim or recipient. When organisms reproduce in host tissue, it is called an infection and it can lead to illness. A person who is having infectious organisms while being asymptomatic is known as a carrier. The spreading of an organism across its surroundings is called dissemination.

**Types**: -

* Descriptive epidemiology
* Analytical epidemiology
* Experimental epidemiology

The three main methods used in epidemiology are experimental, analytical, and descriptive. Although all three can be used to look into the spread of disease, descriptive epidemiology is the approach that is most frequently employed, apart from all these three there is one another type called as theoretical epidemiology.

Following a description of a disease's basic epidemiology, specific analytical techniques can be utilized to learn more about the condition, and a particular experimental strategy can be constructed to test a hypothesis.

Diseases: -

* Respiratory Infection
* Neurological Diseases: - Parkinson and Alzheimer's
* Endocrine: Diabetes mellitus, Osteoporosis

1. **Types of Respiratory tract infections**

Respiratory tract infections are of types:

1) Upper respiratory tract infections

2) Lower Respiratory tract infections

* 1. ***Upper respiratory Tract infections***

These infections are those which infect the nose, Oesophagus, and airways and may result in various symptoms such as chest pain, nausea, dysphagia, odynophagia, vomiting, fever, and bleeding.

Bowel infections, also known as gastrointestinal infections (gastroenteritis), are brought on by microorganisms (sometimes known as "bugs" or germs") in the gut. You can contract a bowel illness by ingesting tainted food or water or by coming into contact with an infected individual. (19) They may last up to 48 to 72 hours.

Upper respiratory tract infections include the following:

* common colds
* sinus infections
* tonsillitis
* laryngitis

Common cold: - it is a viral infection of the nose and throat and windpipe. Cold spreads easily in homes classrooms and in workplaces. There is no cure for common cold it goes away within 10 days More than 200 viruses can cause cold. (20)

Sinus infections: - These infections cause when fluid builds up in a air filled pocket in the face called sinuses the fluid sinuses allow germs to grow. viruses as well as bacteria cause such sinus infection. The symptoms of sinus infection include runny nose post nasal drainage blocked or stuffy nose swelling and pressure around your eye’s cheeks nose or forehead. (20)

Laryngitis: - The Omicron variant is prone to cause severe laryngitis. The vocal cords open and close smoothly, forming sounds through vibration and movement. Laryngitis is a swelling or inflammation of the voice box from overuse, infection or irritation. (20).

* 1. ***Lower respiratory Tract Infections (LRTI)***

LRTI is caused by various types of microorganisms which include bacteria, fungi, and viruses. They may last up to 1 week-2 weeks. Lower respiratory tract infections can cause a variety of different gastro symptoms, depending on how bad the infection is. (21)

Common cold symptoms can also occur in less serious infections, such as:

* a congested or runny nose
* dried-up cough
* minimal fever
* minor throat discomfort
* chronic headache

Symptoms of more serious illnesses can include: fever, difficulty breathing, a blue color to the skin, a strong cough that may produce mucus, and rapid breathing wheezing and chest pain (21)

Lower respiratory tract infections include:

* bronchitis
* pneumonia
* bronchiolitis
* tuberculosis
  1. ***Prevention***

Keeping hydrated and taking medicines are also recommended for stomach infections. Penicillin, cephalosporin, antifolate/sulfa combos, nitroimidazole, penmen, glycopeptide, and monobactam antibiotics are frequently used to treat gastrointestinal infections.

With soap and water, thoroughly wash your hands.

* Practice food safety, including washing fruits and vegetables and preparing meats, and do not replace hand washing with alcohol-based hand sanitizer.
* When ill, avoid preparing food or cooking for others, clean hard surfaces with bleach, and carefully wash any clothing or linens.
* Before, during, and after preparing meals, wash your hands and your work surfaces.
* separating cooked food from raw meats, fish, poultry, and eggs
* Avoiding undercooked food and heating food until it reaches a safe internal temperature
* Within two hours of cooking, place food in the refrigerator below 40°F (4°C).
* Avoid unpasteurized dairy products, unpasteurized juices, and undercooked or raw animal products (22).

1. **Neurological Diseases**
   1. ***Parkinson's disease***
      1. ***Introduction to Parkinson's disease***

Parkinson's disease (PD) is a chronic and developing neurological condition. Clinical examination indicates a resting tremor, bradykinesia, and rigidity; pathologic investigation demonstrates preferential degeneration of dopaminergic neurons in the substantia nigra pars compacta as well as the presence of cytoplasmic inclusions known as Lewy bodies. The disorders collectively referred to as Parkinson's disease and similar syndromes must be distinguished from PD. These conditions, which are very uncommon, share stiffness and bradykinesia, two symptoms of Parkinson's disease. The Parkinson-plus syndromes, on the other hand, are incurable and do not respond to medical intervention.

In developed countries, the prevalence of Parkinson's disease is estimated to be 0.3% of the total population and 1% of those over the age of 60. Parkinson's disease is obviously a disorder associated with ageing. According to studies, the prevalence of Parkinson's disease rises until the ninth decade (ages 80 to 89) of life. There is no reliable data on the ubiquity of Parkinson's disease after the ninth decade. Several studies have been conducted. Men were shown to have a greater incidence of Parkinson's disease than women despite the fact that additional research have disputed this finding.

Classification: -

Parkinson's disease is the most constantly type of parkinsonism, often known as idiopathic parkinsonism because its cause is unknown. Because of the accumulation of the misfolded protein alpha-synuclein in the brain and its spread throughout the brain, Parkinson's disease is categorised as a synucleinopathy and more specifically as an alpha-synucleinopathy.

Similar movement symptoms may also be present in other Parkinson-plus syndromes, along with a variety of additional symptoms. Some of them are synucleinopathies as well. Lewy body dementia is characterised by motor indications that precede cognitive deterioration and hallucinations. Alternately, multiple systems atrophy (MSA) has an early onset of autonomic dysfunction (like orthostasis) and may predominate in the cerebellum, the brainstem, or the Parkinson's illness.

Tau, rather than alpha-synuclein, is involved in several Parkinson-plus disorders. PSP and CBS (progressive supranuclear palsy and corticobasal syndrome, respectively) are two examples. Rigidity, early falls, bulbar symptoms, and vertical gaze restriction are features of PSP, it has been linked to Pick's disease or frontal lobe dementia symptoms. CBS is characterised by asymmetric parkinsonism, dystonia, alien limb, and myoclonic jerks.[35] certain timings of presentation and related symptoms can help distinguish certain movement disorders from idiopathic Parkinson illness

* + 1. ***Neuropathy and pathophysiology***

Parkinson's disease is primarily characterized by cell death in the brain's basal ganglia, which can eventually impact up to 70% of dopamine-secreting neurons in the substantia nigra pars compacta [1]. Parkinson's illness causes alpha-synuclein to misfold and cluster with other alpha-synuclein. Because cells can't get rid of these aggregates, alpha-synuclein turns into cytotoxic and damages cells [2, 3].

