BIO PSYCHOLOGY

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Foundations of Biopsychology

Biopsychology integrates neuroscience, genetics, and psychology to study how biological systems influence behavior, cognition, and emotion.

Key Focus Areas:

- **Neuroplasticity**: Dynamic brain reorganization in response to experience (e.g., post-stroke recovery).
- **Epigenetics**: Gene-environment interactions (e.g., trauma altering DNA methylation in stress-related genes).
- **Neuropharmacology**: Mechanisms of psychotropic drugs (e.g., SSRIs' impact on synaptic serotonin).

2. Historical Evolution

From Dualism to Materialism: Critique of Descartes' mind-body dualism in light of modern fMRI evidence for neural correlates of consciousness.

Localization vs. Holism:

- **Broca's Area**: Language production (supported by lesion studies).
- **Lashley's Mass Action**: Distributed memory networks (challenging strict localization).

20th-Century Advances: Hebb's rule ("fire together, wire together") as the basis for synaptic plasticity.

3. Neuroanatomy & Functional Systems

- **Central Nervous System (CNS)**:
  - **Hippocampus**: Role in spatial memory (O'Keefe's place cells).
  - **Amygdala**: Fear conditioning (LeDoux's dual-pathway model).
- **Peripheral Nervous System (PNS)**:
  - **Autonomic Nervous System (ANS)**: Sympathetic (fight-or-flight) vs. parasympathetic (rest-digest) dysregulation in anxiety disorders.

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4. Neurophysiology

- **Action Potential**: Hodgkin-Huxley model of ion channel dynamics.
- **Synaptic Transmission**:
  - **Long-Term Potentiation (LTP)**: NMDA receptor involvement in memory (Bliss & Lømo, 1973).
  - **Neuromodulators**: Dopamine's role in reinforcement learning (Schultz's reward prediction error).

5. Neuroimaging & Methods

- **fMRI**: BOLD signal limitations (temporal lag, indirect measure).

- **PET**: Radioligands for dopamine D2 receptors in schizophrenia.
- **Optogenetics**: Precision control of neuronal activity in animal models (Deisseroth, 2005).

6. Clinical Applications

- **Schizophrenia**:
  - **Dopamine Hypothesis**: Mesolimbic hyperactivity (positive symptoms) vs. mesocortical hypoactivity (negative symptoms).
  - **Structural Abnormalities**: Reduced gray matter in prefrontal cortex (meta-analysis by Haijma et al., 2013).
- **Depression**:
  - **Neurotrophic Hypothesis**: Low BDNF linked to hippocampal atrophy (Duman & Monteggia, 2006).
  - **Ketamine**: Rapid antidepressant effects via mTOR activation.

7. Advanced Topics

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- **Connectomics**: Human Connectome Project's insights into network disruptions in autism.
- **Gut-Brain Axis**: Microbiota's role in serotonin synthesis (Cryan & Dinan, 2012).
- **Neuroethics**: Deep Brain Stimulation (DBS) and identity alteration in Parkinson's patients.

8. Critical Debates

- **Nature vs. Nurture**: Gene-environment interplay (Caspi's 5-HTTLPR × stress study).
- **Free Will**: Libet's experiments challenging conscious volition.

9. Recent Research Trends

- **AI in Neuroscience**: Predictive modeling of depression using machine learning.
- **Neuroinflammation**: Microglial activation in bipolar disorder (meta-analysis by Mondelli et al., 2017).

10. References for MPhil Scholars

- **Primary Literature**:
  - Kandel, E. R. (2013). *Principles of Neural Science* (6th ed.).
  - Nestler, E. J. (2021). *Molecular Neuropharmacology*.

Enhancements for MPhil Rigor:

1. **Critical Analysis**:
   - Compare competing theories (e.g., monoamine vs. neuroplasticity hypotheses of depression).
2. **Methodological Depth**:
   - Discuss limitations of EEG (poor spatial resolution) vs. MEG.
3. **Clinical Integration**:
   - Link basal ganglia dysfunction to OCD's CSTC loop abnormalities.
4. **Ethical Considerations**:
   - Debate neuroenhancement (e.g., Adderall use in healthy individuals).

History of Biopsychology (a.k.a. Biological Psychology or Behavioral Neuroscience)

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Biopsychology explores how biology—particularly brain function, neurotransmitters, and genetics—affects behavior, thoughts, and emotions. The field integrates psychology, neuroscience, biology, and even evolutionary theory.

📜 1. Ancient Foundations (Before 500 BCE)

- **Egyptians**: Believed the heart, not the brain, controlled thoughts and emotions.
- **Trepanation**: Evidence from skulls suggests early surgical intervention for mental disorders (drilling into the skull).
- **Hippocrates (460–370 BCE)**: First to suggest the **brain is the seat of thought and emotion**. Proposed the **humoral theory** (imbalance of bodily fluids leads to mental illness).

2. Classical Greece & Rome (500 BCE – 200 CE)

- **Plato**: Argued the **soul resides in the brain**.
- **Aristotle**: Believed the **heart** was the center of mental processes.
- **Galen (129–199 CE)**: Roman physician who dissected animals; proposed that **nerves carry messages to and from the brain** through "animal spirits".

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3. Renaissance & Enlightenment (1500–1800)

- **Descartes (1596–1650)**: Introduced **dualism** – mind and body are separate but interact (via the **pineal gland**).
- **Thomas Willis**: Conducted early brain dissections, coined the term “**neurology**”.
- Advances in **microscopy** led to the discovery of **neurons** and brain structure.

4. 19th Century: The Birth of Scientific Biopsychology

Key Milestones:

- **Phrenology (Franz Gall)**: Idea that skull shape reflects mental faculties. Discredited, but it promoted **localization of function**.
- **Broca's Area** (Paul Broca, 1861): Identified brain region for speech production.
- **Wernicke's Area** (Carl Wernicke, 1874): Area for speech comprehension.
- **Hermann von Helmholtz**: Measured **neural conduction speed**, proving nerves use electrical signals.
- **Charles Darwin** (1859): Evolutionary theory influenced biopsychology; behavior as an adaptation.

5. Early 20th Century: The Rise of Neuroscience

- **Santiago Ramón y Cajal**: Identified the **neuron** as the fundamental unit of the nervous system; father of modern neuroscience.
- **Sherrington**: Coined the term **"synapse"**, studied reflexes.
- **Pavlov**: Classical conditioning showed learning could be studied biologically.
- **John B. Watson & B.F. Skinner**: While behaviorists avoided brain study, their work on observable behavior influenced biopsych experiments.

6. Mid 20th Century: Neurotransmitters & Brain Mapping

- Discovery of neurotransmitters like **dopamine, serotonin, acetylcholine**.

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- **Electroencephalogram (EEG)** used to study brain activity.
- **Karl Lashley**: Studied learning and memory in rats (search for the "engram").
- **Penfield's brain stimulation studies** during epilepsy surgeries mapped brain function.
- Split-brain studies (Roger Sperry, Michael Gazzaniga) revealed **hemispheric specialization**.

7. Late 20th Century: Cognitive Neuroscience Emerges

- **Neuroimaging** (fMRI, PET, CT) revolutionized understanding of living brain activity.
- The **biopsychosocial model** integrated biology with psychology and social factors.
- Studies on **neuroplasticity**, showing the brain can change with experience.
- **Biological basis of psychiatric disorders** became central (e.g., schizophrenia, depression, dementia).

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8. 21st Century: Genetics, Connectomics & AI

- **Human Genome Project**: Unlocked genetic contributions to behavior and mental illness.
- **Epigenetics**: Explores how the environment affects gene expression.
- **Connectome mapping**: Mapping the brain's neural connections.
- **AI and machine learning** are used to model brain functioning.
- Focus on **mental health**, neurodevelopmental disorders, and **aging-related diseases** (e.g., dementia, as in your PDF

cases).

Key Areas Today in Biopsychology:

Domain	Focus
Neuroanatomy	Structure of the brain and nervous system
Neurochemistry	Role of neurotransmitters
Neuroendocrinology	Hormonal influences on behavior
Behavioral Genetics	Heredity and behavior
Neuropsychology	Brain-behavior relationship via lesions, strokes, etc.
Psychopharmacology	How drugs affect mood, behavior, cognition

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Integration With Other Fields

- **Clinical Psychology**: Understanding dementia, schizophrenia, mood disorders.
- **Cognitive Science**: Brain basis of memory, language, perception.

- **Artificial Intelligence**: Brain-inspired computing models.
- **Health Psychology**: Brain-body connection in stress and chronic illness.

Evolution of Key Concepts

Period	Focus	Key Contributors
Ancient	Mind-body concepts	Hippocrates, Plato, Galen
Renaissance	Dualism, brain anatomy	Descartes, Willis
19th Century	Localization, reflexes	Broca, Wernicke, Gall
20th Century	Neural basis of behavior	Pavlov, Penfield, Cajal, Lashley
21st Century	Genetics, AI, imaging	Gazzaniga, Human Genome researchers

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Localization vs. Holism

1. Localization of Function

Definition:
The idea that **specific brain areas are responsible for specific functions** (e.g., speech, vision, motor control).

🔍 Key Features:

- Brain functions are **compartmentalized**.
- Damage to a particular area causes **specific deficits**.
- Focus on **structure-function mapping**.

Key Evidence:

- **Broca's area**: Speech production (damage → expressive aphasia)
- **Wernicke's area**: Language comprehension (damage → receptive aphasia)
- **Phineas Gage case**: Frontal lobe damage → personality changes
- **Penfield's stimulation studies**: Identified **motor and sensory cortex maps**
- **fMRI scans**: Show region-specific activity during tasks (e.g., visual cortex lights up during visual tasks)

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Advantages:

- Helpful in **neurosurgery**, **rehabilitation**, and **brain imaging**
- Forms basis for **neuropsychological assessment**

Limitations:

- Overlooks **interconnectedness** of brain regions
- Doesn't explain **recovery after injury** (neuroplasticity)

2. Holism (Holistic Theory)

The belief that the **brain functions as a whole**, and that mental functions are **distributed across multiple areas**.

Key Features:

- Brain areas **work together** rather than in isolation.
- Cognitive functions emerge from **network activity**.
- Damage can be compensated by **other regions** (brain plasticity).

Key Evidence:

- **Karl Lashley's experiments**: Memory not localized to one region in rats
- **Equipotentiality**: Any part of the cortex can take over functions of a damaged part (to an extent)
- **Neuroplasticity studies**: Recovery of function after strokes/injury
- **Distributed processing**: E.g., memory involves hippocampus, prefrontal cortex, amygdala, etc.

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Advantages:

- Explains **compensation and adaptation**
- Supports complex processes like **consciousness and emotions** involving networks

Limitations:

- Less precise; can't explain why **damage to specific areas** causes **specific deficits**
- Difficult to use in **surgical/clinical applications**

Comparison Table

Aspect	Localization	Holism
Core Idea	Specific areas control specific functions	Brain works as an integrated whole
Brain Damage Outcome	Predictable deficits based on lesion site	Variable effects; recovery possible
Key Evidence	Broca, Wernicke, Penfield, Gage	Lashley, Neuroplasticity, fMRI networks
Clinical Use	Neurosurgery, diagnostics	Rehabilitation, therapy
View on Plasticity	Less emphasis	Strong emphasis on adaptability
Processing Style	Linear, modular	Network-based, distributed

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Modern View: Integration of Both

Today, neuroscience recognizes that:

-**Some functions are localized** (e.g., vision, language centers)

-**Many functions are network-based and holistic** (e.g., memory, emotions)

The brain is **both specialized and integrated** → **"Distributed Localization"**

Karl Lashley (1890–1958)

Field: Neuropsychology, Behavioral Neuroscience
Known For: Challenging the **localization** theory of brain function

Key Contributions

1. Search for the Engram

- Lashley's lifelong quest was to **find the engram**, the **physical trace of memory** in the brain.
- He trained rats to run mazes and then surgically removed different parts of their **cerebral cortex**.
- He expected that removing a specific area would erase the memory → **did not happen as expected**.

2. Principle of Mass Action

"The cortex works as a whole in complex functions like learning and memory."

