Toppersnotes
Unleash the topper in you

MP - SET

LIFE SCIENCE

Madhya Pradesh State Eligibility Test

VOLUME – 3

Developmental Biology, System Physiology – Plant & System Physiology

SIERRA INNOVATIONS

Index

V UNIT Developmental Biology

Basic Concepts of Development - Part 1

1. **Overview of Basic Concepts of Development - Part 1**

   Developmental biology explores how a single cell, the zygote, gives rise to a complex organism through tightly regulated cellular processes. The concepts of potency, commitment, specification, induction, and competence are foundational to understanding how cells acquire specific fates and organize into tissues.

- **Potency**:
  - The range of developmental fates a cell can achieve (e.g., totipotent, pluripotent).
  - Reflects a cell's developmental potential.
- **Commitment**:
  - The process by which a cell's developmental fate becomes restricted.
  - Involves intrinsic and extrinsic cues.
- **Specification**:
  - The stage where a cell is programmed to follow a specific fate but remains reversible.
  - Often driven by morphogens and transcription factors.
- **Induction**:
  - The process by which one group of cells influences the fate of neighboring cells via signaling.
  - Critical for tissue patterning.
- **Competence**:
  - The ability of a cell to respond to inductive signals.
  - Depends on receptor expression and signaling pathways.
- **Biological Relevance**:
  - These processes ensure precise cell fate determination, enabling organ formation (~10^{12} cells in humans).
  - Dysregulation leads to developmental defects (e.g., spina bifida).
- **Applications**:
  - Stem cell therapies for regenerative medicine.
  - Developmental models for congenital disorders.
  - Synthetic biology for tissue engineering.

Table 1: Overview of Basic Developmental Concepts

Concept	Definition	Key Feature	Biological Role	Example
Potency	Range of cell fates	Totipotent, pluripotent	Defines developmental potential	Zygote totipotency
Commitment	Fate restriction	Intrinsic/extrinsic cues	Locks cell fate	Neural crest commitment
Specification	Reversible fate programming	Morphogens, TFs	Initiates cell fate	Blastula mesoderm
Induction	Cell-cell signaling	Ligands, receptors	Patterns tissues	Spemann organizer
Competence	Ability to respond to signals	Receptor expression	Enables induction	Ectoderm neural competence

2. Potency

Potency refers to a cell's capacity to differentiate into various cell types, ranging from totipotent to unipotent.

2.1 Types of Potency

- **Totipotent**:
  - **Definition**: Can form all cell types, including embryonic and extra-embryonic tissues (e.g., placenta).
  - **Examples**:
    - Zygote: Forms entire organism (~10^{12} cells in mammals).
    - Early blastomeres (up to 4-cell stage in mammals).
  - **Molecular Basis**:
    - Expression of pluripotency genes (e.g., OCT4, SOX2, NANOG).
    - Open chromatin state (~80% euchromatin).
- **Pluripotent**:
  - **Definition**: Can form all embryonic cell types but not extra-embryonic tissues.
  - **Examples**:
    - Embryonic stem cells (ESCs) from inner cell mass (ICM).
    - Induced pluripotent stem cells (iPSCs) via Yamanaka factors (OCT4, SOX2, KLF4, c-MYC).
  - **Molecular Basis**:
    - High expression of pluripotency TFs (~10^3–10^4 transcripts/cell).
    - Epigenetic marks: H3K4me3 at developmental genes.
- **Multipotent**:
  - **Definition**: Can form multiple cell types within a lineage.
  - **Examples**:
    - Hematopoietic stem cells (HSCs): Form blood lineages (~10^4 cells/HSC).
    - Neural stem cells: Form neurons, glia.
  - **Molecular Basis**:
    - Lineage-specific TFs (e.g., GATA1 for erythroid cells).
    - Restricted chromatin (~50% euchromatin).
- **Oligopotent**:
  - **Definition**: Limited to a few cell types within a lineage.
  - **Example**: Myeloid progenitors form neutrophils, monocytes.
- **Unipotent**:
  - **Definition**: Restricted to one cell type.
  - **Example**: Spermatogonia form sperm.
- **Efficiency**:
  - Totipotent: ~10^6 possible fates/zygote.
  - Pluripotent: ~10^3–10^4 fates/ESC.
  - Multipotent: ~10–10^2 fates/HSC.
- **Energetics**:
  - TF activation: $\Delta G \approx -30$ kJ/mol.
  - Chromatin remodeling: ATP-dependent, $\Delta G \approx -50$ kJ/mol.

2.2 Regulation of Potency

- **Transcription Factors**:
  - OCT4 maintains totipotency/pluripotency, downregulated in multipotent cells.
  - **Example**: OCT4 knockout in ESCs leads to differentiation (~100% loss of pluripotency).
- **Epigenetic Modifications**:
  - **H3K4me3**: Activates pluripotency genes in ESCs.
  - **H3K27me3**: Silences lineage genes in totipotent cells.
  - **DNA Methylation**: Increases in differentiated cells, restricts potency (~70% CpG methylation).
- **Signaling Pathways**:
  - **LIF (Leukemia Inhibitory Factor)**: Maintains ESC pluripotency via STAT3.
  - **Wnt/β-Catenin**: Promotes HSC multipotency.