Under a microscope, these clumps, also known as Lewy bodies, may be seen in neurons. In the substantia nigra, the death of hepatocytes (star-shaped glial cells) and an increase in microglia (another kind of glial cell) numbers occur after the loss of neurons [4]. The severity of the progression of the areas of the brain affected by Parkinson's disease can be evaluated using Braak staging. This staging suggests that Parkinson's disease starts in the medulla and olfactory bulb, moves to the substantia nigra pars compacta, and then affects the rest of the midbrain and basal forebrain. Movement symptoms develop as the disease starts to affect the substantia nigra pars compacta.[5]

The basal ganglia are connected to other parts of the brain through five main pathways. Among these are the limbic, orbitofrontal, associative, motor, and oculomotor circuits. The names identify each circuit's main projection zone. All are impacted by Parkinson's disease, and their disturbance produces locomotion, attention, and learning-related symptoms.[6] The motor circuit has received the greatest scientific attention.[6]

A particular motor circuit conceptual model and its modification with PD have an effect, however some limitations have been identified, leading to changes.[6] According to this paradigm, the basal nuclei ordinarily have a continual repressive influence on a variety of motor systems, Preventing them from triggering at inappropriate times. When a decision is made to undertake a specific action, inhibition for the relevant motor system is lowered, allowing it to be activated. Because dopamine promotes the release of inhibition, high levels of dopamine function tend to enhance motor activity, whereas low levels of dopamine function, as observed in Parkinson's disease, need more effort for each given movement.[6] Hypokinesia, or a general decrease in motor output, is caused by dopamine insufficiency.[6] On the other hand, drugs used to treat Parkinson's disease may generate excessive dopamine activity, allowing motor systems to be stimulated at inappropriate times and resulting in dyskinesias.[6]

Death of brain cells: One mechanism causing brain cell death is the abnormal accumulation of the protein alpha-synuclein coupled to ubiquitin in wounded cells. This insoluble protein forms inclusions known as Lewy bodies inside neurons.[7][8] These bodies begin to form in the olfactory bulb, medulla oblongata, and pontine tegmentum; at this point, people may be asymptomatic or have early nonmotor symptoms (such as loss of smell, difficulty sleeping, or automatic dysfunction). As the disease progresses, Lewy bodies grow in the substantia nigra, sections of the midbrain and basal forebrain, and finally the neocortex.[7] Whereas these brain regions are the main sites of neuronal degeneration in Parkinson's disease, Lewy bodies could protect cells from death (by sequestering or walling off the aberrant protein). Other types of alpha-synuclein (for example, oligomers) that do not aggregate into Lewy bodies and neurites can be dangerous.[9][8] The presence of Lewy bodies in cortical areas is typical in dementia patients. Unless a person has dementia, Alzheimer's disease's hallmarks, neurofibrillary tangles and senile plaques, are infrequent.[10]

* + 1. ***The neuroimmune connection***

The neuroimmune interaction is strongly implicated in Parkinson's disease aetiology. Parkinson's disease and autoimmune illnesses share genetic variants and biological mechanisms. According to one study, several autoimmune illnesses may raise one's risk of acquiring Parkinson's disease by up to 33%. PD is associated with autoimmune disorders due to protein formulation patterns of monocytes and CD4+ T cells. Herpes virus infections can trigger autoimmune responses to alpha-synuclein, presumably via viral protein molecular mimicry. Alpha-synuclein, as well as its aggregate form, Lewy bodies, can bind to microglia. As a result of alpha-synuclein binding to MHC receptors on inflammasomes, microglia can increase and become too active, leading in the release of proinflammatory cytokines such as IL-1β, IFNγ, and TNFα. Microglia stimulation influences astrocyte activation, changing their protective phenotype into a neurotoxic one. In healthy brains, astrocytes preserve neuronal connections. Astrocytes in Parkinson's disease patients are unable to protect dopaminergic synapses in the striatum. Antigens are delivered to T lymphocytes by microglia via MHC-I and MHC-II. This procedure stimulates CD4+ T lymphocytes, allowing them to cross the blood-brain barrier (BBB) and generate more proinflammatory cytokines like IFN, TNF, and IL-1. BBB failure in Parkinson's disease has been linked to mast cell degranulation and subsequent proinflammatory cytokine release. Another immune cell implicated with Parkinson's disease is peripheral monocytes, which have been seen in the substantia nigra of patients with the condition.

These white cells may contribute to further dopaminergic link disruption. Furthermore, monocytes derived from Parkinson's disease patients express larger quantities of the PD-associated protein, LRRK2, as compared to non-PD persons via vasodilation. Furthermore, Pro-inflammatory cytokines, such as IL-6, can stimulate the liver to create C-reactive protein, another protein commonly found in Parkinson's disease patients, which can contribute to an increase in peripheral inflammation. Peripheral inflammation can have an impact on the gut-brain axis, which has been linked to Parkinson's disease. Years before motor impairments develop, Patients with Parkinson's disease have changed gut flora and gastrointestinal issues.

Alpha-synuclein is formed in the gut and can travel to the brainstem and subsequently to the substantia nigra via the vagus nerve. Furthermore, elevated levels of alpha-synuclein and an increase in motor symptoms in Parkinson's disease patients have been connected to the bacteria Proteus mirabilis. A medical citation is required to better understand the pathological progression of Parkinson's disease, more research into the function of alpha-synuclein, inflammation, the axis of the gut-brain, and individual variability in immunological stress responses is needed.

* + 1. ***Signs And Symptoms***

Parkinson's disease is a neurodegenerative disorder that primarily affects movement. It is characterized by a wide range of motor and non-motor symptoms. Please note that the presentation of symptoms can vary from person to person, and not everyone with Parkinson's disease will experience all of these symptoms. Here are some common signs and symptoms of Parkinson's disease:

Motor Symptoms:

1. Tremors: Typically, a resting tremor, most commonly seen in the hands, fingers, or chin. It usually disappears during purposeful movement.

2. Bradykinesia: Slowness of movement, which can lead to difficulties in initiating and completing movements. This can affect various activities like walking, writing, or buttoning a shirt.

3. Rigidity: Stiffness of muscles, which can result in reduced range of motion and difficulty with movements.

4. Postural Instability: Difficulty maintaining balance and an increased risk of falls due to impaired reflexes and posture control.

5. Gait Disturbances: Shuffling steps, reduced arm swing, and a tendency to take small steps, often leading to a characteristic "festinating" gait.

6. Freezing of Gait: A sudden, temporary inability to move the feet, often while attempting to initiate walking or change direction.

7. Micrographia: Small, cramped handwriting that develops due to difficulties in fine motor control.

Non-Motor Symptoms:

1. Loss of Smell: An early and common symptom of Parkinson's disease is a decreased sense of smell (anosmia).

2. Sleep Disturbances: This can include insomnia, excessive daytime sleepiness, restless leg syndrome, and vivid dreams or nightmares.

3. Depression and Anxiety: Many people with Parkinson's experience mood disorders such as depression and anxiety.

4. Cognitive Changes: In later stages of the disease, this can range from mild cognitive impairment to more severe dementia.

5. Speech Changes: Slurred speech, monotone voice, and reduced facial expressions (masked face) are common.

6. Constipation: Gastrointestinal symptoms like constipation are often reported.

7. Urinary Problems: Issues with urinary urgency, frequency, or incontinence can arise.

8. Orthostatic Hypotension: A drop in blood pressure upon standing, leading to dizziness or fainting.

9. Sexual Dysfunction: Parkinson's can lead to decreased libido and other sexual issues.

10. Swallowing Difficulties: Problems with swallowing (dysphagia) can occur, increasing the risk of choking or aspiration pneumonia.

While these symptoms are usually associated with Parkinson's disease, they can also be present in other illnesses. If you or someone you know is experiencing these symptoms, it's best to see a doctor for an accurate evaluation and diagnosis. Individuals with Parkinson's disease can benefit from early diagnosis and effective care.

* + 1. ***Diagnosis***

Diagnosing Parkinson's disease involves a comprehensive assessment that considers both clinical symptoms and medical history. There is no single definitive test for Parkinson's disease; instead, a combination of evaluations is used to reach a diagnosis. The process typically involves:

1. A doctor will study the patient's medical history and perform a complete physical examination to assess the existence and progression of motor and non-motor symptoms associated with Parkinson's disease.

2. Neurological Examination: A neurologist will examine the patient's muscle tone, reflexes, coordination, gait, and other motor functions to identify characteristic signs of Parkinson's, such as Tremors, bradykinesia, discomfort, and postural instability are all symptoms.

3. Elimination of Other Causes: The doctor will rule out other conditions that may mimic Parkinson's disease, such as essential tremor, multiple system atrophy, or drug-induced parkinsonism.

4. Response to Dopaminergic Medication: A positive response to dopaminergic medication (levodopa) can support the diagnosis of Parkinson's disease. Improvement in motor symptoms after taking this medication can help differentiate Parkinson's from other movement disorders.