- Performance impairment was related more to the **amount of cortex removed** than **specific location**.
- Suggests that **learning and memory are distributed**, not localized.

3. Principle of Equipotentiality

"One part of the cortex can take over functions of another part if needed."

- Brain areas are **not strictly fixed** in function; there's flexibility, especially in **early development** or **after injury**.

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Lashley’s Maze Experiments

Step	Action
1.	Rats trained to run a maze
2.	Lashley surgically removed parts of cortex
3.	Observed effects on maze performance
Result	No one area was solely responsible for memory; larger lesions = worse performance

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Theoretical Impact

Lashley's View	Contrast
Opposed strict localization	Supported by Broca, Wernicke, and Penfield
Emphasized distributed processing	Preceded modern ideas of **neural networks**
Highlighted brain plasticity	Anticipated research in **neuroplasticity** and recovery

Criticisms & Limitations

- His methods (e.g., using rats and mazes) may have been **too simple** to detect **localized memory systems** like the **hippocampus**.
- Later research showed that **some memory functions *are* localized** (e.g., in medial temporal lobes).

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Legacy

- Lashley laid the groundwork for modern concepts of:
  - **Neural networks**
  - **Distributed cognition**
  - **Neuroplasticity**
- Inspired new approaches to **brain injury recovery**, **learning theories**, and **connectome mapping**

Methods of Studying Brain and Behavior

I. Neuroanatomical Methods

These help in **studying the structure** of the brain.

1. Dissection

- Study of brain structure post-mortem.
- Helped early scientists map out brain regions.

Advantage: Simple, gives gross structural details
Limitation: No information about function or activity

2. Histology

- Microscopic study of brain tissue using stains (e.g., Golgi stain, Nissl stain).
- Used to identify **neurons, glial cells**, and their **connections**.

Advantage: High resolution of cellular structure
Limitation: Post-mortem; cannot observe live activity

II. Neurophysiological Methods

These study **electrical activity** of the brain.

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3. EEG (Electroencephalography)

- Measures **electrical activity** of the brain via scalp electrodes.
- Detects brain waves (alpha, beta, delta, etc.)

Advantage: Non-invasive, good **temporal resolution**
Limitation: Poor spatial resolution

Uses: Sleep studies, seizure detection, attention and alertness research

4. ERP (Event-Related Potentials)

- A refined form of EEG measuring **brain's electrical response to specific stimuli**.

Advantage: Tracks time course of processing
Limitation: Needs averaging of many trials

5. Single-cell Recording

- Invasive method; electrode implanted in brain records activity from one neuron.

Advantage: Very precise
Limitation: Mostly done in animals; ethically restricted in humans

III. Neuroimaging Techniques

Used to **visualize the brain's structure and function in vivo**.

6. CT Scan (Computed Tomography)

- X-ray-based imaging to view **brain structure**.

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Advantage: Detects bleeding, tumors, injuries
Limitation: Radiation exposure, low resolution

7. MRI (Magnetic Resonance Imaging)

- Uses magnetic fields to create **detailed structural images**.

Advantage: High-resolution structural detail
Limitation: No functional information; expensive

8. fMRI (Functional MRI)

- Measures brain activity by detecting changes in **blood flow (BOLD signal)**.

Advantage: Non-invasive, shows both **structure & function**
Limitation: Poor temporal resolution, expensive

9. PET Scan (Positron Emission Tomography)

- Uses radioactive tracers to track **brain metabolism or neurotransmitter activity**.

Advantage: Good for studying **functional neurochemistry**
Limitation: Invasive, radiation exposure

IV. Experimental Brain Manipulation Techniques

10. Lesion Studies

- Damaging a specific brain area (surgically or chemically) to study behavior changes.
- *Advantage*: Reveals function of brain regions
  Limitation: Ethical issues, mostly animal-based

11. Electrical Stimulation (e.g., Penfield's studies)

- Stimulating brain areas with electrodes during surgery.
- *Advantage*: Shows functional mapping
  Limitation: Invasive, often only done in epilepsy patients

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12. TMS (Transcranial Magnetic Stimulation)

- Uses magnetic fields to **activate or inhibit** brain regions.

Advantage: Non-invasive, reversible lesioning
Limitation: Limited to surface brain areas

13. Deep Brain Stimulation (DBS)

- Surgically implanted electrodes deliver impulses to treat **Parkinson's, depression**.

Advantage: Therapeutic use
Limitation: Invasive, risk of complications

V. Neurochemical and Genetic Methods

14. Neurochemical Analysis

- Examining levels of neurotransmitters, hormones, etc. (e.g., dopamine, serotonin).

Advantage: Understands chemical basis of behavior
Limitation: Often requires invasive sampling (e.g., CSF, blood)

15. Twin and Adoption Studies

- Used to determine **hereditary basis of behavior**.

Advantage: Estimates genetic vs environmental influence
Limitation: Cannot identify specific genes

16. Molecular Genetics / GWAS

- Studies how **specific genes** influence behavior (e.g., genes related to schizophrenia, depression).

Advantage: Pinpoints biological roots of disorders
Limitation: Complex interaction of multiple genes and environment

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Approaches in Biopsychology

Biopsychology explores how the brain, neurotransmitters, genes, and biological systems influence **behavior**, **emotion**, and **cognition**. Several **key approaches** are used to understand these relationships.

🔹 1. Physiological (Neuroscience) Approach

Focus:

- Direct study of **brain structure and function**
- Relationship between **neuronal activity** and behavior

Methods:

- EEG, fMRI, brain lesions, electrical stimulation

Example:

- Investigating how damage to the **hippocampus** affects memory

Key Theorists:

- Penfield, Lashley, Sperry

🔹 2. Neurochemical Approach

Focus:

- Role of **neurotransmitters and hormones** in influencing behavior

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Key Elements:

- Dopamine, serotonin, GABA, norepinephrine
- Hormones like cortisol, oxytocin

Example:

- Low serotonin levels linked to depression
- Dopamine dysregulation in schizophrenia

Methods:

- PET scans, pharmacological studies, blood/CSF analysis

3. Genetic Approach

Focus:

- How **genes and heredity** influence behavior and mental disorders

Key Concepts:

- Heritability, genotype vs phenotype, gene-environment interaction

Methods:

- Twin studies, adoption studies, molecular genetics (e.g., GWAS)

Example:

- Studying genetic predisposition for bipolar disorder or ADHD

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4. Evolutionary Approach

Focus:

- How behavior evolved to increase **survival and reproductive success**

Key Concepts:

- Natural selection, adaptation, evolutionary fitness

Example:

- Fear of snakes as an evolutionary adaptive behavior
- Altruism in kin selection

Key Theorists:

- Charles Darwin, David Buss

5. Neuropsychological Approach

Focus:

- Effects of **brain damage or abnormalities** on psychological functions

Methods:

- Case studies (e.g., Phineas Gage), neuropsychological tests, lesion studies

Example:

- Damage to **Broca's area** leading to speech production deficits

6. Psychopharmacological Approach

Focus:

- Effects of **drugs on mood, cognition, and behavior**

Example:

- Use of SSRIs (Selective Serotonin Reuptake Inhibitors) to treat depression
- Anti-psychotic medications in schizophrenia

Methods:

- Drug trials, receptor binding studies, animal models

7. Comparative/Ethological Approach

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Focus:

- Comparing **animal behavior and brain systems** to understand human behavior

Key Concepts:

- Homologous structures, instinctive behavior, social behavior in animals

Example:

- Studying aggression in primates to understand human aggression
- Songbird studies in learning and memory

Summary Table

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Approach	Focus	Methods	Example
Physiological	Brain structures & functions	EEG, MRI, lesions	Memory and hippocampus
Neurochemical	Neurotransmitters, hormones	PET, blood tests	Serotonin in depression
Genetic	Heredity & genes	Twin studies, GWAS	Bipolar inheritance
Evolutionary	Adaptive value of behavior	Cross-species comparison	Phobias and survival
Neuropsychological	Brain damage & deficits	Case studies, tests	Broca's aphasia
Psychopharmacological	Drug-behavior relationship	Drug trials	SSRI use in anxiety
Comparative	Animal-human similarities	Behavioral observation	Learning in rats

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Integrated View:

Modern biopsychology blends all these approaches. For example:

- Schizophrenia may be studied through **genetic predisposition**, **dopamine imbalance**, **structural brain differences**, and **drug response**.

Mind-Brain Problem

Core Question:

What is the relationship between the **mind** (mental processes: thoughts, emotions, consciousness) and the **brain** (physical structure made of neurons and synapses)?

Major Theories Explaining the Mind-Brain Problem

1. Dualism (Mind and Brain are Separate)

Key Thinker: René Descartes (1641)

"The mind is a non-physical substance that interacts with the physical body."

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Types:

- **Substance Dualism**: Mind and brain are two separate substances.
- **Property Dualism**: Mind is a property (like consciousness) that emerges from brain processes.
- *Strengths*: Explains subjective experiences, free will
  Criticism: How can a non-physical mind affect a physical brain? Lacks scientific testability

2. Materialism / Physicalism (Mind is the Brain)

Core Idea:

Mental processes are entirely **products of physical brain activity**.

Types:

- **Identity Theory**: Each mental state corresponds to a specific brain state
  e.g., Pain = activation of C-fibers
- **Eliminative Materialism**: Common-sense mental concepts (e.g., "beliefs") will be eliminated and replaced by neurobiology
- **Functionalism**: Mental states are defined by their **function**, not structure. (like software run on hardware)
- *Strengths*: Supported by neuroscience and brain imaging
  Criticism: May ignore subjective experience (qualia)

3. Interactionism

- Mind and brain **influence each other** (Descartes' pineal gland theory is one version).
- Popular in clinical psychology where **thoughts can change brain chemistry** (e.g., via CBT).

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4. Epiphenomenalism

- The mind is a **byproduct** of brain activity (like steam from an engine).
- Mental states **do not affect** physical brain processes.
- Criticism: Ignore how therapy, beliefs, and intentions can change behavior and biology.

5. Parallelism

- Mental and physical processes run in **parallel**, pre-determined harmony (Leibniz).

- They do not interact but appear to correlate.
- Criticism: Lacks scientific basis.

6. Emergentism

- The mind **emerges** from complex brain activity.
- Consciousness cannot be reduced to simple neural activity but arises from **system-level complexity**.
- Supported by modern cognitive neuroscience

Modern Neuroscientific View (Monism with Complex Layers)

- **Consciousness, emotion, and thought** are seen as emergent properties of **neural networks**.
- Technologies like **fMRI, EEG, and brain mapping** support the idea that:
  "The mind is what the brain does."