- **Regulation**:
  - **Feedback**: OCT4 represses differentiation genes (e.g., Cdx2).
  - **Microenvironment**: Niche signals (e.g., BMP4) modulate potency.
- **Energetics**:
  - Epigenetic modification: $\Delta G \approx -20$ kJ/mol.
  - Signaling activation: $\Delta G \approx -30$ kJ/mol.

2.3 Biological Applications

- **Embryogenesis**:
  - Totipotent zygote forms ~10^{12} cells in human development.
  - Pluripotent ICM forms three germ layers (~10^4 cells/layer).
- **Regenerative Medicine**:
  - iPSCs reprogrammed from fibroblasts, used for tissue repair (e.g., retinal cells).
- **Disease**:
  - **Teratomas**: Arise from pluripotent cells, form mixed tissues (~10% of germ cell tumors).
- **Therapeutics**:
  - ESC-derived therapies for Parkinson's (~10^5 neurons transplanted).
- **Biotechnology**:
  - CRISPR-edited iPSCs for disease modeling.

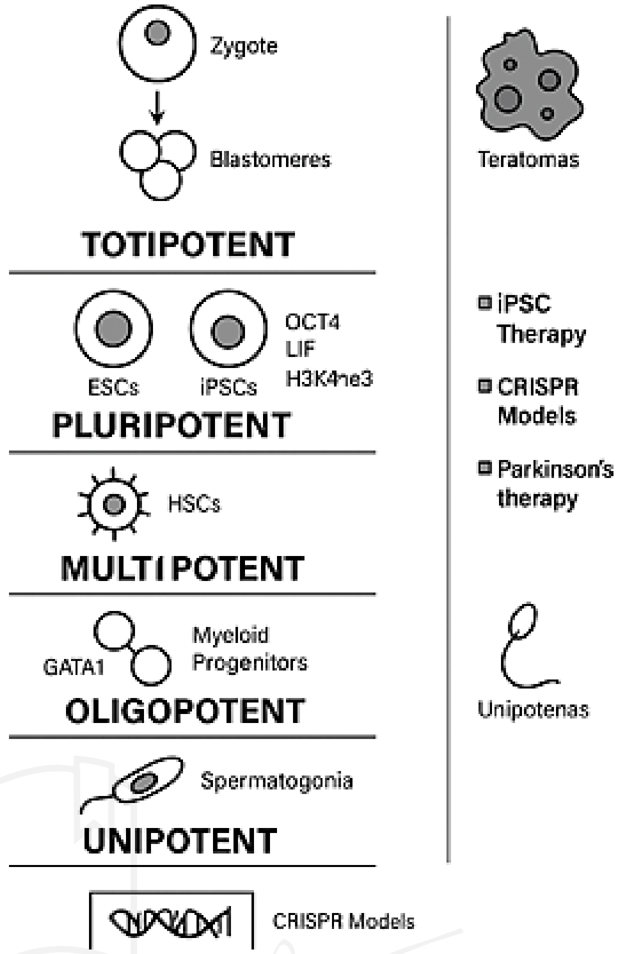

Diagram 1: Cell Potency Hierarchy
[Description: A diagram showing potency levels: totipotent (zygote, blastomeres), pluripotent (ESCs, iPSCs), multipotent (HSCs), oligopotent (myeloid progenitors), unipotent (spermatogonia). Molecular basis (OCT4, GATA1), epigenetic marks (H3K4me3), and signaling (LIF, Wnt) are depicted. Applications (iPSC therapy) and diseases (teratomas) are shown. A side panel illustrates CRISPR models and Parkinson's therapy, with biological roles (e.g., cell fate potential).]

Table 2: Types of Cell Potency

Potency Type	Example	Cell Fates	Molecular Marker
Totipotent	Zygote	All embryonic/extra-embryonic	OCT4, NANOG
Pluripotent	ESCs, iPSCs	All embryonic	SOX2, KLF4
Multipotent	HSCs	Multiple within lineage	GATA1, PAX6
Unipotent	Spermatogonia	Single cell type	PLZF

3. Commitment

Commitment is the process by which a cell's developmental fate becomes progressively restricted, transitioning from a broad to a specific potential.

3.1 Types of Commitment

- **Specification (Reversible)**:
  - **Definition**: Cell is programmed for a fate but can be redirected.
  - **Example**: Blastula ectoderm specified for neural fate, reversible by BMP4.

- **Determination (Irreversible)**:
  - **Definition**: Cell fate is fixed, resistant to external cues.
  - **Example**: Neural crest cells determined for neurons, ~10^3 cells/embryo.
- **Mechanisms**:
  - **Intrinsic Cues**: Cytoplasmic determinants (e.g., Bicoid in Drosophila).
  - **Extrinsic Cues**: Signaling molecules (e.g., Shh in neural tube).
- **Efficiency**:
  - Specification: ~10^2–10^3 cells/embryo specified per germ layer.
  - Determination: ~10–50% of specified cells become determined.
- **Energetics**:
  - TF binding: $\Delta G \approx -30$ kJ/mol.
  - Signal transduction: $\Delta G \approx -50$ kJ/mol.