5. Imaging Studies: While not necessary for diagnosis, imaging tests like brain MRI or CT scans may be performed to rule out other structural causes of symptoms. DaTscan is a specialized imaging test that can assess dopamine transporter levels in the brain, providing supportive evidence for Parkinson's disease.

6. Clinical Criteria: The diagnosis of Parkinson's disease is often based on specific clinical criteria, such as the UK Brain Bank Criteria or the Movement Disorder Society (MDS) Clinical Diagnostic Criteria. These criteria take into account the presence and progression of various motor and non-motor symptoms.

7. Neurologist Consultation: A neurologist who specialises in movement disorders is frequently involved in confirming the diagnosis and distinguishing Parkinson's disease from other illnesses that are similar.

8. Follow-up and Monitoring: Parkinson's disease is a progressive disease with changing symptoms. Follow-up meetings with a neurologist are required on a regular basis to assess symptoms, adjust treatment, and handle any consequences.

It is essential to remember that diagnosing Parkinson's disease can be complex, and the process may take some time. Additionally, early and accurate diagnosis is crucial for starting appropriate treatment and management strategies to improve the quality of life for those with Parkinson's. If you suspect you or someone you know may have Parkinson's disease, it's recommended to seek evaluation and guidance from a medical professional, preferably A neurologist with expertise in movement problems. [11]

* + 1. ***Causes***

The specific cause of Parkinson's disease is unknown; however, it is thought to be a combination of hereditary, environmental, and potentially other factors. Some of the elements hypothesised to contribute to the development of Parkinson's disease are as follows:

1. Genetics: While most cases of Parkinson's disease are not directly inherited, the condition does have a hereditary component. Certain gene mutations, such as those found in the LRRK2, PARKIN, and PINK1 genes, have been linked to an elevated risk of Parkinson's disease development. However, these mutations are uncommon and account for only a small proportion of instances.

2. Environmental Toxins and compounds: Certain environmental toxins and compounds have been linked to an increased risk of Parkinson's disease.

These include:

• Pesticides and Herbicides: Some studies suggest a link between Certain pesticides are linked to an increased risk of Parkinson's disease.

• Industrial Chemicals: Exposure to industrial chemicals like solvents and heavy metals may also be linked to an increased risk.

• Manganese: Prolonged exposure to high levels of manganese, often seen in certain occupations, has been linked to a Parkinson's-like syndrome.

3. Oxidative Stress: Oxidative stress develops when there is an imbalance between the body's ability to neutralise harmful free radicals with antioxidants and the body's ability to produce them. Oxidative stress can harm cells, including neurons, and is thought to play a role in Parkinson's disease development and progression.

4. Mitochondrial Dysfunction: Mitochondria are the structures within cells that provide energy. Mitochondrial dysfunction can result in lower energy production and increased oxidative stress, which may contribute to the degeneration of neurons that produce dopamine in Parkinson's disease.

5. Alpha-Synuclein Aggregation: In Parkinson's disease, there is an accumulation of abnormal protein aggregates, particularly alpha-synuclein, within neurons. These aggregates are a hallmark of the disease and are believed to contribute to neuronal dysfunction and death.

6. Neuroinflammation: Neuroinflammation, or chronic inflammation in the brain, is suspected to play a role in the course of Parkinson's disease. Immune system reactions in the brain can contribute to neurodegeneration.

7. Gut-Brain Axis: Emerging research suggests a potential link between the gut and the brain in Parkinson's disease. Changes in the gut microbiome and inflammation in the digestive tract may influence the development and course of the condition.

8. Age and Genetics: The greatest significant risk factor for Parkinson's disease is getting older. As people get older, the condition becomes more prevalent. A family history of Parkinson's disease or similar neurological diseases may increase the risk even further.

It's interesting that while these factors are believed to contribute to Parkinson's disease, the exact interplay between genetics, environment, and other factors is still an active area of research. Parkinson's disease is a complex condition, and multiple factors likely contribute to its onset and progression.

* 1. ***Alzheimer***

Alzheimer's disease (pronounced "alz-HAI-mirs") is a progressive brain ailment characterised by a decline in memory, thinking, learning, and organising ability. It gradually diminishes a person's ability to do basic daily duties. Alzheimer's disease (AD) is the most common cause of dementia.

Alzheimer's symptoms deteriorate over time. Researchers believe that the sickness process may begin 10 years or more before the first symptoms appear. Adults over the age of 65 are most commonly affected with Alzheimer's disease.

Stages: Alzheimer's disease organisations and healthcare practitioners characterise Alzheimer's disease stages based on symptoms using a range of terms. While the nomenclature varies, the stages all follow the same pattern: Alzheimer's symptoms worsen over time. However, no two people experience AD in the same way. Each person suffering from Alzheimer's disease will go through the stages at their own rate. Not all changes will occur in every person. Because stages often overlap, it can be difficult for physicians to place someone with Alzheimer's disease in a certain stage.Some organisations and providers define Alzheimer's disease phases as dementia:

• Alzheimer's disease in its early stages.

• Alzheimer's disease-related mild cognitive impairment (MCI)

• Alzheimer's disease-related mild dementia

• Alzheimer's disease-related moderate dementia

• Alzheimer's disease-related severe dementia.

Other organizations and providers more broadly explain the stages as:

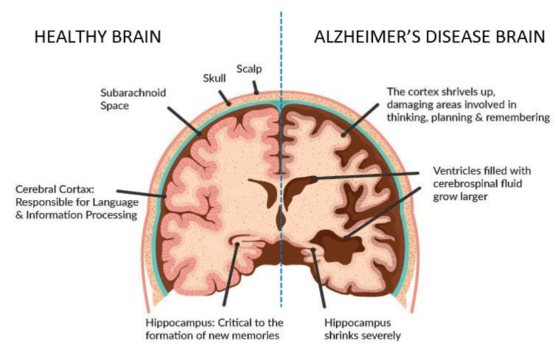
• Mild.

• Moderate.

• Severe.

In Alzheimer's disease, the brain suffers major structural changes compared to a healthy brain, including considerable atrophy and shrinkage, particularly in regions responsible for memory and higher cognitive functions such as the hippocampus and cortex as shown in Fig. 1. Normal brain structure and function are disrupted by abnormal protein accumulations such as beta-amyloid plaques and tau tangles. White matter alterations and ventricular hypertrophy may also occur. These structural changes lead to the disease's cognitive deterioration and memory impairment.

The cortex region of the brain, which is responsible for higher cognitive skills, is harmed by the accumulation of unusual protein deposits such as beta-amyloid plaques and tau tangles in Alzheimer's disease. These inappropriate modifications affect normal brain function, resulting in cognitive decline and memory loss. Alzheimer's disease, on the other hand, does not directly harm the skull; rather, it predominantly affects the brain's structure and function within the limitations of the skull.



**Fig.1. Brain Anatomy in Alzheimer Disease [24]**

* + 1. ***Classification***

Alzheimer's disease is a neurodegenerative ailment that predominantly impairs cognitive functions, including memory and reasoning abilities. There are several ways to classify Alzheimer's disease, including based on its clinical progression, underlying pathology, and stages of severity. One common classification system is based on the stages of the disease:

1. Preclinical Stage: The earliest stage of Alzheimer's disease, occurring before visible symptoms arise. Changes in the brain, such as the deposition of amyloid plaques and tau tangles, are occurring at this stage, but patients do not yet display cognitive problems.

2. MCI (Mild Cognitive Impairment) As a result of Alzheimer's Disease: Some people with preclinical Alzheimer's disease may proceed to the mild cognitive impairment stage. MCI is characterised by obvious cognitive abnormalities that are not severe enough to significantly interfere with daily functioning. MCI does not always progress to Alzheimer's disease.

3. Early Stage (Mild Alzheimer's Disease): In this stage, individuals start to experience more noticeable cognitive impairments, including memory loss, difficulty finding words, challenges with problem-solving, and mood changes. However, they can still manage many of their daily activities independently.