Summary Table

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Theory	Mind-Body Relationship	Strengths	Weaknesses
Dualism	Separate substances	Preserves free will	Hard to test scientifically
Identity Theory	Same thing	Neurological support	Ignores subjectivity
Functionalism	Based on function, not structure	AI-friendly	Doesn't explain qualia
Epiphenomenalism	Mind is a byproduct	Simple explanation	Denies causal power of thought
Emergentism	Mind emerges from complexity	Matches modern brain science	Still developing empirically

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I. Macrostructure of the Nervous System

1. Central Nervous System (CNS)

- **Composed of**: The **brain** and **spinal cord**
- **Function**:

1. Integration and interpretation of sensory input
2. Generation of motor output

3. Cognitive, emotional, and executive functions

A. Brain

Divided into **major regions**:

Region	Components	Major Functions
Forebrain (Prosencephalon)	Cerebrum, thalamus, hypothalamus	Sensory perception, cognition, emotion, voluntary action, autonomic regulation
Midbrain (Mesencephalon)	Tectum, tegmentum	Visual/auditory reflexes, motor relay
Hindbrain (Rhombencephalon)	Pons, medulla, cerebellum	Motor coordination, vital functions (e.g., respiration, cardiovascular regulation)

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Cerebral Cortex: Six-layered neocortex involved in higher-

order functions (planning, decision-making, language, abstract thought)

B. Spinal Cord

- Extends from the medulla to the lumbar region
- **Gray matter (central)**: Neuronal cell bodies
- **White matter (peripheral)**: Myelinated axons

Functions:

- Reflex arcs
- Conduction pathway between brain and body

2. Peripheral Nervous System (PNS)

All neural tissue **outside the CNS**

A. Somatic Nervous System (SNS)

- **Voluntary control**
- Innervates **skeletal muscles**
- Sensory input from skin, muscles, joints

B. Autonomic Nervous System (ANS)

- **Involuntary control**
- Regulates **visceral functions**

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Subsystem	Neurotransmitters	Effects
Sympathetic	Norepinephrine, Epinephrine	Mobilizes energy (↑HR, ↑BP, pupil dilation)
Para sympathetic	Acetylcholine	Conserves energy (↓HR, digestion, pupil constriction)
Enteric	Acetylcholine, Serotonin	Controls gastrointestinal tract independently

II. Microstructure of the Nervous System

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1. Neuron (Functional Unit)

Approximately 86 billion neurons in the human brain

Part	Function
Dendrites	Receive input (graded potentials)
Soma (cell body)	Metabolic center
Axon	Conducts action potentials
Axon hillock	Site of action potential initiation
Myelin sheath	Increases conduction speed (saltatory conduction)
Axon terminals	Neurotransmitter release into synapse

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2. Glial Cells (Support Cells)

Type	Function
Astrocytes	Blood-brain barrier, neurotransmitter recycling
Oligodendrocytes (CNS) / Schwann cells (PNS)	Myelination
Microglia	Immune defense, phagocytosis
Ependymal cells	Line ventricles, produce cerebrospinal fluid (CSF)

III. Functional Organization

1. Neural Circuits

- Involve **excitatory and inhibitory pathways**
- **Feedforward** and **feedback inhibition** regulates activity
- Involved in reflexes, sensory processing, cognition

2. Sensory Systems

- **Transduction**: Converting physical energy into neural signals (e.g., phototransduction in retina)
- Pathways involve **relay nuclei** in the thalamus before reaching cortex

3. Motor Systems

- **Pyramidal system**: Voluntary motor control via corticospinal tract

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- **Extrapyramidal system**: Involuntary movements, posture (e.g., basal ganglia)

IV. Higher Integrative Functions

1. Limbic System

- Involves amygdala, hippocampus, cingulate gyrus
- **Functions**: Emotion regulation, memory consolidation, motivation

2. Prefrontal Cortex

- **Executive functions**: Working memory, decision-making, inhibition, emotional regulation

3. Language Areas

Area	Function	Lesion Result
Broca's area (left inferior frontal gyrus)	Speech production	Expressive aphasia
Wernicke's area (left superior temporal gyrus)	Language comprehension	Receptive aphasia

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V. Neurochemical Systems

1. Major Neurotransmitters

Neurotransmitter	Function	Associated Disorders
Acetylcholine (ACh)	Learning, memory, neuromuscular junction	Alzheimer's disease
Dopamine (DA)	Reward, motor control	Parkinson's, schizophrenia
Serotonin (5-HT)	Mood, sleep, appetite	Depression, OCD
Norepinephrine (NE)	Arousal, attention	Anxiety disorders
GABA	Main inhibitory NT	Epilepsy, anxiety
Glutamate	Main excitatory NT	Excitotoxicity, stroke

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VI. Developmental and Plasticity Aspects

- **Neurogenesis**: Mostly prenatal, but some adult neurogenesis (e.g., hippocampus)
- **Synaptic pruning**: Elimination of unused synapses in development

- **Neuroplasticity**: Functional reorganization after damage or learning (e.g., therapy-induced cortical re-mapping in stroke patients)

Human Brain: Structure and Function (Advanced – M.Phil Level)

I. Overview of the Brain

- The human brain weighs ~1.4 kg and contains ~86 billion neurons.
- It is the central organ of the **Central Nervous System (CNS)**, protected by the **skull, meninges**, and **cerebrospinal fluid (CSF)**.
- Organized into **hierarchical systems** — from molecular (neurotransmitters) to systems (cognitive networks).

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II. Major Divisions of the Brain

1. Forebrain (Prosencephalon)

Subdivided into:

A. Telencephalon

- **Cerebral Cortex**
  - Six-layered neocortex (90% of cortex), critical for **conscious thought, language, planning, memory**.
  - Divided into **lobes** (see below).
- **Basal Ganglia**
  - Includes caudate nucleus, putamen, globus pallidus

    - Involved in **voluntary motor control**, habit learning, reward processing
    - Dysfunction → **Parkinson's disease, Huntington's, OCD**
- **Limbic System**
    - Amygdala: Emotional processing, fear, aggression
    - Hippocampus: Memory consolidation and spatial navigation
    - Cingulate gyrus: Emotional-cognitive integration
    - Septum, mammillary bodies: Reward, memory

B. Diencephalon

- **Thalamus**: Relay station for all sensory information (except smell)
- **Hypothalamus**: Regulates autonomic system, endocrine control (via pituitary), circadian rhythms, hunger, thermoregulation
- **Epithalamus**: Contains pineal gland (melatonin secretion)

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2. Midbrain (Mesencephalon)

- **Tectum**: Includes superior and inferior colliculi (visual and auditory reflexes)
- **Tegmentum**: Includes substantia nigra and red nucleus (motor control, dopamine production)
- **Cerebral peduncles**: Major motor pathways

3. Hindbrain (Rhombencephalon)

A. Metencephalon

- **Pons**: Relay between cerebrum and cerebellum, sleep and arousal regulation

- **Cerebellum**: Coordinates movement, balance, procedural learning, cognitive timing

B. Myelencephalon

- **Medulla Oblongata**: Controls **vital autonomic functions** (breathing, heartbeat, blood pressure)

III. Lobes of the Cerebral Cortex

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Lobe	Functions	Brodmann Areas	Lesion Effects
Frontal	Executive function, motor planning, speech (Broca), inhibition	BA 4, 6, 8, 9, 10, 44, 45	Personality change, apraxia, Broca's aphasia
Parietal	Somatosensory processing, spatial attention	BA 1, 2, 3, 5, 7, 39, 40	Hemispatial neglect, tactile agnosia
Temporal	Auditory processing, language comprehension (Wernicke), memory	BA 22, 41, 42, 20, 21, 37	Wernicke's aphasia, auditory agnosia, anomia
Occipital	Visual processing	BA 17 (V1), 18, 19	Visual field deficits, visual agnosia

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IV. Functional Systems in the Brain

1. Motor System

- **Primary Motor Cortex (BA 4)**: Initiates voluntary movements
- **Premotor and Supplementary Areas**: Planning movement
- **Cerebellum and Basal Ganglia**: Modulate fine motor control and motor learning
- **Corticospinal and Extrapyramidal Tracts**: Pathways for descending motor signals

2. Sensory Systems

- **Somatosensory Cortex (BA 1, 2, 3)**: Touch, pressure, proprioception
- **Visual Cortex (BA 17–19)**: Primary and associative vision
- **Auditory Cortex (BA 41, 42)**: Tonotopic mapping of sound
- **Olfactory Bulb & Cortex**: Unique — does not relay through thalamus
- **Insular Cortex**: Interoception, taste, disgust processing

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3. Language System

- **Broca's Area (BA 44, 45)**: Speech production, grammar
- **Wernicke's Area (BA 22)**: Language comprehension
- **Arcuate Fasciculus**: Connects Broca and Wernicke — damage → conduction aphasia

4. Memory System

- **Declarative Memory**: Hippocampus, entorhinal cortex
- **Procedural Memory**: Basal ganglia, cerebellum

- **Working Memory**: Dorsolateral prefrontal cortex

5. Emotion & Motivation

- **Amygdala**: Emotional salience, fear conditioning
- **Orbitofrontal cortex**: Emotional regulation, social behavior
- **Hypothalamus**: Drives (hunger, sex), homeostasis
- **Mesolimbic dopamine system**: Reward and reinforcement (VTA → Nucleus Accumbens)

V. Neurochemical Modulation

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System	Neurotransmitter	Origin	Function	Related Disorder
Cholinergic	Acetylcholine	Basal forebrain	Learning, memory	Alzheimer’s
Dopaminergic	Dopamine	Substantia nigra, VTA	Movement, reward	Parkinson’s, addiction
Noradrenergic	Norepinephrine	Locus coeruleus	Attention, arousal	PTSD, anxiety
Serotonergic	Serotonin	Raphe nuclei	Mood, sleep	Depression
GABAergic	GABA	Cortex, basal ganglia	Inhibition	Epilepsy
Glutamatergic	Glutamate	Widespread	Excitation, plasticity	Excitotoxicity in stroke

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VI. Clinical-Neuropsychological Correlates

Syndrome/Disorder	Affected Brain Region	Description
Broca's Aphasia	Left inferior frontal gyrus	Non-fluent, effortful speech
Wernicke's Aphasia	Left posterior superior temporal gyrus	Fluent but nonsensical speech
Parkinson's Disease	Basal ganglia (substantia nigra)	Bradykinesia, rigidity, tremor
Alzheimer's Disease	Hippocampus, temporal cortex	Memory loss, disorientation
Hemispatial Neglect	Right parietal lobe	Ignoring one side of space
Phineas Gage Syndrome	Ventromedial prefrontal cortex	Personality, impulsivity changes

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VII. Plasticity and Connectivity

A. Neuroplasticity

- Structural and functional brain changes due to:
  - Learning
  - Injury (e.g., stroke recovery)
  - Environmental stimulation

B. Connectomics

- The study of the **brain's structural and functional connections**
- Technologies like **DTI (diffusion tensor imaging)** and **resting-state fMRI** explore networks such as:
  - **Default Mode Network (DMN)**
  - **Salience Network**
  - **Central Executive Network**

VIII. Summary Key Points

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Aspect	Description
Structural Divisions	Forebrain, midbrain, hindbrain
Cortical Lobes	Frontal, parietal, temporal, occipital
Functional Circuits	Motor, sensory, language, emotion
Neurotransmitters	Modulate cognition, emotion, and behavior
Pathologies	Result from focal or network-level brain dysfunction
Plasticity	Brain's capacity to reorganize across lifespan

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Resting Membrane Potential (RMP)

What is it?

- The **electrical charge difference across the neuronal membrane** when the neuron is at rest.
- Typical RMP in a neuron: **–70 mV** (inside is negative relative to outside)

Ionic Basis:

Ion	Intracellular (↑ inside)	Extracellular (↑ outside)	Effect
K^+ (Potassium)	High	Low	Wants to move out (↓)
Na^+ (Sodium)	Low	High	Wants to move in (↑)
Cl^- (Chloride)	Low	High	Moves in
Proteins (A^-)	High	Nil	Negative charge stays inside

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Mechanisms Maintaining RMP:

1. **Selective Permeability**
   - Membrane is **more permeable to K^+** than Na^+.
   - K^+ leaks out → inside becomes negative.
2. **Sodium-Potassium Pump (Na^+/K^+-ATPase)**
   - Pumps **3 Na^+ out and 2 K^+ in**, using ATP
   - Maintains gradient and negativity inside
3. **Electrochemical Gradient**
   - K^+ wants to leave (concentration gradient) but is pulled back in (electrical gradient)

- Creates a dynamic **equilibrium** (~–70 mV)

II Action Potential (AP)

What is it?

- A **rapid, transient, all-or-none electrical impulse** generated when the membrane potential reaches a **threshold**(around –55 mV).

