Schizophrenia, Depression & Alzheimer’s Disease

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Schizophrenia (meaning the “splitting of mental functions”) is a chronic and severe mental disorder often characterized by abnormal social behavior and failure to recognize what is real.
Where are those voices coming from?

The Radio Told Me To Free All The Zoo Animals

I Saw Elephants Under My Bed

Aliens are contacting me

Maybe I Am Jesus

They're Following Me!
CAUSES

1. Multiple areas in the PREFRONTAL lobe where the processing of the neural signals has become blocked or dysfunctional. The underlying cause is usually decreased responsiveness to the neurotransmitter GLUTAMATE.

2. Excessive responsiveness of a group of neurons to DOPAMINE, esp. in the PREFRONTAL LOBE.
   - Dopamine agonists (Amphetamine) exacerbate the condition.
   - Post-mortem shows increased Dopamine binding sites in the brain.
   - Neuroleptics directly block the Dopamine receptors in the brain.

3. Abnormal function of a crucial part of HIPPOCAMPUS (it is often reduced in the disease).
Genetic Predisposition + Environmental, Social and Psychological Factors → Neurodevelopmental abnormalities and target features → Brain dysfunction, improper balance of chemicals → Schizophrenia
TREATMENT

• **Anti-psychotic (Neuroleptic) Drugs**: they block Dopamine receptors.
  - Chlorpromazine

• **Social treatment**: structured work and social environment.
Depression is a state of low mood and aversion to activity. It can affect a person's thoughts, behavior, feelings and sense of well-being. Depressed patients experience symptoms of grief, unhappiness, despair and misery. They also lose their appetite and have insomnia.
What is the difference between being Sad and being Depressed?
Signs of Depression

Lethargy
Trouble sleeping
Trouble focusing
Apathy
Sadness
Irritability
Feelings of worthlessness
Detaching from friends
Appetite and weight
Low sex drive
Pain
Recklessness
Alcohol abuse
Drug abuse (prescription or other)
Suicidal tendencies
• **CAUSE:**
  - Diminished formation in the brain of **norepinephrine** or **serotonin** or both.

• **TREATMENT:**
  - **Monoamine oxidase inhibitors.** E.g. phenelzine and Amphetamine related (block the destruction of NE & serotonin once they are formed).
  - **Tricyclic antidepressants.** E.g. imipramine (block reuptake of NE and serotonin by nerve endings so they remain active for longer periods after secretion).
  - **Electroconvulsive Therapy (ECT):** It is also called Shock Therapy. An electric current is passed between 2 electrodes applied to the anterior temporal areas of the scalp. Before the treatment is given, the patient is anesthetized and receives a muscle relaxant. The ECT causes a generalized seizure similar to epilepsy and leads to an increased secretion of NE.
Depression can lead to **Bipolar disorder** (previously called manic-depressive psychosis).

It is a mental condition marked by alternating periods of elation and depression.

(Drugs diminishing formation or action of NE & serotonin can be effective in treating the manic phase of this condition.)
ALZHEIMER’S DISEASE

Alzheimer’s disease is defined as premature aging of the Brain. It is a fatal neurodegenerative disease usually beginning in mid adult life and progressing rapidly to extreme loss of mental powers—similar to that seen in very, very old age. It is a metabolic degenerative disease of the brain.
What causes Alzheimer’s?
1. Role of Amyloid Plaques in Alzheimer’s (Remnants of Amyloid Precursor Protein)

2. Beta-Amyloid

3. Beta-Amyloid Plaque
AD BRAIN

- TANGLES
- BETA AMYLOID PLAQUE

NORMAL BRAIN

- NEURON
Evidence for role of Beta- Amyloid peptide in pathogenesis of Alzheimer’s disease:

1. All currently known mutations associated with Alzheimer’s disease increase the production of beta-amyloid peptide;

2. Patients with trisomy 21 (Down syndrome) have three copies of the gene for amyloid precursor protein and develop neurological characteristics of Alzheimer’s disease by midlife;

3. Patients who have abnormality of a gene that controls apolipoprotein E, a blood protein that transports cholesterol to the tissues, have accelerated deposition of amyloid and greatly increased risk for Alzheimer’s disease;

4. Transgenic mice that overproduce the human amyloid precursor protein have learning and memory deficits in association with the accumulation of amyloid plaques; and

5. Generation of anti-amyloid antibodies in humans with Alzheimer’s disease appears to attenuate the disease process.
Vascular disorders may contribute to progression of Alzheimer’s disease.

Cerebrovascular diseases contribute towards Alzheimer’s:

• Hypertension
• Hyperlipidemia
• Diabetes
What happens in Alzheimer’s?
Onset of Alzheimer's disease slowly progresses over 7-10 years.

**SYMPTOMS**

**Early stages include:**
- Memory lapses
- Forgetting names of people and places
- Difficulty finding words for things
- Inability to remember recent events & appointments.

As the **disease progresses**, symptoms include:
- Difficulties with language
- Apraxia
- Problems with planning and decision making
- Confusion

In the **later stages**, symptoms include:
- Wandering, disorientation
- Apathy
- Psychiatric symptoms - depression, hallucinations, delusions
- Behavioural problems - aggression, agitation
- Altered eating habits
- Incontinence