4. Intermediate Stage (Moderate Alzheimer's Disease): As the disease advances, people reach the middle stage. Memory and cognitive deficits become more pronounced, leading to increased difficulty with tasks like managing finances, organizing daily activities, and recognizing familiar people. Behavioral and psychological symptoms, such as agitation, anxiety, and wandering, may also emerge.

5. Late Stage (Severe Alzheimer's Disease): At this point, individuals experience severe cognitive decline, resulting in an inability to communicate coherently, recognize loved ones, or perform basic self-care activities. Motor function can also be impaired, leading to difficulties with mobility and swallowing. Individuals require full-time assistance and care.

It's important to note that Alzheimer's disease causes a steady degradation in cognitive function and the specific symptoms and progression can vary from person to person. The underlying pathology of Alzheimer's disease involves the addition of abnormal proteins, including tau tangles and amyloid plaques, which lead to neuronal dysfunction and cell death.[12]

Diagnosing Alzheimer's disease is typically done through clinical assessments, cognitive testing, and ruling out other potential causes of cognitive decline. It's important for individuals who suspect they or a loved one may have Alzheimer's disease to seek medical evaluation and guidance from a healthcare professional, ideally a neurologist or geriatric specialist, for accurate diagnosis and appropriate management strategiesTop of Form

* + 1. ***Pathophysiology***

The pathophysiology of Alzheimer's disease is complex and involves multiple biological processes that result in progressive degeneration of neurons in the brain. The disease is distinguished by the formation of aberrant protein deposits, including amyloid plaques and tau tangles, as well as inflammation and synaptic dysfunction. Here's an overview of the key pathophysiological mechanisms involved in Alzheimer's disease:

1. Amyloid Plaques Formation:

• Amyloid Beta (Aβ) Protein: Amyloid plaques are clusters of abnormal protein fragments called beta-amyloid (Aβ). Aβ is formed through a sequence of enzymatic cleavages from a bigger protein known as amyloid precursor protein (APP).

• Abnormal Aggregation: In Alzheimer's disease, Aβ peptides tend to misfold and aggregate, forming insoluble plaques between neurons. These plaques disrupt normal neuronal function and contribute to neuronal damage.

2. Tau Protein and Neurofibrillary Tangles:

• Tau Protein: Tau is a protein that plays a role in stabilizing microtubules within neurons, which are essential for proper cell structure and function.

• Hyperphosphorylation: In Alzheimer's disease, tau proteins become hyperphosphorylated, causing them to detach from microtubules and aggregate into twisted fibers called neurofibrillary tangles.

• Disrupted Transport: Accumulation of neurofibrillary tangles disrupts the transport of nutrients and other essential molecules within neurons, leading to cell dysfunction and death.

3. Neuronal Dysfunction and Death:

• Synaptic Dysfunction: Amyloid plaques and tau tangles contribute to the dysfunction of synapses, the connections between neurons crucial for communication and memory formation.

• Neuronal Inflammation: The presence of inappropriate proteins in the brain causes an inflammatory response involving immune cells known as microglia. However, chronic inflammation can lead to neuronal damage.

• Neuronal Death: Over time, the accumulation of Aβ and tau, along with inflammation and impaired synaptic function, leads to the progressive death of neurons, particularly in regions crucial for memory and cognition.

4. Cholinergic System Impairment:

• Acetylcholine Deficiency: The brain's cholinergic system, responsible for the production and transmission of acetylcholine, is impaired in Alzheimer's disease.

• Impaired Cognitive Function: Reduced acetylcholine levels contribute to cognitive deficits, including memory loss and impaired thinking.

5. Oxidative Stress and Mitochondrial Dysfunction:

• Oxidative Stress: The abnormal protein aggregates and inflammation generate oxidative stress, which damages cellular components and contributes to neuronal dysfunction.

• Mitochondrial Dysfunction: Mitochondria are the structures in cells that generate energy, are impaired, leading to reduced energy production and increased oxidative stress.

6. Blood-Brain Barrier Dysfunction:

• Leaky Blood-Brain Barrier: The blood-brain barrier's integrity, which typically protects the brain from hazardous substances, is compromised in Alzheimer's disease. This allows toxic molecules and inflammatory factors to enter the brain.

These complex interactions between protein aggregation, inflammation, synaptic dysfunction, and cell death ultimately result in the cognitive impairments and memory loss characteristic of Alzheimer's disease. Understanding these pathophysiological mechanisms is crucial for developing effective treatments that target the underlying causes of the disease and slow its progression.[13]

* + 1. ***Signs And Symptoms***

Alzheimer's disease is a chronic neurological disease that predominantly affects cognitive functions, especially memory, thinking, and personality. The signs and symptoms of Alzheimer's disease symptoms might differ from person to person and can alter as the disease advances. Common early signs and symptoms include:

1. Memory Loss: Difficulty remembering recently learned information,

2. Difficulty with Planning and Problem-Solving: Struggling to develop and follow plans, manage finances, or solve problems that were once manageable.

3. Confusion with Time and Place: Getting disoriented about dates, seasons, or places, and not recognizing familiar locations.

4. Trouble Completing Familiar Tasks: Struggling with tasks that were once routine, such as cooking a meal or managing household chores.

5. Challenges with Language: Experiencing difficulty in finding the right words, following or joining a conversation, or repeating phrases.

6. Misplacing Items: Putting things in unusual places, such as putting a wallet in the refrigerator, and being unable to retrace steps to find them.

7. Decreased or Poor Judgment: Making questionable decisions, showing poor judgment in handling money, or falling for scams.

8. Withdrawal from Work or Social Activities: Losing interest in previously enjoyed hobbies, avoiding social activities, and becoming increasingly isolated.

9. Changes in Mood and Personality: Experiencing mood swings, becoming confused, suspicious, fearful, or anxious, especially in unfamiliar environments.

10. Difficulty Recognizing People: Struggling to recognize family members, friends, or even oneself in the mirror.

11. Loss of Initiative: Becoming less motivated to engage in activities, even those that were once enjoyable.

It's important to note that while memory loss is often associated with Alzheimer's disease, individuals may experience a combination of these symptoms to varying degrees. As the disease progresses, these symptoms worsen, leading to more severe cognitive impairment and an increased need for assistance with daily activities. Alzheimer's disease in its final stages, individuals may have difficulty communicating, become disoriented, and require round-the-clock care.

If you or someone you know is exhibiting these signs and symptoms, especially if they interfere with daily life and functioning, it's important to seek medical evaluation and diagnosis from a healthcare professional. Individuals having Alzheimer's disease and family members can benefit from early detection and suitable management precautions.

The specific aetiology of Alzheimer's disease is unknown; however, it is thought to be a combination of hereditary, environmental, and lifestyle factors. The following are some of the important elements thought to contribute to the development of Alzheimer's disease:

1. Genetics: While most cases of Alzheimer's disease are not directly inherited, there are certain genetic factors that can increase the risk. Mutations in specific genes, such as the APOE gene (especially the APOE ε4 allele), have been linked to an increased chance of acquiring Alzheimer's disease. However, having these genetic variants does not guarantee that an individual will develop the disease.

2. Age: The most important risk factor for Alzheimer's disease is age. The risk of developing the disease increases as people get older, especially after the age of 65.

3. Amyloid Plaques and Tau Tangles: Abnormal protein aggregates, such as amyloid plaques (composed of beta-amyloid protein) and tau tangles (resulting from hyperphosphorylated tau protein), are hallmarks of Alzheimer's disease. These protein deposits disrupt normal cellular function and contribute to neuronal damage and death.

4. Neuroinflammation: Chronic inflammation in the brain is thought to play a role in the development and progression of Alzheimer's disease. Inflammatory processes may contribute to the accumulation of abnormal proteins and damage to neurons.

5. Oxidative Stress: When there is an imbalance between the generation of reactive oxygen species (free radicals) and the body's ability to eliminate them with antioxidants, oxidative stress arises. Oxidative stress can damage cells, including neurons, and is believed to be involved in the development of Alzheimer's disease.