Phases of Action Potential

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Phase	Description	Ionic Movement
1. Resting State	–70 mV maintained	Na^+/K^+ pump active
2. Depolarization	Threshold reached → Na^+ channels open	**Na^+ influx** → MP becomes positive
3. Peak	+30 to +40 mV	Na^+ channels inactivate
4. Repolarization	K^+ channels open	**K^+ efflux** → MP becomes negative
5. Hyperpolarization	MP dips below –70 mV	K^+ channels slow to close
6. Return to Rest	RMP restored	Na^+/K^+ pump restores gradient

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Graph Representation

A typical AP graph shows:

- **Rising phase (Na^+ in)**
- **Falling phase (K^+ out)**
- **Undershoot (hyperpolarization)**
- **Return to baseline**

Properties of Action Potential

Property	Description
All-or-None	If the threshold is reached, AP fires fully. If not, no AP.
Unidirectional	AP moves one-way due to the refractory period.
Non-decremental	AP maintains amplitude along the axon.
Refractory Period:	Time during which no or limited AP can occur.
Absolute Refractory:	No AP possible (Na^+ channels inactivated)
Relative Refractory	Requires stronger stimulus (membrane still hyperpolarized)

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Saltatory Conduction (in Myelinated Axons)

- AP **jumps** from one **Node of Ranvier** to another
- Increases **speed** and **efficiency**

Summary Table: Resting vs. Action Potential

Feature	Resting Potential	Action Potential
Typical Value	–70 mV	+30 to +40 mV
Ion Involved	K^+ dominant	Na^+ (in), then K^+ (out)
Active Process?	Yes (Na^+/K^+ pump)	Yes (voltage-gated channels)
Function	Maintains readiness	Neural signaling
Energy Use	ATP-dependent	Passive channels (once triggered)

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Concept Integration

- **Synaptic Transmission**: AP leads to **neurotransmitter release** at axon terminals
- **Neurotransmitters** then bind to receptors on next neuron → graded potentials → possibly another AP
- Forms the **electrochemical basis of brain function**

Synaptic Transmission

Synaptic transmission is the process by which a **neuron communicates with another neuron or target**

cell(muscle/gland) via **chemical or electrical signals** at a synapse.

Types of Synapses

Type	Description
Chemical Synapse	Involves **neurotransmitters**; most common
Electrical Synapse	Involves **direct ionic current** through gap junctions; faster but less flexible

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Steps in Chemical Synaptic Transmission

1. **Action Potential Arrival**
    - Electrical impulse reaches **axon terminal** of presynaptic neuron
2. **Calcium Influx**
    - Voltage-gated **Ca^{2+} channels open**, allowing Ca^{2+} to enter terminal
3. **Neurotransmitter Release**

- Ca^{2+} causes **synaptic vesicles** to fuse with membrane and release neurotransmitters into **synaptic cleft**

4. **Receptor Binding**
    - Neurotransmitters bind to **specific receptors** on the postsynaptic membrane
5. **Postsynaptic Response**
    - **Excitatory (EPSP)** or **Inhibitory (IPSP)** potentials are generated depending on the neurotransmitter and receptor type
6. **Termination**
    - Neurotransmitter action is ended by:
        - **Reuptake** (e.g., serotonin reabsorbed by presynaptic cell)
        - **Enzymatic degradation** (e.g., acetylcholine broken down by AChE)
        - **Diffusion away** from the synaptic cleft

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Types of Receptors

Type	Characteristics
Ionotropic	Fast-acting, direct ion channel opening (e.g., GABA-A)
Metabotropic	Slower, involves second messengers (e.g., dopamine D1 receptors)

Major Neurotransmitters and Functions

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Neurotransmitter	Function
Acetylcholine (ACh)	Muscle movement, memory
Dopamine	Reward, movement, motivation
Serotonin (5-HT)	Mood, sleep, appetite
GABA	Main inhibitory neurotransmitter
Glutamate	Main excitatory neurotransmitter
Norepinephrine	Arousal, alertness

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Neural Coding

Neural coding refers to the way **neurons represent information** (stimuli, sensations, decisions) through their **electrical activity** (spike patterns).

Types of Neural Coding

1. Rate Coding (Frequency Coding)

- **Definition**: Information is encoded in the **firing rate** (number of spikes per second) of a neuron.
- **Example**: Stronger stimulus (e.g., louder sound) → higher firing rate
- **Most common** form in many brain areas

2. Temporal Coding (Time Coding)

- **Definition**: Information is encoded in the **precise timing** of spikes or **synchrony** across neurons.
- Useful for:
  - **Fast sensory processing** (e.g., auditory system)
  - **Oscillatory patterns** (like theta, gamma waves)

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3. Population Coding

- **Definition**: A group of neurons together represent information; the **pattern of activity across multiple neurons** is important.
- Example: Visual system encodes object location or orientation using many neurons

4. Sparse Coding

- **Definition**: Only a small subset of neurons is active at a given time to represent specific information
- Seen in **olfactory** and **visual** systems
- Energy-efficient, improves storage capacity

5. Place Coding

- **Definition**: Information is encoded based on **which neuron** is active (its location)
- Example: Tonotopic map in auditory cortex (different neurons for different sound frequencies)

Key Concepts

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Type	What is Encoded	Example Use
Rate Coding	Spike frequency	Touch pressure, pain intensity
Temporal Coding	Precise timing of spikes	Auditory signals, rhythm detection
Population Coding	Pattern across many neurons	Motor control, perception
Sparse Coding	Few active neurons for efficient coding	Visual object recognition
Place Coding	Spatial location of neural activation	Sound frequency (cochlea)

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Why It Matters

- Neural coding explains how the brain **represents sensory input, plans actions**, and **forms memories**
- It's foundational for fields like **neuroinformatics**, **brain-machine interfaces**, and **computational neuroscience**

Neuroplasticity

Neuroplasticity is the brain's ability to **change its structure and function** in response to experience, learning, or injury.

Types of Neuroplasticity

1. **Structural Plasticity**

- Changes in the **physical structure** of neurons (e.g., dendrites, axons, synapses)
- Includes **synaptogenesis** (formation of new synapses) and **synaptic pruning** (elimination of weak/unused synapses)

2. **Functional Plasticity**

- Changes in the **strength** or efficiency of synaptic connections
- Example: **Long-Term Potentiation (LTP)** — long-lasting increase in synaptic strength, important for learning and memory

Mechanisms

- **Synaptogenesis**: Formation of new synapses between neurons
- **Synaptic pruning**: Removal of excess synapses to improve efficiency
- **Long-Term Potentiation (LTP)**: Strengthening of synapses through repeated activation
- **Neurogenesis** (limited): Formation of new neurons, mainly in hippocampus

Role in

- **Learning and memory**
- **Recovery from brain injury** (e.g., stroke)
- **Adaptation to environmental changes**

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Factors Influencing Neuroplasticity

- Age (more plastic in early life)
- Experience and learning
- Environmental stimulation
- Physical exercise
- Stress and injury

Clinical Relevance

- Rehabilitation after brain injury depends on neuroplasticity
- Mental health disorders (e.g., depression) may involve impaired plasticity
- Neuroplasticity underlies effectiveness of therapies like cognitive behavioral therapy (CBT)

Myelination and Saltatory Conduction

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1. Myelination

Definition:
Myelination is the process of covering axons with a **fatty insulating sheath called myelin**, produced by:

- **Oligodendrocytes** in the **central nervous system (CNS)**
- **Schwann cells** in the **peripheral nervous system (PNS)**

Function:

- **Increases speed** of electrical signal (action potential)
- **Prevents signal loss**

- **Conserves energy** by reducing need for ion exchange along the entire axon

2. Nodes of Ranvier

- Small **gaps between myelin sheaths** along the axon
- Rich in **voltage-gated Na^+ and K^+ channels**
- Action potentials are **regenerated** only at these nodes

3. Saltatory Conduction

Definition:
Saltatory conduction is the **jumping of action potentials** from one Node of Ranvier to the next, along a **myelinated axon**

Key Benefits:

- **Much faster** than continuous conduction (in unmyelinated axons)
- **More energy-efficient**, as ion exchange is limited to nodes

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Comparison: Myelinated vs Unmyelinated Axons

Feature	Myelinated Axons	Unmyelinated Axons
Conduction speed	Fast (saltatory)	Slow (continuous)
Energy use	Low	High
Signal reliability	High	Lower

Clinical Relevance

- **Multiple Sclerosis (MS)**: Autoimmune disorder where myelin in CNS is damaged → leads to slower conduction, motor and cognitive deficits
- **Guillain-Barré Syndrome**: Demyelination in PNS → weakness, numbness

Role of Neuroplasticity in Learning, Memory, and Recovery

1. Learning

- Neuroplasticity allows the brain to **adapt to new experiences** by forming and strengthening neural connections.
- **Synaptogenesis** increases the number of synapses between neurons, facilitating new information processing.
- **Long-Term Potentiation (LTP)** strengthens synaptic connections through repeated stimulation, enhancing signal transmission.
- **Functional reorganization** of brain areas can occur to support acquisition of new skills.

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2. Memory

- Memory formation relies heavily on **synaptic plasticity**, especially LTP, which stabilizes and strengthens synaptic connections.
- Different types of memory (e.g., declarative, procedural) involve plastic changes in different brain regions like the **hippocampus** and **cortex**.

- **Synaptic pruning** helps refine memory networks by removing unnecessary connections, improving efficiency.

3. Recovery Post-Injury

- After brain injury (e.g., stroke, trauma), neuroplasticity supports **functional recovery** by:
    - **Rewiring** undamaged neurons to take over lost functions
    - Enhancing **synaptogenesis** and strengthening existing pathways
- Rehabilitation therapies leverage neuroplasticity to promote **re-learning** and regain abilities.
- Plasticity decreases with age, making early intervention critical.

Neurotransmitters

Neurotransmitters are chemical messengers released by neurons at synapses to **transmit signals** to other neurons, muscles, or glands.

Key Characteristics

- Stored in **synaptic vesicles**
- Released into **synaptic cleft** upon action potential
- Bind to **specific receptors** on the postsynaptic membrane
- Can be **excitatory** or **inhibitory**

Function

- Fast-acting chemical communication

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- Crucial for **brain function and behavior**
- Imbalances lead to **neurological and psychiatric disorders**

Neuromodulators

Neuromodulators are chemicals released by neurons that **do not directly cause excitatory or inhibitory postsynaptic potentials**, but instead **modulate** the activity of other neurotransmitters and influence the overall excitability of neurons.

Key Characteristics

- Released in **larger quantities**
- Affect a **broader area** and act on **multiple neurons**
- Effects are **slower** but **longer-lasting** than neurotransmitters
- Regulate **mood, arousal, attention, motivation, and plasticity**

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Common Neuromodulators

(*Some act as both neurotransmitters and neuromodulators*)

- **Dopamine:** Modulates reward, motivation, motor control
- **Serotonin (5-HT):** Modulates mood, aggression, appetite, sleep
- **Norepinephrine:** Enhances attention, stress response
- **Acetylcholine:** Modulates arousal, attention, learning

- **Endorphins:** Natural opioids that modulate pain and pleasure
- **Substance P:** Involved in pain transmission
- **Oxytocin and Vasopressin:** Modulate social behavior and bonding

Functional Role

- Adjust the **sensitivity of neurons** to other neurotransmitters
- Influence **neural circuits**, often in emotion, memory, and motivation
- Essential for **homeostasis**, **behavioral regulation**, and **adaptive responses**

Clinical Significance

- Imbalances in neuromodulators are implicated in **depression, anxiety, schizophrenia, ADHD, Parkinson's disease**, etc.
- **Psychotropic drugs** (e.g., SSRIs, antipsychotics) often target neuromodulatory systems.

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Sensory and Perceptual Systems

- **Sensory systems** detect environmental stimuli and convert them into neural signals.
- **Perception** is the brain's interpretation of sensory input — it involves **integration, interpretation, and conscious experience**.