3.2 Molecular Basis

- **Transcription Factors**:
  - **Pax6**: Commits ectoderm to neural fate, activates NeuroD.
  - **GATA1**: Commits HSCs to erythroid lineage, upregulates HBB.
- **Epigenetic Regulation**:
  - **H3K27me3**: Silences alternative fate genes (e.g., Hox in neural cells).
  - **DNA Methylation**: Locks in erythroid genes (~80% methylation at non-erythroid loci).
- **Signaling Pathways**:
  - **Notch**: Promotes neural commitment via Hes1.
  - **TGF-β**: Drives mesoderm commitment via Smad2/3.
- **Regulation**:
  - **Feedback**: Pax6 represses non-neural TFs (e.g., Sox9).
  - **Crosstalk**: Notch inhibits TGF-β in ectoderm.
- **Energetics**:
  - Epigenetic silencing: $\Delta G \approx -20$ kJ/mol.
  - Pathway activation: $\Delta G \approx -30$ kJ/mol.

3.3 Biological Applications

- **Embryogenesis**:
  - Neural crest commitment forms ~10^3 neurons/glia in vertebrates.
- **Developmental Disorders**:
  - **Holoprosencephaly**: Shh defects disrupt neural commitment (~1/10,000 births).
- **Therapeutics**:
  - Directed differentiation of iPSCs for neural repair (~10^5 cells/transplant).
- **Biotechnology**:
  - Single-cell RNA-seq to study commitment dynamics.

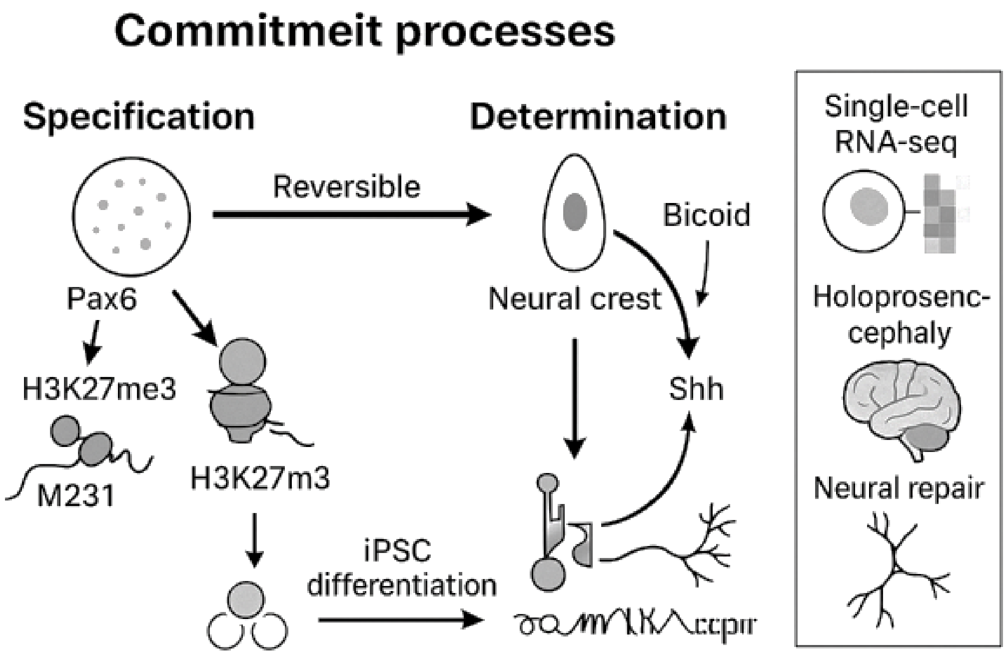

Diagram 2: Commitment Processes

[Description: A diagram showing specification (ectoderm, reversible) and determination (neural crest, irreversible). Mechanisms (Pax6, H3K27me3, Notch) and cues (Bicoid, Shh) are depicted. Applications (iPSC differentiation) and diseases (holoprosencephaly) are shown. A side panel illustrates single-cell RNA-seq and neural repair, with biological roles (e.g., fate restriction).]

4. Specification

Specification is the reversible programming of a cell to follow a specific developmental fate, often driven by morphogens and transcription factors.

4.1 Mechanisms

- **Morphogen Gradients**:
  - **Definition**: Concentration gradients of signaling molecules that specify cell fates.
  - **Example**: Bicoid gradient in Drosophila specifies anterior-posterior axis (~10^3 molecules/embryo).
  - **Mechanism**: High Bicoid activates hunchback, low Bicoid allows Kruppel.
- **Transcription Factors**:
  - **Sox2**: Specifies neural ectoderm in vertebrates.
  - **Tbx5**: Specifies limb bud mesoderm.
- **Signaling Pathways**:
  - **Hedgehog (Hh)**: Specifies ventral neural tube fates via Gli.
  - **BMP**: Specifies dorsal ectoderm via Smad1/5.
- **Efficiency**:
  - ~10^2–10^3 cells specified per morphogen gradient.
  - ~10–50% reversible by external signals.
- **Energetics**:
  - Morphogen diffusion: $\Delta G \approx -20$ kJ/mol.
  - TF activation: $\Delta G \approx -30$ kJ/mol.

4.2 Regulation

- **Gradient Formation**:
  - **Diffusion**: Bicoid diffuses ~100 µm in Drosophila syncytium.
  - **Degradation**: Shh degraded by proteases, shapes gradient.
- **Feedback Loops**:
  - **Positive**: Hh upregulates Gli, reinforces specification.
  - **Negative**: Sox2 represses non-neural genes.
- **Crosstalk**:
  - Hh inhibits BMP in neural tube, balances fates.
- **Regulation**:
  - **Timing**: Specification occurs in blastula (~10^2–10^3 cells).
  - **Microenvironment**: ECM modulates morphogen spread.
- **Energetics**:
  - Gradient stabilization: $\Delta G \approx -20$ kJ/mol.
  - Crosstalk signaling: $\Delta G \approx -30$ kJ/mol.