6. Mitochondrial Dysfunction: Mitochondria are responsible for producing energy within cells. Dysfunction in mitochondria can lead to reduced energy production and increased oxidative stress, which may contribute to neuronal damage.

7. Neurotransmitter Imbalance: Neurotransmitters are chemical messengers that transmit signals between nerve cells. In Alzheimer's disease, there is a disruption in neurotransmitter systems, particularly those involving acetylcholine, which is important for memory and cognitive function.

8. Vascular Factors: Cardiovascular risk factors, such as Diabetes, high blood pressure, and high cholesterol have all been related to an increased risk of Alzheimer's disease. These factors can impair blood flow to the brain, contributing to cognitive decline.

9. Environmental Factors: Certain environmental factors may rise the Alzheimer's disease risk. These include a past history of head trauma, exposure to certain toxins, and chronic stress.

10. Lifestyle Factors: Unhealthy lifestyle choices, such as a diet heaviest in saturated fats and sugar, lack of physical activity, smoking, and excessive alcohol consumption, can all contribute to the development of Alzheimer's disease.

It's important to note that Alzheimer's disease is likely caused by a combination of these factors, and the interplay between genetic susceptibility, environmental influences, and other mechanisms is still an active area of research. While some risk factors cannot be changed (such as age and genetics), adopting a healthy lifestyle, managing cardiovascular risk factors, and staying mentally and socially engaged may help reduce the risk of getting Alzheimer's disease.

1. **Endocrine disorder and diseases**

The endocrine system, also referred to as the hormonal system, is a network of organs and glands which generate hormones. Sometimes the body produces excessive or insufficient hormones, or it may stop functioning them as required. Therefore, endocrine problems and illnesses may develop.

* 1. ***Diabetes insipidus***

Diabetes mellitus (DM) is a disorder in which blood glucose levels are not properly controlled. India had Three hundred and fifteen million inhabitants with high blood pressure, 254 million with Obesity in general, particularly 351 million with abdominal obesity. There were 101 tens of millions of people with diabetes along with 136 hundred million with pre-hyperglycemia. [38]

Sugar in the blood (glucose) Excessive amounts can result in diabetes. It occurs when your body is unable to efficiently metabolise insulin or when your pancreas fails to produce any insulin at all. Every age group is impacted by diabetes.

***4.1.1. Types***

There are four types of Diabetes mellitus such as Diabetes of the types 1 and 2, and diabetes associated with pregnancy are all examples of diabetes. Hyperglycemia of the Young with Maturity (MODY).

Among all four types, the most frequent type of diabetes is type 2. If you have a family history of the disorder and risk factors for it, such as being overweight or obese. Type 2 diabetes may affect anyone at any age, even children. [14]

***4.1.2. Symptoms***

The symptoms of Diabetes mellitus such as Polydipsia (excessive thirst) and dry mouth, Urination on a regular basis, Fatigue, Vision distortion, Unknown cause of weight reduction, Numbness or tingling sensations in your hands or feet, Sores or cuts that take a long time to heal, Skin and/or vaginal yeast infections on a regular basis.[26]

The many other symptoms are also there apart from all this mention in Fig. 2. like weight loss smell acetone, hyperventilation Lethargy, stupor, Glycosuria, and polyurea. In Fig. 2 the symptoms are classified on the basis of their origin means from where they are originated.



**Fig. 2. Symptoms of Diabetes [33] Mellitus**

***4.1.3. Causes***

Obesity, excess weight, and inactivity

Type 1 Diabetes: -

The immune system plays a role in type 1 diabetes. incorrectly targets along with kills the insulin-secreting cells in the pancreas. Although the precise source of this autoimmune reaction is not entirely understood, genetic and environmental factors are thought to both contribute. Environmental triggers, certain viral infections, and genetic predisposition have all been mentioned as potential reasons.[39]

Type 2 Diabetes: -

kind 2 diabetes is the most common kind of diabetes., is distinguished by insulin resistance, where the body's cells do not react to insulin as well as they should. High blood sugar levels are the result of this. Type 2 diabetes has a complex aetiology involving both hereditary and environmental factors:

Genetics: Type 2 diabetes risk can be raised by family history and genetic susceptibility. The sensitivity of the body to insulin as well as other aspects of glucose metabolism may be impacted by specific gene variations.

Obesity: Being overweight, especially in the abdominal area, increases the risk of type 2 diabetes. Inflammation and insulin resistance brought on by obesity have an impact on how glucose is regulated.

Physical inactivity: Being inactive on a daily basis makes the body less sensitive to insulin and increases weight gain, which raises the Type 2 diabetes risk.

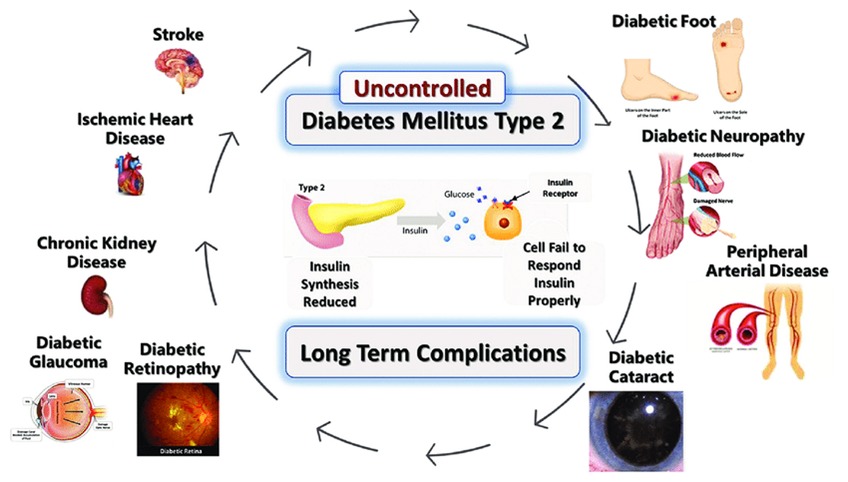
Unhealthy Diet: Resistance to insulin and obesity can be brought on by a diet high in processed foods, sweet drinks, and unhealthy fats.

Age: As people get older, especially after the age of 45, their proclivity to acquire type 2 diabetes rises.

Gestational Diabetes: -

When hormonal changes during pregnancy interfere with insulin function, Diabetes develops throughout pregnancy. Although it usually goes away after delivering birth, women who have had gestational diabetes are more prone to get it. later develop type 2 diabetes. Although the precise causes of gestational diabetes are not entirely understood, it is thought that hormonal and genetic factors are involved [15].

Fig. 3 explains the Complication and risk factors of Type-2 Diabetes Mellitus like Type 2 Diabetes Mellitus increases the risk of a variety of complications, including an increased susceptibility to strokes, diabetic cataracts that can impair vision, the development of chronic kidney diseases leading to kidney dysfunction, an increased risk of heart diseases such as coronary artery disease and myocardial infarctions, and the possibility of diabetic glaucoma and retinopathy, both of which can seriously impair eyesight. Furthermore, diabetic neuropathy can cause tingling, numbness, or discomfort in the extremities, emphasising the significance of complete diabetes care and monitoring to avoid these potentially serious and life-altering outcomes.



**Fig. 3. Diabetes mellitus pathogenesis and chronic consequences [34]**

***4.1.4. Treatment***

There is currently no treatment for type 2 diabetes, but our researchers are working on a groundbreaking study on weight management to assist people in putting their condition into remission.

Diabetes type 1 is treated. with Insulin shots or a device that pumps insulin, routine blood sugar monitoring, and carbohydrate tracking. Pancreas transplantation or islet cell transplantation may be options for some persons with type 1 diabetes.

Oral Medication: -

Gliptins are a class of oral diabetes drugs that have been given the Food and Drug Administration's (FDA) approval to treat adults with type 2 diabetes mellitus. FDA-approved Alogliptin, linagliptin, saxagliptin, and sitagliptin are DPP-4 inhibitors.[40]

Diabetes type 2 management includes

• A nutritious diet.