Major Sensory Modalities & Systems

Modality	Sensory Organ	Cortex/Area
Vision	Eye (retina)	Occipital lobe (Visual cortex)
Audition	Ear (cochlea)	Temporal lobe (Auditory cortex)
Touch	Skin	Parietal lobe (Somatosensory cortex)
Olfaction	Nose (olfactory epithelium)	Olfactory bulb & cortex
Gustation	Tongue (taste buds)	Insular cortex & frontal operculum
Vestibular	Inner ear (semicircular canals)	Brainstem & cerebellum

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Sensory Processing Pathways (Simplified)

- **Stimulus → Receptors → Neural signal → Thalamus (except smell) → Primary Sensory Cortex → Association Areas → Perception**

Perception Features

- **Feature Detection:** Neurons tuned to specific attributes (e.g., edges, motion)
- **Multisensory Integration:** Combining input from multiple senses for coherent perception
- **Top-Down Processing:** Expectations and prior knowledge influence perception
- **Bottom-Up Processing:** Perception driven by sensory input alone

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Key Brain Areas

- **Primary Sensory Cortices** (e.g., V1 for vision, A1 for sound, S1 for touch)
- **Association Cortices**: Integrate and interpret multisensory information
- **Thalamus**: Relay station for most senses (except olfaction)

Visual Pathway & Disorders

Pathway:
Retina → Optic Nerve → Optic Chiasm → Optic Tract → Lateral Geniculate Nucleus (LGN, thalamus) → Primary Visual Cortex (V1, occipital lobe)

Disorders:

- **Blindsight:**
  - Damage to primary visual cortex (V1)
  - Patient reports no conscious vision but can respond to visual stimuli (due to intact subcortical pathways)
- **Visual Agnosia:**
  - Inability to recognize objects despite normal vision (due to occipito-temporal damage)
- **Hemianopia:**
  - Loss of vision in one half of the visual field (commonly due to damage in optic tract or V1)

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Auditory System & Lateralization

Pathway:
Cochlea → Auditory Nerve → Brainstem (Cochlear nuclei, superior olive) → Inferior Colliculus → Medial Geniculate Nucleus (thalamus) → Primary Auditory Cortex (A1, temporal lobe)

Lateralization:

- Left hemisphere: Processes **language-related sounds** (speech)
- Right hemisphere: Processes **music, tone, pitch**
- Auditory input from each ear goes to **both hemispheres**, but with stronger contralateral projections

Disorders:

- **Wernicke's Aphasia:** Impaired language comprehension (left temporal damage)
- **Central Auditory Processing Disorder (CAPD):** Difficulty processing auditory information despite normal hearing

Somatosensory System

Pathways:

- **Touch & Proprioception:** Dorsal column-medial lemniscal pathway
- **Pain & Temperature:** Spinothalamic tract

Primary Cortex: Postcentral gyrus (parietal lobe)

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Modalities:

- **Touch (mechanoreception):** Pressure, vibration, texture
- **Temperature (thermoreception):** Warm and cold receptors
- **Pain (nociception):** Free nerve endings; fast (Aδ) and slow (C) fibers

Disorders:

- **Analgesia:** Absence of pain sensation
- **Allodynia:** Pain from non-painful stimuli
- **Phantom Limb Pain:** Sensation in amputated limb due to cortical reorganization

Olfactory and Gustatory Processing

Olfaction (Smell):

- **Pathway:** Olfactory receptors → Olfactory bulb → Olfactory cortex (bypasses thalamus) → Limbic system
- **Features:** Strong link to **emotion and memory**

Disorders:

- **Anosmia:** Loss of smell (e.g., after head injury or infection)
- **Hyposmia:** Decreased smell sensitivity

Gustation (Taste):

- **Receptors:** Taste buds (on tongue, soft palate, etc.)
- **Pathway:** Facial (VII), Glossopharyngeal (IX), and Vagus (X) nerves → Brainstem → Thalamus → Gustatory cortex (insula & frontal operculum)

Five Basic Tastes: Sweet, salty, sour, bitter, umami

Disorders:

- **Ageusia:** Loss of taste
- **Dysgeusia:** Distorted taste perception

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Motor Systems and Coordination

Motor systems control **voluntary and involuntary movement**, including **muscle contraction**, **posture**, and **coordination**. They involve both **central** and **peripheral** pathways.

Major Motor Pathways

a. Pyramidal System (Corticospinal Tract)

- Origin: **Primary Motor Cortex (M1)**
- Function: Controls **voluntary, precise, fine motor movements**
- Crosses (decussates) at **medulla (pyramidal decussation)**
- Damage → **Paralysis**, weakness

b. Extrapyramidal System

- Includes: **Basal Ganglia, Cerebellum, Brainstem nuclei**
- Function: **Posture, balance, coordination, automatic movements**
- Damage → **Involuntary movements**, tremors (e.g., Parkinsonism)

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Brain Structures in Motor Control

Structure	Function
Primary Motor Cortex (M1)	Executes voluntary movements
Premotor Cortex & SMA	Plans and sequences movements
Basal Ganglia	Initiation and inhibition of movement
Cerebellum	Coordination, precision, timing, motor learning
Brainstem	Reflexes, posture, locomotion
Spinal Cord	Reflexes and direct motor control via motor neurons

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Cerebellar Function & Disorders

- **Role:** Coordinates movement, corrects errors, balance, motor learning
- **Damage effects:**
  - **Ataxia** – uncoordinated movement
  - **Dysmetria** – inability to judge distance
  - **Intention tremor**

Basal Ganglia & Movement Disorders

- **Structures:** Caudate, Putamen, Globus Pallidus, Substantia Nigra
- **Function:** Regulate motor output, suppress unwanted movement
- **Disorders:**
  - **Parkinson's Disease:** Dopamine loss → tremors, bradykinesia
  - **Huntington's Disease:** Genetic → chorea, personality changes

Motor Units & Neuromuscular Junction

- **Motor Unit:** One motor neuron + all the muscle fibers it innervates
- **Neuromuscular Junction (NMJ):**
  - Uses **acetylcholine**
  - Site of **muscle contraction initiation**

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Reflexes

- **Spinal Reflexes:** Involuntary, rapid (e.g., knee-jerk reflex)
- **Cortical Modulation:** Higher centers can inhibit or modify reflexes
- **Clinical Use:** Reflex testing for spinal cord integrity

Motor Disorders

1. Parkinson's Disease

Cause: Degeneration of **dopaminergic neurons** in the **substantia nigra** (part of basal ganglia)

Neurotransmitter Involved: Dopamine (deficiency)

Symptoms (TRAP):

- **T**remor (resting)
- **R**igidity (muscle stiffness)
- **A**kinesia/Bradykinesia (slowness of movement)
- **P**ostural instability

Cognitive/Emotional: Depression, mild cognitive impairment, executive dysfunction

Treatment:

- **Levodopa (L-DOPA)**, dopamine agonists
- Deep brain stimulation (advanced cases)

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2. Huntington's Disease

Cause: Genetic mutation (CAG repeat expansion in **HTT gene**) → neuronal loss in **striatum** (caudate and putamen)

Inheritance: Autosomal dominant

Symptoms:

- **Chorea** (involuntary, jerky movements)
- Muscle rigidity or dystonia
- Impaired coordination
- **Cognitive decline**, personality changes, psychiatric symptoms

Onset: Typically mid-adulthood (30s–50s)

Treatment: No cure; symptom management with antipsychotics, mood stabilizers

3. Ataxia

Cause: Damage to the **cerebellum** or its pathways (can be genetic, acquired, or due to tumors, stroke, alcohol)

Symptoms:

- Uncoordinated movement
- Poor balance and gait
- Difficulty with fine motor tasks
- **Dysmetria** (impaired distance judgment)
- **Intention tremor** (during movement, not at rest)

Types:

- **Cerebellar ataxia:** Most common
- **Sensory ataxia:** Due to loss of proprioception (e.g., in peripheral neuropathy)
- **Vestibular ataxia:** Due to inner ear or vestibular nerve damage

Treatment: Depends on cause (e.g., vitamin B12 for deficiency, rehab for coordination)

Endocrine System & Neuroendocrinology

- The **endocrine system** uses hormones to regulate body processes.

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● **Neuroendocrinology** studies interactions between the nervous system and endocrine system — especially how the **hypothalamus** controls hormone release.

Major Endocrine Glands

Gland	Location	Main Hormones	Function
Pituitary	Base of brain	ACTH, LH, FSH, GH, Oxytocin	Master gland; regulates other glands
Thyroid	Neck	Thyroxine (T4), Triiodothyronine (T3)	Metabolism, growth, development
Adrenal	Above kidneys	Cortisol, adrenaline, aldosterone	Stress response, BP, metabolism
Gonads	Testes/Ovaries	Testosterone, estrogen, progesterone	Reproduction, sexual development

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Key Hormones & Functions

- **Cortisol:**
  - Released by adrenal cortex during **stress**
  - Increases glucose, suppresses immune function
- **Estrogen & Testosterone:**
  - Regulate **sexual development**, behavior, reproduction
  - Influence **mood**, aggression, libido
- **Oxytocin:**
  - Involved in **bonding, trust, childbirth**, lactation
  - Released by **posterior pituitary**, synthesized in hypothalamus
- **Melatonin:**
  - Regulates **sleep-wake cycles**
  - Secreted by **pineal gland**, influenced by light exposure

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Hormonal Influence on Behavior

Behavior	Hormonal Basis
Stress	Cortisol ↑ in HPA axis activation (hypothalamus–pituitary–adrenal)
Mood	Estrogen/testosterone levels affect mood; low serotonin linked to depression
Aggression	Elevated testosterone linked to increased aggression
Reproduction	LH/FSH regulate ovulation and spermatogenesis; oxytocin in bonding/childbirth

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HPA Axis – Stress Response Pathway

1. **Hypothalamus** releases CRH (corticotropin-releasing hormone)
2. **Pituitary gland** releases ACTH (adrenocorticotropic hormone)
3. **Adrenal cortex** releases **cortisol**
4. Cortisol acts on **brain and body** to manage stress

(Feedback loop regulates levels)

Biological Rhythms and Sleep

Biological Rhythms

- **Circadian rhythms:** ~24-hour cycles (e.g., sleep-wake cycle)
- **Ultradian rhythms:** Cycles shorter than 24 hours (e.g., stages of sleep, hormone secretion)
- **Infradian rhythms:** Cycles longer than 24 hours (e.g., menstrual cycle)

Pineal gland:

- Produces **melatonin**, regulates circadian rhythms
- Secretion influenced by light/dark signals via the suprachiasmatic nucleus (SCN)

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Sleep Stages

- **NREM (Non-Rapid Eye Movement) Sleep:**
  - Stages 1-3 (Stage 3 = deep slow-wave sleep)
  - Physical restoration, memory consolidation
- **REM (Rapid Eye Movement) Sleep:**
  - Dreaming stage, brain activity similar to wakefulness
  - Important for emotional regulation and memory

Theories of Sleep

1. Restorative Theory

- Sleep restores physical and mental functions.
- Repairs tissue, replenishes energy stores, clears metabolic waste from the brain.
- Supports immune system and brain plasticity.

2. Memory Consolidation Theory

- Sleep helps consolidate and stabilize memories.
- NREM sleep aids declarative memory (facts, knowledge).
- REM sleep supports procedural memory (skills, emotional memories).

3. Evolutionary (Adaptive) Theory

- Sleep conserves energy and protects from dangers during inactivity periods.
- Sleep patterns evolved based on environmental threats and food availability.

4. Brain Plasticity Theory

- Sleep supports brain development and neural plasticity.
- Essential for learning, synaptic growth, and pruning.

Sleep Disorders

1. Insomnia

- Difficulty falling asleep, staying asleep, or non-restorative sleep
- Causes: Stress, anxiety, depression, poor sleep habits, medical conditions

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- Effects: Daytime fatigue, impaired concentration, mood disturbances
- Treatment: Sleep hygiene, cognitive-behavioral therapy, medication

2. Narcolepsy

- Excessive daytime sleepiness and sudden sleep attacks
- Symptoms: Cataplexy (sudden muscle weakness), sleep paralysis, hallucinations
- Cause: Dysfunction of **hypocretin (orexin)** system in hypothalamus
- Treatment: Stimulants, antidepressants, lifestyle management

3. Sleep Apnea

- Repeated breathing interruptions during sleep
- Types:
  - **Obstructive Sleep Apnea (OSA):** Airway blockage
  - **Central Sleep Apnea:** Brain fails to signal breathing muscles
- Symptoms: Loud snoring, gasping, daytime sleepiness, headaches
- Treatment: CPAP machine, weight loss, surgery

4. Other Disorders

- **Restless Leg Syndrome:** Urge to move legs, uncomfortable sensations

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- **Parasomnias:** Abnormal behaviors during sleep (sleepwalking, night terrors)

Hunger and Thirst Regulation

1. Hunger Regulation

- **Hypothalamus** is the key brain region controlling hunger and satiety.
- **Lateral Hypothalamus (LH):**
  - Known as the “hunger center”
  - Stimulates eating behavior when activated
- **Ventromedial Hypothalamus (VMH):**
  - Known as the “satiety center”
  - Signals fullness and inhibits eating
- **Other factors influencing hunger:**
  - Blood glucose levels
  - Hormones like **ghrelin** (increases hunger) and **leptin** (signals fullness)
  - Neural signals from the digestive tract

2. Thirst Regulation

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- Controlled primarily by the **hypothalamus**.
- **Osmoreceptors** detect changes in blood osmolarity (concentration of solutes).
- **Decrease in blood volume** or increase in osmolarity triggers thirst.
- **Antidiuretic hormone (ADH)/vasopressin** released to conserve water in kidneys.