4.3 Biological Applications

- **Embryogenesis**:
  - Bicoid specifies ~10^3 anterior cells in Drosophila.
  - Shh patterns neural tube, ~10^4 neurons formed.
- **Developmental Disorders**:
  - **Cyclopia**: Shh gradient defects (~1/100,000 births).
- **Therapeutics**:
  - Morphogen mimics for tissue engineering (e.g., Shh for neural repair).
- **Biotechnology**:
  - Synthetic morphogen gradients for organoid patterning.

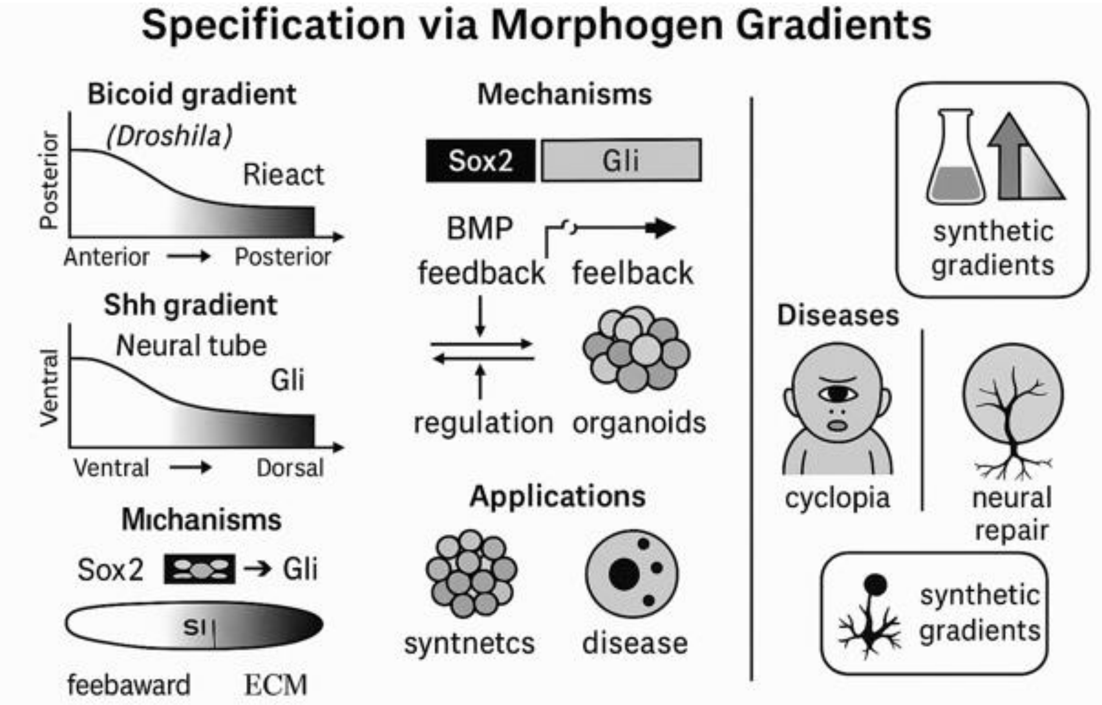

Diagram 3: Specification via Morphogen Gradients

[Description: A diagram showing specification: Bicoid gradient (*Drosophila*, hunchback), Shh gradient (neural tube, Gli). Mechanisms (Sox2, BMP) and regulation (feedback, ECM) are depicted. Applications (organoids) and diseases (cyclopia) are shown. A side panel illustrates synthetic gradients and neural repair, with biological roles (e.g., patterning).]

5. **Induction**

Induction is the process by which one group of cells influences the fate of neighboring cells through signaling molecules.

5.1 Mechanisms

- **Inductive Signals**:
  - **Ligands**: Shh, BMP4, FGF, Wnt.
    - **Example**: Spemann organizer secretes Noggin, induces dorsal mesoderm in amphibians.
  - **Receptors**: Patched (Shh), BMPR (BMP), Frizzled (Wnt).
  - **Pathways**: Smad (BMP), β-catenin (Wnt), Gli (Shh).
- **Key Examples**:
  - **Spemann Organizer**: Induces neural plate via Noggin/Chordin (~10^3 cells in Xenopus).
  - **Lens Induction**: Optic vesicle induces lens placode via FGF (~10^2 cells in chick).
- **Efficiency**:
  - ~10^2–10^3 cells induced/signal.
  - ~10–50% cells respond to inducer.
- **Energetics**:
  - Ligand-receptor binding: $\Delta G \approx -40$ kJ/mol.
  - Pathway activation: $\Delta G \approx -50$ kJ/mol.