• Consistently moving.

• Losing weight.

• Insulin treatment or possibly diabetes medications.

• Monitoring of blood sugar

By taking these actions, it is more probable that blood sugar levels will remain within a healthy range. And they could aid in delaying or avoiding difficulties.[27][38]

Insulin therapy: -

Certain type 2 diabetics require insulin therapy. Today, insulin therapy may be recommended earlier if blood sugar targets are not achieved by lifestyle changes and other treatments, as opposed to the past when it was only used as a last choice.

The time it takes to start working and how long it takes to have an effect vary between different forms of insulin. For instance, long-acting insulin is made to function all day or overnight to maintain stable blood sugar levels. At mealtimes, short-acting insulin is typically administered.

***4.1.5. Diagnosis***

Fasting Blood Sugar Test: -

A fasting blood glucose level of 99 mg/dL or less is considered normal, a reading of 100 to 125 mg/dL denotes prediabetes, and a reading of 126 mg/dL or above denotes diabetes.

HbA1c Test: -

For the diagnosis of type 2 diabetes, the haemoglobin A1C blood test is incredibly sensitive and precise. In order to assess whether your blood glucose is regularly excessive, it checks your average blood level glucose over a two- or three-month period [16].

***4.2. Osteoporosis***

Osteoporosis is a bone disease that occurs when bone quality or structure decreases, mineral density of the bones turns it down, or when bone mass decreases. This may result in a loss of bone density and an elevated risk of breaking bones.[17]

Osteoporosis is known as a "silent" disease because it often goes undetected until a bone is fractured, and sometimes even then. The leading cause of fractures in elderly men and postmenopausal women is osteoporosis. Any bone can shatter, although the hip, spine, and wrist vertebrae are the most commonly broken.[34][36]

***4.2.1. Symptoms***

Osteoporosis does not have symptoms like many other medical disorders. That is why healthcare experts refer to it as a quiet sickness.

You will not feel or see any symptoms of osteoporosis. There will be no headache, fever, or stomachache to alert you that something is amiss with your body.

The most typical "symptom" is unexpectedly breaking a bone, especially after a little fall or mishap that would normally not affect you.

Although osteoporosis undirectly create symptoms, a little change in your body may indicate that your bones are weakening or becoming less dense. These osteoporosis warning signals can include:

Lacking at least one inch from your height.

alterations to your normal posture (more bending forward or stooping).

Breathlessness (should your spinal discs be compressed sufficiently to lower your lung capacity).

Lumbar spine discomfort, often known as lower back ache.

It could be difficult to recognize aesthetic changes in oneself.

Someone close to you might be more likely to notice physical changes, particularly in your posture or height. Older people are sometimes made fun of for "shrinking" as they age, but this is actually an indication that you should have a bone density test and see a doctor.[28]

***4.2.2. Causes***

Osteoporosis is a condition characterized by weakened and brittle bones, making them more susceptible to fractures and breaks. Many factors can contribute to the development of osteoporosis:

1. Age: As you age, your bones naturally become less dense and more fragile, increasing the risk of osteoporosis.

2. Gender: Women are more likely to develop osteoporosis, especially after menopause., due to the decrease in estrogen levels that plays a protective role for bone density.

3. Hormonal Changes: Hormonal imbalances, such as low levels of estrogen or testosterone, can contribute to bone loss.

4. Lack of Calcium and Vitamin D: A diet deficient calcium and vitamin D deficiency can result to reduced bone density and increased risk of osteoporosis.

5. Sedentary Lifestyle: A Lack of weight-bearing exercise and physical activity can lead to weaker bones.

6. History of family: A family history of osteoporosis or fractures can raise your risk,

7. Certain Medical Conditions: Conditions such as hyperthyroidism, hypogonadism, rheumatoid arthritis, celiac disease, and some autoimmune disorders can increase the risk of osteoporosis.

8. Medications: Long-term use of certain medications, such as glucocorticoids (steroids), anticonvulsants, and some cancer treatments, can negatively affect bone health.

9. Excessive smoking and drinking Alcohol Consumption: Both smoking and heavy alcohol consumption can weaken bones and increase the risk of osteoporosis.

10. Low Body Weight and BMI: Having a low body weight or low body mass index (BMI) can be associated with lower bone density.

11. Ethnicity: Individuals of Asian or Caucasian descent are more likely to develop osteoporosis.

12. Gender Reassignment: Transgender individuals who undergo gender-affirming hormone therapy may be at risk for bone density changes.

13. Malabsorption Issues: Digestive disorders or surgeries that affect nutrient absorption, such as celiac disease or gastric bypass surgery, can impact bone health.

Preventing osteoporosis involves maintaining a healthy lifestyle that includes a calcium and vitamin D-rich diet, and weight-bearing exercise exercises, avoiding smoking and excessive alcohol consumption, and addressing any underlying medical conditions. If you're concerned about osteoporosis, it is best to get the advice and assessment of a healthcare expert.[28][29]

***4.2.3. Pathophysiology***

The pathophysiology of osteoporosis involves an imbalance between bone formation and bone resorption, resulting in decreased bone density and a higher probability of fractures. Bone is a living tissue that undergoes constant remodeling is a procedure of breaking down old bone tissue and replacing it with new bone. Osteoporosis disrupts this balance, resulting in weaker bones. Here's a simplified overview of the pathophysiological processes involved:

1. Normal Bone Remodeling:

• Bone remodeling involves two main cell types: osteoblasts and osteoclasts.

• Osteoblasts are responsible for building new bone tissue by depositing minerals (like calcium and phosphorus) and collagen.

• Osteoclasts are responsible for breaking down old bone tissue, releasing minerals into the bloodstream.

2. Bone Resorption and Formation Imbalance:

• In osteoporosis, there has been a rise in osteoclast activity (bone resorption) and a reduction in osteoblast activity (bone formation).

• Osteoclasts break down bone tissue faster than osteoblasts can build new bone, leading to a net loss of bone mass.

3. Hormonal Factors:

• Estrogen plays a vital function in bone density maintenance by inhibiting osteoclast activity.

• In postmenopausal women, the decline in estrogen levels contributes to increased osteoclast activity and bone loss.

• Similarly, low testosterone levels in men can also contribute to bone loss.

4. Inflammatory Factors:

• Chronic inflammation can stimulate the production of certain molecules that promote osteoclast activity and bone resorption.

5. Parathyroid Hormone (PTH) and Calcium Regulation:

• PTH is released by the parathyroid glands when blood calcium levels are low., which promotes osteoclasts to break down bone to release calcium into the bloodstream.

• Chronic elevation of PTH due to calcium imbalance can contribute to bone loss.

6. Genetic Factors:

• Genetic factors can influence an individual's susceptibility to osteoporosis, including how their bone cells function and respond to hormones.

7. Age-Related Changes:

• With age, the ability of osteoblasts to form new bone decreases, and bone repair becomes less efficient.

8. Microarchitectural Changes:

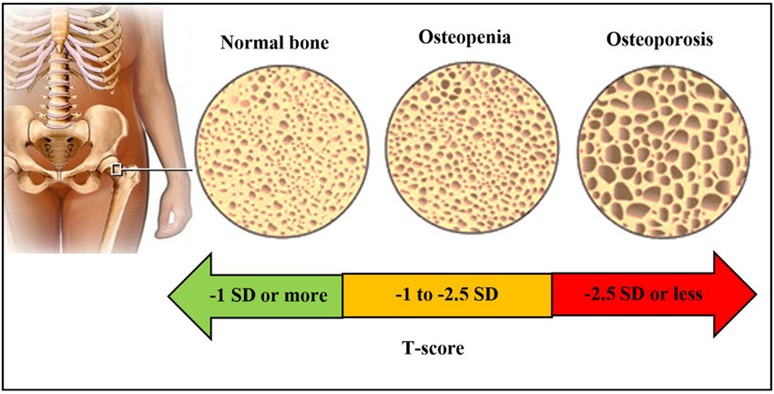
• Osteoporosis affects the structure of bone tissue at a microscopic level, leading to thinning and weakening of the bone trabeculae (microscopic bone structures).