3. Interaction with Behavior

- Hunger and thirst drive motivated behaviors essential for survival.
- Disruptions in regulation can lead to disorders like obesity or dehydration.

Aggression and Fear Responses

1. Aggression

- **Amygdala** plays a central role in aggressive behavior.
- **Testosterone** is linked to increased aggression.
- Prefrontal cortex (PFC) helps regulate and inhibit aggressive impulses.

2. Fear

- Fear processing primarily involves the **amygdala**.
- The **amygdala** detects threats and triggers fear responses.
- Prefrontal cortex modulates fear responses, enabling regulation and extinction of fear.

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Emotions and Motivation

1. Emotions

- **Definition:** Complex psychological and physiological states involving feelings, bodily arousal, and behavior.
- **Key Brain Areas:**
  - **Amygdala:** Processes emotions, especially fear and anger.
  - **Prefrontal Cortex (PFC):** Regulates emotions and decision-making.
  - **Insula:** Involved in awareness of bodily states and emotions like disgust.
  - **Hypothalamus:** Coordinates autonomic and hormonal emotional responses.

Limbic System and Emotion

- **Amygdala:** Central to processing emotions, especially fear and aggression.
- **Hypothalamus:** Regulates autonomic and endocrine responses linked to emotions.

Reward Circuits

- **Mesolimbic Dopamine Pathway:**
  - Key neural circuit for reward and motivation.
  - Dopamine release in areas like the **nucleus accumbens** reinforces pleasurable behaviors.
- **Theories of Emotion:**

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- **James-Lange:** Emotion results from physiological reactions.
- **Cannon-Bard:** Emotion and physiological response occur simultaneously.
- **Schachter-Singer:** Emotion arises from physiological arousal plus cognitive interpretation.

2. Motivation

- **Definition:** Internal processes that initiate, direct, and sustain behavior toward goals.
- **Types of Motivation:**
  - **Intrinsic:** Driven by internal rewards (e.g., curiosity).
  - **Extrinsic:** Driven by external rewards (e.g., money, praise).
- **Brain Structures Involved:**
  - **Hypothalamus:** Regulates basic drives (hunger, thirst, sex).
  - **Limbic System (including nucleus accumbens):** Processes reward and reinforcement.
  - **Prefrontal Cortex:** Planning and goal-directed behavior.
- **Motivational Theories:**
  - **Drive Reduction Theory:** Behavior aims to reduce biological drives (e.g., hunger).
  - **Incentive Theory:** Behavior is motivated by external rewards.

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- **Maslow's Hierarchy of Needs:** Motivations range from physiological needs to self-actualization.

Hormones and Behavior (Neuroendocrinology)

1. Endocrine System

- Major glands:
  - **Pituitary:** "Master gland," controls other endocrine glands
  - **Adrenal:** Produces cortisol, adrenaline (stress hormones)
  - **Thyroid:** Regulates metabolism
  - **Gonads (ovaries/testes):** Produce sex hormones (estrogen, testosterone)

2. Hormonal Regulation

- **Stress:** Cortisol released by adrenal glands via HPA axis
- **Sex:** Testosterone and estrogen regulate reproductive behaviors
- **Growth:** Growth hormone from pituitary regulates body growth
- **Metabolism:** Thyroid hormones regulate energy use and metabolism

3. Psychoneuroendocrinology

- Studies how hormones like **cortisol** affect mental health
- Chronic high cortisol linked to anxiety, depression, cognitive impairment

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4. Interaction of Hormones and Neurotransmitters

- Hormones influence neurotransmitter systems (e.g., cortisol affects serotonin, dopamine)
- Neurotransmitters can regulate hormone release (e.g., dopamine inhibits prolactin)

Cognitive Neuroscience

1. Brain Mechanisms

- **Memory:**
  - Hippocampus critical for forming new memories
  - Prefrontal cortex involved in working memory and retrieval
- **Attention:**
  - Parietal lobes and frontal cortex regulate focus and selective attention
- **Executive Function:**
  - Prefrontal cortex manages planning, decision-making, inhibition, and problem-solving

2. Neural Basis of Learning

- **Long-Term Potentiation (LTP):**
  - Strengthening of synaptic connections following repeated activity

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- Basis of learning and memory formation

- **Hebbian Learning:**
  - "Cells that fire together, wire together" — synaptic efficiency increases with co-activation

3. Disorders

- **Alzheimer's Disease:**
  - Progressive memory loss, cognitive decline
  - Associated with amyloid plaques and neurofibrillary tangles
- **Amnesia:**
  - Memory loss due to brain injury or disease
  - Types: Anterograde (new memories), retrograde (past memories)
- **ADHD (Attention Deficit Hyperactivity Disorder):**
  - Impaired attention, impulsivity, hyperactivity
  - Linked to frontal lobe and neurotransmitter dysfunction

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Genetics and Behavior

Mendelian Genetics and Complex Traits – Crash Course Notes

1. Mendelian Genetics

- Traits are determined by **single genes** with **dominant** or **recessive** alleles.
- Principles include:
    - **Law of Segregation:** Each parent contributes one allele for a trait.
    - **Law of Independent Assortment:** Genes for different traits segregate independently.
- Examples:
    - Dominant traits: Huntington's disease
    - Recessive traits: Cystic fibrosis

2. Complex Traits

- Controlled by **multiple genes (polygenic)** and influenced by **environmental factors**.
- Show continuous variation (e.g., height, intelligence, personality).
- Genetic influence is **additive** and interacts with environment.

3. Importance in Behavior

- Most psychological traits and disorders are complex traits, not Mendelian.
- Example: Schizophrenia involves many genes plus environmental triggers.

Genetic Contribution to Psychological Disorders

1. Genetic Influence

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- Many psychological disorders have a **genetic component** but are rarely caused by a single gene.
- Disorders are usually **polygenic**, involving multiple genes each contributing a small effect.

2. Examples of Genetic Links

- **Schizophrenia:** High heritability (~70-80%), multiple risk genes identified.
- **Bipolar Disorder:** Strong genetic basis with complex inheritance patterns.
- **Autism Spectrum Disorder:** Polygenic with both inherited and spontaneous mutations.
- **Depression and Anxiety:** Moderate heritability, influenced by gene-environment interaction.

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3. Gene-Environment Interaction

- Genes influence susceptibility, but **environmental factors** (stress, trauma) affect disorder onset and course.
- Epigenetic mechanisms can modify gene expression in response to environment.

4. Implications

- Understanding genetic contributions helps in diagnosis, treatment, and prevention strategies.

- Genetic research informs personalized medicine approaches.

Epigenetics

1. Definition

- Epigenetics studies **changes in gene expression** without altering the DNA sequence.
- These changes affect how genes are turned “on” or “off.”

2. Mechanisms

- **DNA Methylation:** Addition of methyl groups to DNA, usually suppressing gene expression.
- **Histone Modification:** Chemical changes to histone proteins affect DNA packaging and gene accessibility.
- **Non-coding RNA:** Small RNAs can regulate gene expression post-transcriptionally.

3. Environmental Influence

- Factors like stress, diet, toxins, and early life experiences can cause epigenetic changes.
- Explains how environment impacts behavior and risk for mental disorders.

4. Reversibility

- Epigenetic changes are often reversible, which is important for therapeutic interventions.

5. Importance in Psychology

- Helps understand complex gene-environment interactions in disorders like depression, schizophrenia,

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PTSD.

Heritability Studies

1. Purpose

- Estimate the relative contribution of **genetics vs. environment** to individual differences in traits or disorders.

2. Types of Studies

- **Twin Studies:**
  - Compare identical (monozygotic) twins (share ~100% genes) with fraternal (dizygotic) twins (share ~50% genes).
  - Greater similarity in identical twins suggests genetic influence.
- **Family Studies:**
  - Examine trait/disorder prevalence among biological relatives.
  - Higher rates in close relatives indicate genetic contribution.
- **Adoption Studies:**
  - Compare adoptees with biological vs. adoptive parents.
  - Helps separate genetic from environmental effects.

3. Heritability Estimates

- Expressed as a proportion (0 to 1) indicating genetic influence on a trait.

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- Example: Intelligence heritability ~0.5-0.8, varies with age and environment.

4. Limitations

- Heritability does not indicate how much a trait is determined by genes for an individual.
- Does not capture gene-environment interactions.

Brain and Psychological Functions

Localization of Functions

Concept : The brain has **specific areas responsible for distinct psychological functions** (e.g., language, movement).

Key Areas

- **Broca's Area:**
  - Location: Left frontal lobe
  - Function: Speech production; damage causes **Broca's aphasia** (slow, effortful speech).
- **Wernicke's Area:**
  - Location: Left temporal lobe
  - Function: Language comprehension; damage causes **Wernicke's aphasia** (fluent but nonsensical speech).
- **Motor Cortex:** Controls voluntary movement.
- **Somatosensory Cortex:** Processes sensory information from the body.

Importance

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- Supports the idea that complex behaviors can be mapped to brain regions.
- Basis for neuropsychological assessment and brain surgery planning.

Hemispheric Specialization

Concept

- The two brain hemispheres have **specialized functions**, often complementary.

Left Hemisphere

- Dominant for **language** (speech production and comprehension).
- Involved in **logical reasoning, analytical thinking,** and **mathematical skills**.

Right Hemisphere

- Specializes in **spatial abilities**, **face recognition**, **music perception**, and **emotional processing**.
- Processes **holistic and creative tasks**.

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Split-Brain Studies

- In patients with severed corpus callosum, hemispheres cannot communicate.
- Demonstrate that each hemisphere processes information independently (e.g., left hemisphere verbalizes, right hemisphere better at spatial tasks).

Frontal Lobe and Executive Functions

1. Frontal Lobe Overview

- Largest lobe of the brain, located at the front.
- Key role in higher cognitive processes and voluntary motor control.

2. Executive Functions

- **Planning:** Ability to set goals and develop steps to achieve them.
- **Decision-Making:** Choosing appropriate actions based on evaluation of options.
- **Inhibition:** Suppressing inappropriate or unwanted behaviors.
- **Working Memory:** Holding and manipulating information temporarily.
- **Cognitive Flexibility:** Shifting attention and adapting to changing rules or environments.

3. Clinical Relevance

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- Damage to frontal lobe can cause deficits in judgment, impulse control, problem-solving, and personality changes (e.g., Phineas Gage case).
- Important for understanding disorders like ADHD, schizophrenia, and frontal lobe dementia.

Clinical Neuropsychology

Study of how brain injury or neurological illness affects cognitive, emotional, and behavioral functions.

Methods

- **Lesion Studies:** Analyze behavior changes following localized brain damage to infer brain function.
- **Neuropsychological Assessment:** Use standardized tests to evaluate cognitive abilities (memory, attention, language, executive function).
- **Brain Imaging:** Supports diagnosis by locating brain abnormalities.

Common Conditions Studied

- **Traumatic Brain Injury (TBI):** Impairments depend on injury location; can affect memory, attention, personality.
- **Stroke:** May cause aphasia, paralysis, neglect, or cognitive deficits depending on affected brain area.

Neurodegenerative Disorders: Alzheimer's, Parkinson's, affecting cognition and behavior.

Applications

- Guides rehabilitation and treatment planning.
- Helps differentiate between neurological and psychiatric conditions.

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Biological Basis of Mental Disorders

1. Genetic Factors

- Many mental disorders have **heritable components** (e.g., schizophrenia, bipolar disorder, depression).
- Involve **multiple genes** with small effects, interacting with environment.

2. Neurochemical Imbalances

- Altered levels of **neurotransmitters** implicated:
  - **Dopamine:** Linked to schizophrenia and Parkinson's.
  - **Serotonin:** Associated with depression, anxiety.
  - **GABA:** Related to anxiety disorders.
  - **Glutamate:** Involved in cognitive functions, schizophrenia.