5.2 Regulation

- **Competence**:
  - **Definition**: Ability of cells to respond to inductive signals.
  - **Example**: Ectoderm competent for neural induction via FGF receptors (~10^3 receptors/cell).
- **Signaling Range**:
  - Short-range: Paracrine signaling (~10–100 μm).
  - Long-range: Morphogen gradients (~100–500 μm).
- **Feedback**:
  - **Positive**: Shh upregulates Patched, amplifies signaling.
  - **Negative**: Noggin inhibits BMP, restricts induction.
- **Regulation**:
  - **Timing**: Induction occurs in gastrula (~10^3 cells).
  - **Receptor Expression**: FGFR1 upregulated in competent ectoderm.

- **Energetics**:
  - Receptor upregulation: ΔG ≈ -20 kJ/mol.
  - Feedback signaling: ΔG ≈ -30 kJ/mol.

5.3 Biological Applications

- **Embryogenesis**:
  - Neural induction forms ~10^4 neurons in vertebrate embryos.
  - Lens induction ensures ~10^2 lens cells in eye development.
- **Developmental Disorders**:
  - **Anencephaly**: Defective neural induction (~1/1,000 births).
- **Therapeutics**:
  - FGF-based induction for retinal repair (~10^5 cells transplanted).
- **Biotechnology**:
  - Induced organoids for developmental studies.

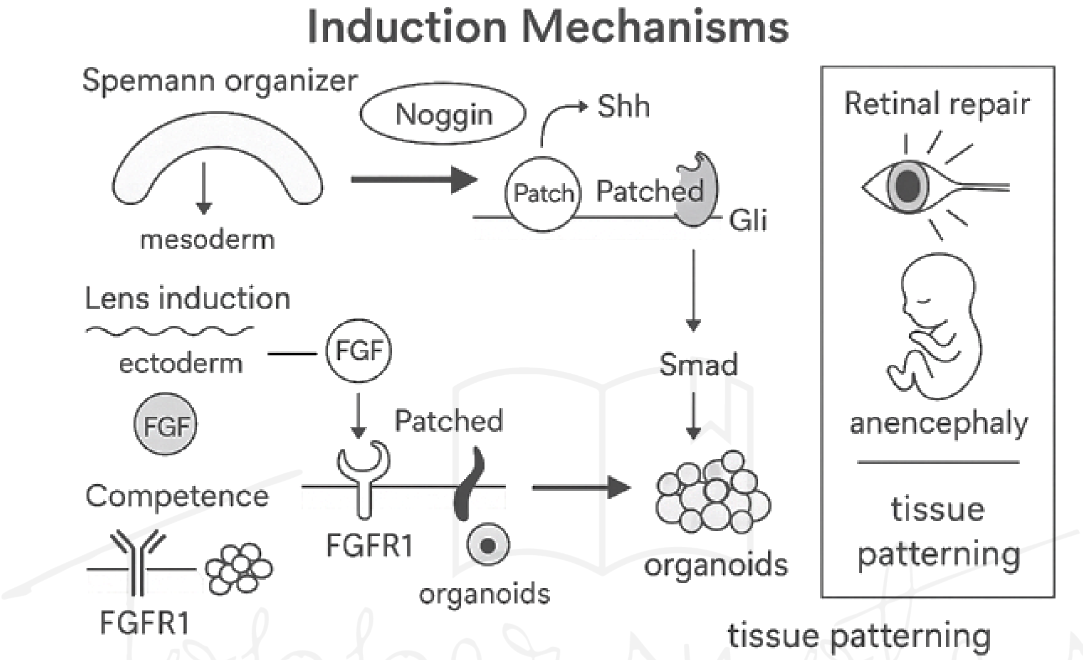

Diagram 4: Induction Mechanisms

[Description: A diagram showing induction: Spemann organizer (Noggin, mesoderm), lens induction (FGF, placode). Signals (Shh, BMP), receptors (Patched), and pathways (Smad, Gli) are depicted. Competence (FGFR1) and applications (organoids) are shown. A side panel illustrates retinal repair and anencephaly, with biological roles (e.g., tissue patterning).]

6. Competence

Competence is the ability of a cell to respond to inductive signals, determined by receptor expression and intracellular signaling.

6.1 Mechanisms

- **Receptor Expression**:
  - **FGFR**: Ectoderm competent for neural induction (~10^3 receptors/cell).
  - **BMPR**: Mesoderm competent for bone induction.
- **Signaling Pathways**:
  - **MAPK**: Activated by FGF, induces neural fate via ERK.
  - **Smad**: Activated by BMP, induces mesodermal fate.
- **Epigenetic State**:
  - **H3K4me3**: Opens neural genes in competent ectoderm.
  - **H3K27me3**: Silences non-responsive genes.
- **Examples**:
  - **Neural Competence**: Xenopus ectoderm responds to Noggin (~10^2 cells induced).
  - **Lens Competence**: Chick ectoderm responds to FGF (~10^2 lens cells).
- **Efficiency**:
  - ~10–50% of cells competent in gastrula.
  - ~10^2–10^3 receptors/cell in competent tissues.
- **Energetics**:
  - Receptor binding: ΔG ≈ -40 kJ/mol.
  - Epigenetic activation: ΔG ≈ -20 kJ/mol.