9. Fracture Risk:

• As bone density decreases, bones become more porous and brittle, increasing the risk of fractures, especially in weight-bearing areas like the spine, hips, and wrists.

Understanding the underlying processes of osteoporosis helps guide preventive and treatment strategies. These may include dietary interventions, calcium and vitamin D supplementation, weight-bearing exercises, therapy of harmone replacement (in some cases), medications that target bone turnover, and lifestyle modifications to minimize fracture risk [30].

Fig. 4 explains Difference in normal bone and osteoporosis like Normal bone appears dense and resilient in a cross-section, with a well-maintained arteries and cortical bone structure that provides strength and stability. Osteoporotic bone, on the other hand, has decreased density and altered microarchitecture, making it appear porous and feeble. This structural degeneration makes osteoporotic bone more prone to fractures, especially in weight-bearing areas like the hip and spine, highlighting the condition's underlying frailty and risk to injury



**Fig. 4. Normal bone, osteopenia, and osteoporosis are all compared [35]**

***4.2.4. Sign and symptoms***

Osteoporosis since it is often referred to as a "silent disease" it doesn't usually cause noticeable Symptoms will last until a fracture occurs. However, as the condition progresses, some individuals may experience certain signs and symptoms:

1. Loss of Height: Osteoporosis can lead to compression fractures in the spine, causing a gradual loss of height over time.

2. Back Pain: Back pain can be caused by compression fractures in the spine chronic or acute back ache. The pain may worsen with movement and can be exacerbated by standing or walking for extended periods.

3. Fractures: Osteoporosis significantly increases the incidence of fractures, especially in the hip, spine, and wrist are all affected. These fractures are possible due to minor trauma or even with no apparent cause.

4. Stooped Posture: Compression fractures in the spine can result in a stooped or hunched posture, Known colloquially as a "dowager's hump,” or kyphosis.

5. Easy Fractur ability: Osteoporotic bones are more fragile, making fractures more likely from minor incidents that might not typically cause fractures in healthy individuals.

6. Loss of Bone Mass: While this cannot be observed directly, advanced bone density scans (like DEXA scans) can reveal decreased bone mass, indicating osteoporosis.

7. Limited Mobility: Pain and fractures can lead to limited mobility and difficulties completing daily activities.

It's worth noting that these symptoms can be subtle and might not be solely indicative of osteoporosis. Additionally, many people with osteoporosis remain asymptomatic until they experience a fracture. Therefore, regular bone health assessments, especially for individuals at higher risk, are crucial for early detection and intervention.[37]

***4.2.5. Risk factors for osteoporosis include***

• Age (postmenopausal women and older individuals are at higher risk)

• Gender (women are more susceptible)

• Family history of osteoporosis

• Low body weight or BMI

• Smoking

• Excessive alcohol consumption

• Sedentary lifestyle

• Poor nutrition and low calcium intake

• Certain medical conditions (rheumatoid arthritis, hyperthyroidism, etc.)

• Long-term use of certain medications (glucocorticoids, anticonvulsants, etc.)

If you're concerned about osteoporosis or its risk factors, It is best to seek the advice of a healthcare practitioner. evaluate your individual risk and recommend appropriate measures for prevention, early detection, and management.[31][36]

***4.2.6. Prevention***

Osteoporosis prevention

Maintain healthy and diverse a diet high in fresh fruits and vegetables and healthy grains.

Consume calcium-rich meals.

Get enough vitamin D.

Smoking should be avoided.

Reduce your alcohol consumption.

Caffeine should be avoided.

carry out regular weight-bearing and strength-training exercises

***4.2.7. Treatment***

The goal of osteoporosis treatment is to strengthen bones, decrease the danger of fractures, and improve overall bone health. The approach to treatment often involves a combination of lifestyle changes, medications, and other interventions. The specific treatment plan will depend on the severity of osteoporosis, individual risk factors, and the recommendations of a healthcare provider. Here are some common treatment strategies:[32][17]

1. Lifestyle Modifications:

• Adequate Calcium and Vitamin D Consumption: Consuming foods rich in calcium and ensuring sufficient vitamin D intake is crucial in order to maintain bone health.

• Weight-Bearing Exercises: Engaging in weightlifting exercises like jogging, walking, dancing, and resistance training can help improve strength and bone density.

• Fall Prevention: Preventing falls can minimize the chance of fractures. This comprises using assistive devices, improving home safety, and maintaining good balance.

2. Medications:

• Bisphosphonates: These medications inhibit bone resorption and help maintain bone density. Examples include alendronate, risedronate, and ibandronate.

• SERMs (Selective estrogen Receptor Modulators): Medications such as raloxifene imitate the actions of estrogen. in some parts of the body, promoting bone density.

• Denosumab: This injectable medication targets a protein involved in bone resorption, slowing down bone loss.

• Teriparatide and Abaloparatide: These are synthetic forms of parathyroid hormone that stimulate bone formation. They are typically used for severe osteoporosis.

• Hormone Replacement Therapy (HRT): Estrogen therapy, often in conjunction with progestin, can be prescribed for postmenopausal women to help maintain bone density. However, the risks and benefits should be carefully considered.

3. Calcitonin: This hormone helps regulate calcium levels and can be administered as a nasal spray to slow down bone resorption.

4. Osteoporosis Management Clinics: Specialized clinics provide comprehensive care, including education, exercise programs, and medical interventions.

5. Nutritional Supplements: In cases of severe deficiency, calcium and vitamin D supplements may be recommended.

6. Fall Risk Assessment and Prevention: Identifying and addressing factors that contribute to falls can significantly reduce the risk of fractures.

7. Surgery: In cases of severe fractures or spinal deformities, surgical interventions such as kyphoplasty or vertebroplasty may be considered.

It's important to note that treatment plans are tailored to individual needs, and the decision to start or change a treatment regimen should be made in consultation with a healthcare provider. Monitoring and follow-up on a regular basis appointments are crucial to evaluate the effectiveness of treatment and make any necessary adjustments. Osteoporosis management also involves ongoing lifestyle modifications to support bone health and minimize fracture risk.[18][17]

Observational research is used in descriptive epidemiology to examine how diseases are distributed through time, space, and individuals. The study does not consider any additional or causative hypotheses; instead, it describes the distribution of a set of variables.

**5. Conclusion**

The epidemiology of respiratory tract infections, diabetes, Parkinson's disease, Alzheimer's disease, and Parkinson's disease highlights the significance of managing these diseases within the context of global health. The effects of these disorders must be reduced through targeted interventions that take risk factors, genetics, and environmental variables into account. For effective preventive, early diagnosis, and management techniques to be developed, doctors, researchers, and public health authorities must work together continuously. This will ultimately enhance one's quality of life for those affected by these diseases. Promoting prevention, early diagnosis, and efficient care is crucial as the prevalence of chronic diseases rises, driven by a number of variables such as ageing populations, alterations in lifestyle, and environmental impacts. For the development of comprehensive approaches that include education, risk reduction, and novel therapies, multidisciplinary collaboration across medical research, public health initiatives, and healthcare delivery systems is essential

Acknowledgments

This book chapter was supported by Reva University, Bengaluru, Karnataka 560064, India and National Horticulture Research and Development Foundation (NHRDF), Nashik-422003, Maharashtra, India

**Conflicts of interest**

The authors declare no conflict of interest, financial or otherwise.

**Ethical approval**

Ethics approval was not required for this work.