3. Brain Structure and Function

- Abnormalities in brain regions observed in mental disorders:
  - **Prefrontal cortex:** Dysfunction linked to schizophrenia, ADHD.
  - **Amygdala:** Involved in mood disorders, anxiety.
  - **Hippocampus:** Reduced volume in depression, PTSD.

4. Neuroendocrine Factors

- Dysregulation of stress hormones (e.g., **cortisol**) linked to depression and anxiety.
- HPA axis hyperactivity often seen in mood disorders.

5. Environmental Influences

- Stress, trauma, and early life adversity interact with biology to influence disorder onset and progression.

Psychopharmacology

Mechanisms of Drug Action

1. Receptor Binding

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- Drugs interact with **neurotransmitter receptors** as:
  - **Agonists:** Activate receptors, mimicking neurotransmitters.
  - **Antagonists:** Block receptors, preventing neurotransmitter action.
  - **Partial Agonists:** Activate receptors but produce weaker effects.

2. Reuptake Inhibition

- Drugs block **reuptake transporters**, increasing neurotransmitter levels in the synaptic cleft.
- Example: SSRIs inhibit serotonin reuptake to enhance mood.

3. Enzyme Inhibition

- Some drugs inhibit enzymes that degrade neurotransmitters, increasing their availability.
- Example: MAO inhibitors prevent breakdown of monoamines (serotonin, dopamine).

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4. Modulation of Neurotransmitter Release

- Drugs may increase or decrease release of neurotransmitters from presynaptic neurons.

5. Ion Channel Modulation

- Some drugs affect ion channels (e.g., calcium, sodium) to alter neuron excitability.

Classes of Psychotropic Drugs

1. Antidepressants

- **SSRIs (Selective Serotonin Reuptake Inhibitors):** Increase serotonin (e.g., fluoxetine).
- **SNRIs (Serotonin-Norepinephrine Reuptake Inhibitors):** Increase serotonin and norepinephrine.
- **Tricyclic Antidepressants (TCAs):** Block reuptake of serotonin and norepinephrine but with more side effects.
- **MAO Inhibitors:** Prevent breakdown of monoamines.

2. Antipsychotics

- **Typical (First Generation):** Block dopamine D2 receptors (e.g., haloperidol).
- **Atypical (Second Generation):** Block dopamine and serotonin receptors (e.g., risperidone).

3. Anxiolytics

- **Benzodiazepines:** Enhance GABA activity, used for anxiety and insomnia (e.g., diazepam).
- **Buspirone:** Serotonin receptor agonist used for anxiety.

4. Mood Stabilizers

- **Lithium:** Used in bipolar disorder to stabilize mood.
- **Anticonvulsants:** Also used as mood stabilizers (e.g., valproate).

5. Stimulants

- Increase dopamine and norepinephrine; used mainly in ADHD (e.g., methylphenidate, amphetamines).

Tolerance, Dependence, and Withdrawal

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1. Tolerance

- **Definition:** Reduced response to a drug after repeated use.
- **Effect:** Requires higher doses to achieve the same effect.
- **Types:**
  - *Pharmacodynamic tolerance* (receptor changes)
  - *Metabolic tolerance* (increased drug metabolism)

2. Dependence

- **Physical Dependence:** Body adapts to the drug; cessation causes physical symptoms.
- **Psychological Dependence:** Craving and compulsive drug use despite harm.

3. Withdrawal

- **Definition:** Symptoms occurring after stopping or reducing drug intake.
- **Symptoms:** Can be physical (e.g., tremors, nausea) or psychological (e.g., anxiety, depression).
- **Severity:** Varies by drug type and duration of use.

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Neurobiology of Addiction

1. Brain Reward System

- Addiction primarily involves the **mesolimbic dopamine pathway** (ventral tegmental area to nucleus accumbens).

- Drugs increase dopamine release here, producing **pleasure and reinforcement**.

2. Neurochemical Changes

- Increased dopamine release reinforces drug-taking behavior.
- Other neurotransmitters involved: **glutamate, GABA, serotonin, endorphins**.

3. Neuroadaptation

- Repeated drug use causes **changes in receptor density and function**, leading to tolerance and dependence.
- Reduced dopamine function during abstinence contributes to craving and relapse.

4. Brain Regions Involved

- **Prefrontal Cortex:** Impaired decision-making and impulse control.
- **Amygdala:** Emotional memories linked to drug cues.
- **Hippocampus:** Contextual memory of drug experiences.

5. Behavioral Consequences

- Compulsive drug seeking and use despite negative consequences.
- Loss of control over drug intake.

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Neurological and Psychiatric Disorders

1. Organic Disorders

- Caused by **physical or structural brain damage** or disease.
- Examples: Stroke, epilepsy, traumatic brain injury, multiple sclerosis, dementia.
- Diagnosis often supported by **neurological signs and imaging**.
- Symptoms: Cognitive, motor, sensory impairments depending on lesion site.

2. Functional Disorders

- No detectable **structural brain abnormalities** despite symptoms.
- Primarily affect **mental functions** such as mood, thought, and behavior.
- Examples: Schizophrenia, depression, anxiety disorders, OCD.
- Diagnosed based on **clinical presentation** and symptom patterns.

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Common Neurological Disorders

Epilepsy

1. Definition and Basics

- **Epilepsy** is a neurological disorder marked by recurrent, unprovoked seizures due to abnormal **electrical activity in the brain**.

- Can be **focal (partial)** or **generalized** based on origin and spread of activity.

2. Brain Mechanisms

- **Hyperexcitability of neurons:**
  - Excessive neuronal firing due to imbalances in **excitatory (glutamate)** and **inhibitory (GABA)**neurotransmission.
- **Hypersynchrony:**
  - Neurons fire in a highly synchronized manner during a seizure.
- **Key structures involved:**
  - **Hippocampus** (temporal lobe epilepsy), **thalamus**, and **cortex**.

3. Neurotransmitter Imbalance

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- **Increased glutamate (excitatory)** → triggers seizures.
- **Reduced GABA (inhibitory)** → fails to suppress abnormal firing.
- Many antiepileptic drugs (AEDs) work by enhancing GABA or inhibiting glutamate.

4. Causes and Triggers

- **Genetic mutations:** Channelopathies affecting ion channels.
- **Structural abnormalities:** Tumors, trauma, infections, stroke.

- **Idiopathic cases:** No identifiable cause in many patients.

5. Diagnosis and Management

- **EEG (electroencephalogram):** Key tool for detecting abnormal brain activity.
- **MRI/CT:** Identifies structural causes.
- **Treatment:**
  - **Antiepileptic drugs (e.g., valproate, carbamazepine)**
  - **Surgical options:** Temporal lobectomy, vagus nerve stimulation.

Multiple Sclerosis (MS)

1. Definition

- **Multiple Sclerosis** is a **chronic autoimmune demyelinating disorder** of the central nervous system (CNS).
- It involves **immune-mediated damage** to the **myelin sheath**, leading to disrupted neural conduction.

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2. Pathophysiology

- **Immune attack on myelin** (primarily by T-cells and B-cells).
- Leads to formation of **sclerotic plaques** in CNS (brain and spinal cord).
- Results in **axonal degeneration**, inflammation, and **slowed or blocked nerve signals**.

3. CNS Structures Affected

- **White matter tracts** in:
  - **Optic nerves** (→ visual disturbances),
  - **Spinal cord** (→ motor/sensory deficits),
  - **Cerebellum** (→ ataxia, balance issues).

4. Symptoms (Depend on lesion sites)

- Motor: Weakness, spasticity, coordination problems
- Sensory: Numbness, tingling, pain
- Visual: Optic neuritis, diplopia
- Cognitive: Memory, attention deficits
- Fatigue, bladder/bowel dysfunction

5. Diagnosis

- **MRI**: Shows lesions/plaques (often periventricular).
- **Lumbar puncture**: Oligoclonal bands in CSF.
- **Evoked potentials**: Delayed responses indicating demyelination.

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6. Neurobiology Highlights

- **Loss of saltatory conduction** due to demyelination.
- Neurons become less efficient or non-functional.
- **Neuroinflammation and oxidative stress** contribute to disease progression.

7. Course and Variants

- **Relapsing-remitting MS (RRMS)**: Most common; periods of recovery and flare-ups.

- **Secondary progressive MS**: Gradual worsening after initial relapsing phase.
- **Primary progressive MS**: Continuous worsening from onset.

8. Treatment

- **Immunomodulators**: Interferon-beta, glatiramer acetate
- **Immunosuppressants**: Natalizumab, fingolimod
- **Symptomatic treatment**: Spasticity, fatigue, depression

Stroke

1. Definition

- **Stroke** is a **sudden neurological deficit** due to **interruption of blood supply** to the brain.
- Two main types:
  - **Ischemic (≈80%)** – due to blood vessel blockage (e.g., thrombus, embolus).
  - **Hemorrhagic (≈20%)** – due to rupture of blood vessel (e.g., aneurysm, hypertension-related bleed).

2. Pathophysiology

- **Ischemic Stroke:**
  - ↓ Blood flow → ↓ oxygen and glucose → neuronal energy failure.
  - **Excitotoxicity**: Excess glutamate release → calcium overload → cell death.

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- Formation of **core (dead tissue)** and **penumbra (salvageable area)**.

- **Hemorrhagic Stroke:**
  - Bleeding into brain tissue → ↑ intracranial pressure (ICP), mechanical damage.

3. Neuroanatomy of Stroke Deficits

- **Middle cerebral artery (MCA)**: Hemiplegia, aphasia, sensory loss.
- **Anterior cerebral artery (ACA)**: Leg weakness, abulia (lack of will).
- **Posterior cerebral artery (PCA)**: Visual field deficits (e.g., hemianopia).
- **Brainstem strokes**: Cranial nerve signs, ataxia, coma.

4. Symptoms (depend on location)

- Sudden weakness or numbness (face/arm/leg), speech difficulties, vision loss, dizziness, severe headache.

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5. Diagnosis

- **CT scan**: Rule out hemorrhage.
- **MRI**: Sensitive to ischemia, detects early infarcts.
- **Angiography**: Visualize blood vessels.

6. Treatment

- **Ischemic Stroke**:
  - **tPA (tissue plasminogen activator)** within 4.5 hours.
  - Antiplatelets, anticoagulants, thrombectomy.

- **Hemorrhagic Stroke**:
  - Manage blood pressure, surgery if needed.
  - Avoid anticoagulants.
- **Rehabilitation**:
  - Physiotherapy, speech therapy, occupational therapy for long-term recovery.

7. Neurobiological Concepts

- **Neuroplasticity** plays a key role in post-stroke recovery.
- **Glial response** and **inflammatory cascades** can worsen or aid recovery.
- Research on **neuroprotection and stem cell therapy** is ongoing.

Dementia

1. Definition

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- **Dementia** is a **progressive decline in cognitive function** severe enough to interfere with daily life.
- Affects **memory, language, attention, executive function, and personality**.
- Multiple causes, **Alzheimer's disease (AD)** is the most common.

2. Types and Causes

- **Alzheimer's Disease (AD)** – most common, gradual memory loss.
- **Vascular Dementia** – due to multiple small strokes.

- **Lewy Body Dementia** – includes hallucinations, Parkinsonian features.
- **Frontotemporal Dementia (FTD)** – early personality/language changes.

3. Neurobiology of Alzheimer's Disease (AD)

- **Amyloid plaques**: Accumulation of **β-amyloid protein** outside neurons.
- **Neurofibrillary tangles**: Aggregates of **hyperphosphorylated tau protein** inside neurons.
- **Cholinergic deficit**: ↓ Acetylcholine → memory impairment.
- Brain atrophy (especially **hippocampus**, **cortex**), enlarged ventricles.
- **Neuroinflammation and oxidative stress** also contribute.

4. Neurobiology of Other Dementias

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- **Vascular Dementia**: Neuronal loss from **chronic ischemia**, infarcts.
- **Lewy Body Dementia**: Presence of **α-synuclein (Lewy bodies)** in cortex.
- **FTD**: Loss of neurons in **frontal and temporal lobes**; tau or TDP-43 protein pathology.