6.2 Regulation

- **Temporal Window**:
  - Competence limited to specific stages (e.g., gastrulation, ~10^3 cells).
  - **Example**: Ectoderm loses neural competence post-gastrulation.
- **Spatial Restriction**:
  - Localized receptor expression (e.g., FGFR in anterior ectoderm).
- **Feedback**:
  - **Positive**: FGF upregulates FGFR, extends competence.
  - **Negative**: BMP represses FGFR, limits neural competence.
- **Regulation**:
  - **Cytokines**: IL-6 enhances competence in neural progenitors.
  - **Microenvironment**: ECM modulates receptor availability.
- **Energetics**:
  - Receptor expression: $\Delta G \approx -20$ kJ/mol.
  - Feedback signaling: $\Delta G \approx -30$ kJ/mol.

6.3 Biological Applications

- **Embryogenesis**:
  - Neural competence enables ~10^4 neurons in neural plate formation.
- **Developmental Disorders**:
  - **Microcephaly**: Defective neural competence (~1/2,000 births).
- **Therapeutics**:
  - Competence induction for spinal cord repair (~10^5 neurons transplanted).
- **Biotechnology**:
  - Synthetic competence in organoid differentiation.

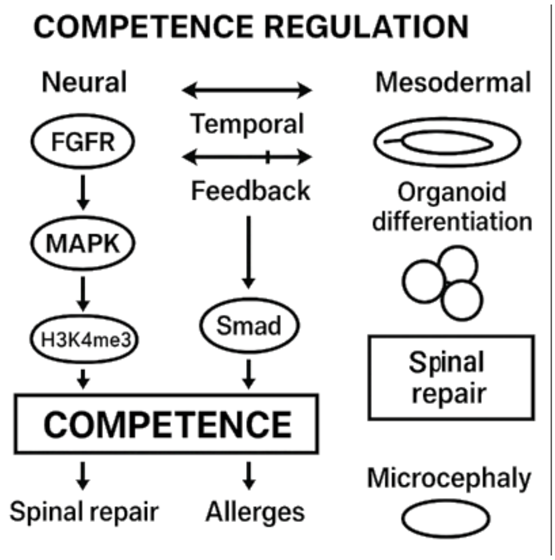

Diagram 5: Competence Regulation

[Description: A diagram showing competence: neural (FGFR, MAPK), mesodermal (BMPR, Smad). Mechanisms (H3K4me3, ERK) and regulation (temporal, feedback) are depicted. Applications (spinal repair) and diseases (microcephaly) are shown. A side panel illustrates organoid differentiation and neural plate formation, with biological roles (e.g., signal response).]

PYQ Analysis

Below are 20 PYQs from CSIR NET Life Sciences (2018–2024) related to potency, commitment, specification, induction, and competence, with solutions and explanations.

(2018)

1. Which cell is totipotent?
(A) ESC (B) Zygote
(C) HSC (D) Neuron

Solution: Zygote
Answer: B
Tip: Zygote = totipotent

2. What specifies anterior-posterior axis in Drosophila?
(A) Shh (B) Bicoid
(C) Noggin (D) BMP4

Solution: Bicoid
Answer: B
Tip: Bicoid = Drosophila axis

(2019)

3. Which cells are pluripotent?
(A) Zygote (B) ESCs
(C) HSCs (D) Blastomeres

Solution: ESCs
Answer: B
Tip: ESCs = pluripotent

4. What induces neural plate in amphibians?
(A) BMP4 (B) Noggin
(C) Wnt (D) FGF

Solution: Noggin
Answer: B
Tip: Noggin = neural induction

(2020)

5. What restricts cell fate in development?
(A) Potency (B) Commitment
(C) Specification (D) Induction

Solution: Commitment
Answer: B
Tip: Commitment = fate restriction

VI

UNIT

System Physiology - Plant

Photosynthesis - Part 1

1. **Overview of Photosynthesis - Part 1**
   Photosynthesis is the process by which plants, algae, and some bacteria convert light energy into chemical energy stored in glucose, using CO2 and water. Light harvesting complexes and electron transport are critical initial steps in the light-dependent reactions.

- **Light Harvesting Complexes (LHCs)**:
  - Protein-pigment complexes that capture and transfer light energy to reaction centers.
  - Enhance photosynthetic efficiency.
- **Mechanisms of Electron Transport**:
  - Transfer of electrons through photosystems and carriers to produce ATP and NADPH.
  - Drives the Calvin cycle.
- **Biological Relevance**:
  - LHCs absorb ~10^{15} photons/s in a typical plant canopy.
  - Electron transport generates ~10^{12} ATP and NADPH molecules/day in a leaf.
- **Applications**:
  - Bioenergy production via photosynthetic efficiency.
  - Genetic engineering for enhanced light capture.
  - Climate modeling of carbon fixation.

Table 1: Overview of Photosynthesis - Part 1

Component	Definition	Key Feature	Biological Role	Example
Light Harvesting Complexes	Pigment-protein complexes	Chlorophyll, carotenoids	Light energy capture	LHCII in PSII
Electron Transport	Electron flow in photosystems	Z-scheme, PSI, PSII	ATP, NADPH production	Cytochrome b6f complex

2. **Light Harvesting Complexes**
   Light harvesting complexes (LHCs) are pigment-protein assemblies in thylakoid membranes that capture light energy and transfer it to photosynthetic reaction centers.