**References**: -

1). Davie CA (2008). "A review of Parkinson's disease". British Medical Bulletin. 86 (1): 109–127. doi:10.1093/bmb/ldn013. PMID 18398010]

2). Villar-Piqué A, Lopes da Fonseca T, Outeiro TF (October 2016). "Structure, function and toxicity of alpha-synuclein: the Bermuda triangle in synucleinopathies". Journal of Neurochemistry. 139 (Suppl 1): 240–255. doi:10.1111/jnc.13249. PMID 26190401. S2CID 11420411

3)Burré J, Sharma M, Südhof TC (March 2018). "Cell Biology and Pathophysiology of α-Synuclein". Cold Spring Harbor Perspectives in Medicine. 8 (3): a024091. doi:10.1101/cshperspect.a024091. PMC 5519445. PMID 2810853

4)Dickson DV (2007). "Neuropathology of movement disorders". In Tolosa E, Jankovic JJ (eds.). Parkinson's disease and movement disorders. Hagerstown, MD: Lippincott Williams & Wilkins. pp. 271–283. ISBN 978-0-7817-7881-7

5) Armstrong MJ, Okun MS (February 2020). "Diagnosis and Treatment of Parkinson Disease: A Review". JAMA. 323 (6): 548–560. doi:10.1001/jama.2019.22360. PMID 32044947. S2CID 211079287.

6) Obeso JA, Rodríguez-Oroz MC, Benitez-Temino B, Blesa FJ, Guridi J, Marin C, Rodriguez M (2008). "Functional organization of the basal ganglia: therapeutic implications for Parkinson's disease". Movement Disorders. 23 (Suppl 3): S548–S559. doi:10.1002/mds.22062. PMID 18781672. S2CID 13186083.

7)Davie CA (2008). "A review of Parkinson's disease". British Medical Bulletin. 86 (1): 109–127. doi:10.1093/bmb/ldn013. PMID 18398010

8)Schulz-Schaeffer WJ (August 2010). "The synaptic pathology of alpha-synuclein aggregation in dementia with Lewy bodies, Parkinson's disease and Parkinson's disease dementia". Acta Neuropathologica. 120 (2): 131–143. doi:10.1007/s00401-010-0711-0. PMC 2892607. PMID 20563819.

9) Obeso JA, Rodriguez-Oroz MC, Goetz CG, et al. (June 2010). "Missing pieces in the Parkinson's disease puzzle". Nature Medicine. 16 (6): 653–661. doi:10.1038/nm.2165. PMID 20495568. S2CID 3146438.

10) Dickson DV (2007). "Neuropathology of movement disorders". In Tolosa E, Jankovic JJ (eds.). Parkinson's disease and movement disorders. Hagerstown, MD: Lippincott Williams & Wilkins. pp. 271–283. ISBN 978-0-7817-7881-7.

1. Jennifer A. Pallone DO, Introduction to Parkinson’s Disease, Elsevier
2. Alzheimer's Disease: A General Introduction and Pathomechanism, Finder, Verena H, the journal of clinical investigation.
3. Michael A. DeTure & Dennis W. Dickson, The neuropathological diagnosis of Alzheimer’s disease
4. Introduction to Diabetes Mellitus, Kirti Kaul, Joanna M. Tarr, Shamim I. Ahmad, Eva M. Kohner & Rakesh Chibber
5. Diabetes mellitus: an overview of the types, symptoms, complications and management., Cloete L , Nursing Standard (Royal College of Nursing (Great Britain) : 1987), 28 Oct 2021, 37(1):61-66
6. Type 2 Diabetes Mellitus: Update on Diagnosis, Pathophysiology, and Treatment Richard J. Mahler, Michael L. Adler, The Journal of Clinical Endocrinology & Metabolism, Volume 84, Issue 4, 1 April 1999
7. Diagnosis and Treatment of Osteoporosis, MARY GAYLE SWEET, MD, JON M. SWEET, MD, MICHAEL P. JEREMIAH, MD, AND SIM S. GALAZKA, MD
8. Diagnosis, Screening, Prevention, and Treatment of Osteoporosis, Author links open overlay panelKaren F. Mauck MD, MSc a, Bart L. Clarke MD b, Elsevier
9. Introduction: Acute Respiratory Tract Infections: The Forgotten Pandemic

Frank Shann, Ann Woolcock, Robert Black, Allan Cripps, Hjordis Foy, Mark Harris and Rennie D'Souza

1. Upper Respiratory Tract InfectionMicah Thomas; Paul A. Bomar.
2. Lower respiratory tract infections: What to know by Alana Biggers, M.D., MPH — By Jenna Fletcher
3. Prevention of paediatric respiratory tract infections: emphasis on the role of OM-85 U. B. Schaad
4. Upper Respiratory Tract Infection by Dr. Avni Serasiya December 15, 2022
5. Therapeutic and Diagnostic Applications of Nanocomposites in the Treatment Alzheimer's Disease Studies, Siddhartha Dan, Deeksha Sharma, Kartikey Rastogi, Shaloo, Himanshu Ojha, Mallika Pathak, Rahul Singhal, volume 12, Biointerface Research in Applied Chemistry.
6. A Non-Invasive Medical Device for Parkinson’s Patients with Episodes of Freezing of Gait, C. Punin, B. Barzallo, Roger Clotet, Llumiguano Carlos, MDPI.
7. Dental and oral symptoms of diabetes mellitus,Mária Albrecht, Jolán Bánóczy, Gyula Tamás Jr. ,Volume 16, Community Dentistry and Oral Epidemiology.
8. Prevention and Treatment of the Complications of Diabetes Mellitus, Charles M. Clark, Jr., M.D., and D. Anthony Lee, M.D.
9. AETIOLOGY AND PRESENTING SYMPTOMS IN MALE OSTEOPOROSIS, P. PERIS, N. GUAÑABENS, A. MONEGAL, X. SURIS, L. ALVAREZ, M. J. MARTINEZ DE OSABA, M. V. HERNANDEZ, J. MUÑOZ-GOMEZ.
10. Secondary Causes of Osteoporosis, Lorraine A. Fitzpatrick MD, Elsevier
11. A comprehensive overview on osteoporosis and its risk factors, Farkhondeh Pouresmaeili, Behnam Kamalidehghan, Maryam Kamarehei & Yong Meng Goh
12. Diagnosis of osteoporosis and assessment of fracture risk, Prof John A Kanis, The Lancet.
13. Osteoporosis in elderly: prevention and treatment, Manish Srivastava, MD, Chad Deal, MD, VOLUME 18, ISSUE 3, P529-555, AUGUST 2002
14. Incidence of Diabetes Mellitus in Patients with Hepatitis B and C virus Infection, March 2019, International Journal of Scientific and Research Publications 9(3):p8726, 10.29322/IJSRP.9.03.2019.p8726
15. Invited Commentary: Smash of Diabetes Mellitus on Smile, June 2021Advances in Human Biology, 10.4103/aihb.aihb\_68\_21, NC-SA 4.0
16. Advances in Sensing Technologies for Monitoring of Bone Health, April 2020, Biosensors 10(4):42, 10.3390/bios10040042
17. Osteoporosis prevention, diagnosis, and therapy Anne Klibanski, Lucile Adams-Campbell, Tamsen Bassford, Steven N. Blair, Scott D. Boden, Kay Dickersin, David R. Gifford, Lou Glasse, Steven R. Goldring, Keith Hruska, Susan R. Johnson, Laurie K. McCauley, William E. Russell
18. Wright NC, Looker AC, Saag KG, Curtis JR, Delzell ES, Randall S, et al. The recent prevalence of osteoporosis and low bone mass in the United States based on bone mineral density at the femoral neck or lumbar spine. J Bone Miner Res. 2014;29:2520–6.
19. G.P. Fadini et al.Characteristics and outcomes of the hyperglycemic \hyperosmolar non-ketotic syndrome in a cohort of 51 consecutive cases at a single center Diabetes Res Clin Pract (2011)
20. E.S. Ford et al. Pre-diabetes and the risk for cardiovascular disease: a systematic review of the evidence J Am Coll Cardiol (2010)
21. C.S. Gray et al. Glucose-potassium-insulin infusions in the management of post-stroke hyperglycaemia: the UK Glucose Insulin in Stroke Trial (GIST-UK) Lancet Neurol (2007)