5. Symptoms

- **Cognitive**: Memory loss, language problems, disorientation.
- **Behavioral**: Apathy, aggression, personality changes.

- **Neurological**: Motor signs in Lewy body or vascular types.

6. Diagnosis

- **Neuropsychological tests** (e.g., MMSE).
- **MRI/CT**: Brain atrophy, vascular changes.
- **PET scans**: Amyloid imaging (for AD).
- **Biomarkers** in CSF: ↓ Aβ42, ↑ tau.

7. Treatment and Management

- No cure; treatment is symptomatic and supportive.
- **Cholinesterase inhibitors**: Donepezil, rivastigmine (for AD).
- **Memantine**: NMDA antagonist for moderate to severe AD.
- Manage comorbidities, cognitive training, caregiver support.

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Neurobiology of Psychiatric Disorders

Schizophrenia

1. Neurochemical Basis

- **Dopamine Hypothesis:**
  - Overactivity in **mesolimbic dopamine pathway** → Positive symptoms (hallucinations, delusions).
  - Underactivity in **mesocortical pathway** → Negative symptoms (flat affect, avolition) and cognitive deficits.

- Other neurotransmitters involved:
  - **Glutamate** (NMDA receptor hypofunction)
  - **GABA** (inhibitory control deficits)

2. Structural Brain Abnormalities

- **Enlarged lateral ventricles**
- **Reduced grey matter volume** (especially in prefrontal cortex, hippocampus)
- **Thinner cortex and disorganized neural connectivity**

3. Functional Brain Abnormalities

- Hypofrontality: Reduced activity in **prefrontal cortex**
- Disrupted communication between **frontal and temporal lobes**

4. Developmental Factors

- Prenatal factors: Maternal infection, hypoxia, malnutrition
- Early neurodevelopmental abnormalities may predispose brain to later dysfunction.

5. Genetics and Risk

- High heritability (~80%)
- Associated genes: **DISC1**, **COMT**, **NRG1**
- Polygenic risk with environmental interactions (stress, substance use)

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Depression

1. Neurochemical Theories

- **Monoamine Hypothesis:**
  - Deficiency in **serotonin (5-HT)**, **norepinephrine (NE)**, and **dopamine (DA)**.
  - Basis for antidepressant action (e.g., SSRIs, SNRIs, tricyclics).
- **Serotonin Dysfunction:**
  - Linked to mood regulation, anxiety, and sleep disturbances.
  - SSRIs increase serotonin availability in synaptic cleft.

2. Neuroplasticity and BDNF

- **Reduced Brain-Derived Neurotrophic Factor (BDNF)** affects neuronal survival and synaptic plasticity.
- Decreased BDNF in **hippocampus** and **prefrontal cortex** seen in depression.
- Antidepressants may increase BDNF levels over time.

3. HPA Axis Dysregulation

- **Hyperactivity of the hypothalamic-pituitary-adrenal (HPA) axis** → Elevated cortisol.
- Chronic stress leads to hippocampal atrophy, emotional dysregulation.

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- Implicated in melancholic and atypical subtypes of depression.

4. Brain Structure and Function

- **Reduced hippocampal volume** (linked to memory and stress regulation).
- **Decreased activity in prefrontal cortex** (executive function, mood regulation).
- **Increased activity in amygdala** (emotion and threat processing).

5. Genetic and Epigenetic Factors

- Genes like **SLC6A4** (serotonin transporter), **5-HTTLPR** linked to susceptibility.
- **Gene-environment interaction** (e.g., childhood trauma + genetic risk) contributes.
- Epigenetic changes affect gene expression under chronic stress.

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Bipolar Disorder

1. Neurochemical Dysregulation

- **Dopamine Hypothesis:**
  - **Mania:** Increased dopamine transmission.
  - **Depression:** Decreased dopamine activity.
- **Other neurotransmitters involved:**

- **Glutamate:** Elevated in mania, affecting excitatory signaling.
- **GABA:** Reduced inhibitory control.
- **Serotonin and norepinephrine:** Imbalanced across mood states.

2. Brain Structure and Function

- **Prefrontal Cortex:**
  - Decreased activity in depression; increased or erratic activity in mania.
- **Amygdala:**
  - Hyperactive in both mania and depression.
- **Hippocampus:**
  - Volume reductions; related to memory and emotional regulation.

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3. Neural Circuitry Abnormalities

- Disruption in **fronto-limbic circuits**: affects emotional regulation and impulse control.
- Altered connectivity between **prefrontal cortex, amygdala, and basal ganglia**.

4. Neuroendocrine Changes

- **HPA Axis Dysfunction:**
  - Elevated cortisol levels during depressive phases.
  - Stress sensitivity may trigger episodes.

5. Genetic and Molecular Factors

- High heritability (~70–85%)
- Genes implicated: **ANK3**, **CACNA1C**, **BDNF**
- Mitochondrial dysfunction and oxidative stress also play a role.

Anxiety Disorders

1. Brain Structures Involved

- **Amygdala:**
  - Central to fear processing and threat detection.
  - **Hyperactivity** is a hallmark of anxiety disorders.
- **Prefrontal Cortex (PFC):**
  - Regulates emotional responses.
  - **Reduced top-down control** over amygdala in anxiety.
- **Hippocampus:**
  - Involved in context-based memory of fear.
  - Dysfunction contributes to inappropriate fear responses.

2. Neurotransmitter Systems

- **GABA (Gamma-Aminobutyric Acid):**
  - Primary inhibitory neurotransmitter.

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- **Low GABA activity** → reduced inhibition → heightened anxiety.
- Target of benzodiazepines (enhance GABA-A activity).

- **Serotonin (5-HT):**
  - Regulates mood and anxiety.
  - **Dysfunction** associated with generalized anxiety disorder (GAD), panic disorder.
  - Target of SSRIs.
- **Norepinephrine (NE):**
  - Involved in arousal and stress.
  - **Overactivity** → physical symptoms of anxiety (e.g., heart rate, sweating).

3. HPA Axis Dysregulation

- **Hypothalamic-Pituitary-Adrenal (HPA) axis** is overactivated.
  - Increased cortisol levels → chronic stress response.
  - Contributes to anxiety sensitivity and hypervigilance.

4. Genetic and Epigenetic Factors

- Genetic predisposition (e.g., variations in **5-HTTLPR**, **COMT**)
- Epigenetic changes due to early-life stress or trauma increase vulnerability.

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Obsessive-Compulsive Disorder (OCD)

1. Brain Circuitry Involved

- **Cortico-Striato-Thalamo-Cortical (CSTC) Loop** dysfunction:
  - Overactivity in the **orbitofrontal cortex (OFC)**, **anterior cingulate cortex (ACC)**, and **caudate nucleus**.
  - Fails to suppress intrusive thoughts → leads to compulsions.
- **Basal Ganglia Dysfunction**
  - Inadequate filtering of repetitive thoughts and behaviors.

2. Neurotransmitter Dysregulation

- **Serotonin (5-HT):**
  - Primary neurotransmitter implicated.
  - **Low serotonin activity** associated with obsessions and compulsions.
  - SSRIs are first-line treatments.
- **Dopamine:**
  - Involved in reward and reinforcement of compulsive behaviors.
  - Elevated dopaminergic activity may exacerbate symptoms.

3. Functional and Structural Changes

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- **Increased activity** in OFC, caudate, thalamus on neuroimaging.
- **Structural changes**: Abnormal volume in basal ganglia and frontal regions.

4. Genetic and Environmental Contributions

- Genetic links: Twin studies show moderate heritability.
- Possible involvement of genes affecting serotonin transport and dopamine regulation.
- Childhood infections (PANDAS hypothesis) and stress may trigger or worsen symptoms.

Advanced & Applied Biopsychology

Neuroimaging Techniques

1. Structural Imaging

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- **CT (Computed Tomography)**
  - Uses X-rays to create brain images.
  - Detects bleeding, tumors, fractures, and brain atrophy.
  - Quick, widely available but lower resolution than MRI.
- **MRI (Magnetic Resonance Imaging)**
  - Uses magnetic fields and radio waves.
  - Provides high-resolution images of brain anatomy.

- Detects lesions, tumors, white and gray matter differences.

2. Functional Imaging

- **fMRI (Functional MRI)**
  - Measures brain activity through blood oxygen level changes (BOLD signal).
  - Used in cognitive neuroscience to map active brain areas.
- **PET (Positron Emission Tomography)**
  - Uses radioactive tracers to measure metabolic activity.
  - Assesses neurotransmitter systems and brain metabolism.
- **EEG (Electroencephalography)**
  - Records electrical activity via scalp electrodes.
  - Excellent temporal resolution; used in epilepsy, sleep studies.
- **MEG (Magnetoencephalography)**
  - Detects magnetic fields from neural electrical activity.
  - High temporal and spatial resolution.

Brain Stimulation Techniques

1. TMS (Transcranial Magnetic Stimulation)

- Non-invasive magnetic pulses applied to scalp.

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- Temporarily modulates cortical activity.
- Used in depression, research on brain function.

2. tDCS (Transcranial Direct Current Stimulation)

- Applies weak electrical currents to scalp.
- Alters neuronal excitability (increases or decreases).
- Used in cognitive enhancement, rehabilitation.

3. DBS (Deep Brain Stimulation)

- Invasive technique with implanted electrodes.
- Delivers electrical stimulation to deep brain areas.
- Used in Parkinson's disease, OCD, treatment-resistant depression.

Neurofeedback and Brain-Computer Interface (BCI)

Neurofeedback

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- Real-time monitoring of brain activity (usually via EEG).
- Individuals learn to self-regulate brain waves through feedback.
- Used in ADHD, anxiety, PTSD, epilepsy for improving brain function.

Brain-Computer Interface (BCI)

- Direct communication pathway between brain and external devices.
- Translates neural signals into commands to control computers, prosthetics, or wheelchairs.

- Applications in motor rehabilitation, assistive technology for paralysis.

Biopsychosocial Model in Clinical Settings

- Integrates **Biological**, **Psychological**, and **Social** factors to understand health and illness.
- **Biological**: Genetics, neurobiology, physical health.
- **Psychological**: Emotions, cognition, behavior, coping skills.
- **Social**: Family, culture, socioeconomic status, social support.
- Used for holistic diagnosis, treatment, and rehabilitation.
- Helps tailor interventions to individual patient needs.
- Widely applied in **mental health**, **chronic diseases**, **pain management**.

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Neuroethics

- **Neuroethics** studies ethical issues related to neuroscience research and applications.
- Key concerns include:
  - **Consent and autonomy** in brain interventions.
  - **Privacy** of neural data and brain imaging (mind-reading risks).
  - **Safety** and side effects of neuro-enhancement techniques.

- **Identity and agency**: Impact of brain treatments on personality and free will.

- Topics include ethical use of:
  - **Neuro-enhancement** (cognitive/behavioral enhancement via drugs or devices).
  - **Brain-computer interfaces** and neural data privacy.
  - **Deep Brain Stimulation (DBS)** and invasive procedures.
  - Research ethics in neuroimaging and clinical trials.
- Balances potential benefits with respect for human rights and dignity.

Recent Research & Trends

1. Connectome Project and Brain Mapping

- Large-scale effort to map all neural connections (the "connectome").
- Aims to understand brain network organization and function.
- Advances brain disorders research and personalized medicine.

2. Gut-Brain Axis and Microbiome Research

- Studies bidirectional communication between gut microbiota and brain.
- Links gut health with mood, anxiety, cognition, and neurodevelopment.

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- Potential new treatments via diet and probiotics.

3. Inflammation and Psychiatric Illness

- Chronic inflammation implicated in depression, schizophrenia, bipolar disorder.
- Immune system interactions with brain influence mental health.
- Anti-inflammatory treatments being explored.

4. Neurodiversity Movement

- Views neurological differences (e.g., autism, ADHD) as natural variations.
- Emphasizes acceptance, strengths, and rights over pathology.
- Influences research and clinical approaches.

5. Artificial Intelligence in Brain Modeling

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- AI used to simulate brain processes and analyze complex neural data.
- Helps in diagnosis, brain-computer interfaces, and drug discovery.
- Advances understanding of cognition and disorders.

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