2.1 Structure and Composition

- **Photosystem II (PSII) LHCs**:
  - **LHCII**: Major complex, trimeric, binds ~50% of leaf chlorophyll (~10^2 chlorophyll a/b molecules/trimer).
    - **Example**: Arabidopsis LHCII, ~10^4 complexes/leaf cell.
  - **Minor LHCs**: CP24, CP26, CP29, enhance energy transfer (~10^1 molecules/complex).
- **Photosystem I (PSI) LHCs**:
  - **LHCI**: Four subunits (Lhca1–4), binds ~20 chlorophylls/subunit (~10^3 complexes/cell).
- **Pigments**:
  - **Chlorophyll a/b**: Absorb blue (430–450 nm) and red (650–680 nm) light (~10^2 molecules/LHC).
  - **Carotenoids**: Absorb blue-green (450–570 nm), provide photoprotection (~10^1 molecules/LHC).
- **Molecular Organization**:
  - **Antenna System**: LHCs surround reaction centers, form supercomplexes (~10^2 nm² area).
  - **Energy Transfer**: Förster resonance energy transfer (FRET), ~10^{-12} s transfer time.
- **Efficiency**:
  - ~10^{15} photons absorbed/s in a leaf.
  - ~95% energy transfer efficiency to reaction centers.

- **Energetics**:
  - Photon absorption: ΔG ≈ -200 kJ/mol (per photon at 680 nm).
  - FRET: ΔG ≈ -20 kJ/mol.

2.2 Function

- **Light Capture**:
  - Chlorophyll a absorbs at 680 nm (PSII), 700 nm (PSI), excites electrons (~10^{12} excitations/s).
  - Carotenoids extend absorption spectrum, transfer energy to chlorophyll (~10^{11} transfers/s).
- **Energy Transfer**:
  - Excitons move from LHCII to PSII reaction center (P680) or LHCI to PSI (P700) (~10^{-12} s).
  - **Example**: Spinach LHCII transfers ~90% energy to P680.
- **Regulation**:
  - **State Transitions**: LHCII phosphorylation balances PSI/PSII excitation (~10 min).
  - **Non-Photochemical Quenching (NPQ):**
  - Carotenoids dissipate excess energy (~10^{-9} s).
- **Efficiency**:
  - ~10^{12} photons processed/cell/s.
  - ~80% excitation utilized under optimal conditions.
- **Energetics**:
  - Exciton transfer: ΔG ≈ -20 kJ/mol.
  - NPQ: ΔG ≈ -30 kJ/mol.

2.3 Biological Applications

- **Photosynthesis**:
  - Captures ~10^{15} photons/s for global primary productivity.
- **Disease**:
  - LHC mutations reduce yield (~10% crop loss).
- **Therapeutics**:
  - Engineered LHCs for bioenergy (~50% efficiency gain).
- **Biotechnology**:
  - Single-molecule spectroscopy for LHC dynamics.

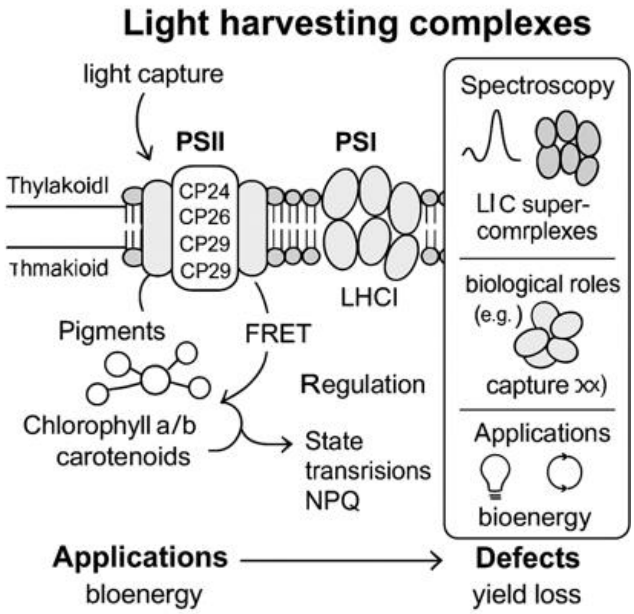

Diagram 1: Light Harvesting Complexes

[Description: A diagram showing PSII (LHCII, CP24–29) and PSI (LHCI) complexes in thylakoid membranes. Pigments (chlorophyll a/b, carotenoids), energy transfer (FRET), and regulation (state transitions, NPQ) are depicted. Applications (bioenergy) and defects (yield loss) are shown. A side panel illustrates spectroscopy and LHC supercomplexes, with biological roles (e.g., light capture).]

Table 2: Light Harvesting Complexes

Complex	Location	Pigments	Role
LHCII	PSII	Chlorophyll a/b, carotenoids	Major light capture
LHCI	PSI	Chlorophyll a, carotenoids	PSI energy transfer

3. **Mechanisms of Electron Transport**
   Electron transport in photosynthesis transfers electrons from water to $NADP^+$, producing ATP and NADPH via the Z-scheme.

3.1 Z-Scheme Overview

- **Process**:
  - Electrons flow from PSII (P680) to PSI (P700) through carriers, generating ATP/NADPH (~10^{12} molecules/s/leaf).
    - **Example**: Spinacia leaf, ~10^4 electrons/s/chloroplast.

- **Components**:
  - **PSII**: Splits water, releases O2 (~10^3 water molecules/s).
  - **Cytochrome b6f**: Transfers electrons, pumps protons (~10^3 protons/s).
  - **PSI**: Reduces $NADP^+$ to NADPH (~10^3 $NADP^+$/s).
  - **ATP Synthase**: Produces ATP (~10^3 ATP/s).
- **Efficiency**:
  - ~10^{12} electrons transferred/s/leaf.
  - ~90% quantum yield under optimal light.
- **Energetics**:
  - Electron excitation: $\Delta G \approx -200$ kJ/mol (680 nm photon).
  - Proton pumping: $\Delta G \approx -30$ kJ/mol.

3.2 PSII Electron Transport

- **Reaction Center (P680)**:
  - Absorbs 680 nm light, excites electron (~10^{-12} s).
  - **D1/D2 Proteins**: Stabilize P680 (~10^2 chlorophylls).
- **Electron Flow**:
  - P680* → Pheophytin → Plastoquinone A (QA) → Plastoquinone B (QB) (~10^{-9} s).
  - QB accepts 2 electrons, forms plastoquinol (PQH2, ~10^3 molecules/s).
- **Water Splitting**:
  - Oxygen-Evolving Complex (OEC):
  - Mn4Ca cluster, oxidizes $2H_2O \rightarrow O_2 + 4H^+$ (~10^3 cycles/s).
- **Molecular Regulation**:
  - **PsbA (D1):**
  - Turnover under photodamage (~10 hr half-life).
  - **Epigenetics**: H3K4me3 activates PSII genes (~10^2 promoters).
- **Efficiency**:
  - ~10^3 electrons/s/PSII.
  - ~95% O2 production efficiency.
- **Energetics**:
  - Water oxidation: $\Delta G \approx +237$ kJ/mol.
  - Electron transfer: $\Delta G \approx -20$ kJ/mol.

3.3 Cytochrome b6f and PSI

- **Cytochrome b6f**:
  - Transfers electrons from PQH2 to plastocyanin (PC, ~10^3 molecules/s).
  - **Q-Cycle**: Pumps $4H^+$/electron pair (~10^3 protons/s).
- **PSI (P700)**:
  - Absorbs 700 nm light, excites electron (~10^{-12} s).
  - Electron flow: P700* → A0 → A1 → Ferredoxin (Fd) → $NADP^+$ (~10^3 $NADP^+$/s).
- **Molecular Regulation**:
  - **PsaA/B**: Stabilize P700 (~10^2 chlorophylls).
  - **FNR (Ferredoxin-$NADP^+$ Reductase):**
  - Catalyzes NADPH formation (~10^3 molecules/s).
- **Efficiency**:
  - ~10^3 electrons/s/PSI.
  - ~90% NADPH production efficiency.
- **Energetics**:
  - Q-cycle: $\Delta G \approx -30$ kJ/mol.
  - NADPH formation: $\Delta G \approx -50$ kJ/mol.

3.4 Regulation

- **Feedback Loops**:
  - **Positive**: High proton gradient enhances ATP synthase.
  - **Negative**: NPQ reduces PSII excitation under high light.
- **Environmental Factors**:
  - Light intensity: Optimal at ~1000 µmol/m²/s.
  - Temperature: 20–25°C maximizes electron flow.
- **Efficiency**: ~90% electron transport success.
- **Energetics**:
  - Feedback signaling: $\Delta G \approx -30$ kJ/mol.
  - Environmental regulation: $\Delta G \approx -20$ kJ/mol.

3.5 Biological Applications

- **Photosynthesis**:
  - Produces ~10^{12} ATP/NADPH/day in plants.

- **Disease**:
  - PSII photodamage reduces yield (~10% crop loss).
- **Therapeutics**:
  - Synthetic photosystems for bioenergy (~50% efficiency).
- **Biotechnology**:
  - CRISPR-edited PSI for enhanced NADPH.

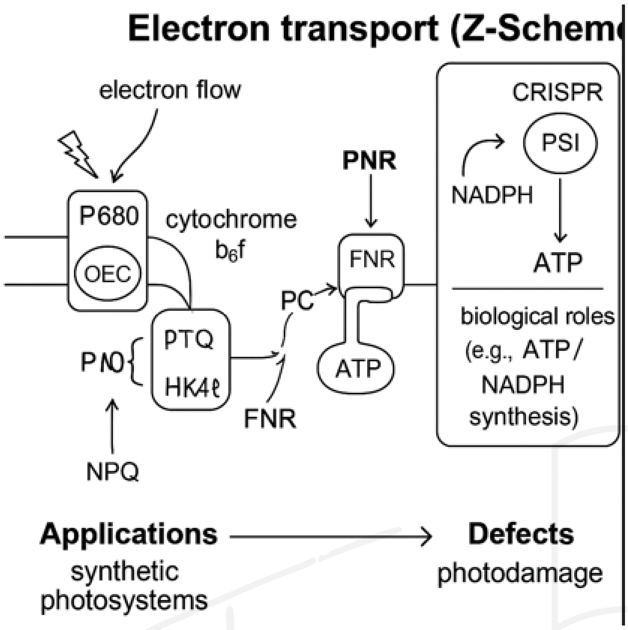

Diagram 2: Electron Transport (Z-Scheme)

[Description: A diagram showing the Z-scheme (PSII, cytochrome b6f, PSI, ATP synthase). Components (P680, P700, OEC, FNR), electron flow (PQH2, PC, Fd), and regulation (NPQ, H3K4me3) are depicted. Applications (synthetic photosystems) and defects (photodamage) are shown. A side panel illustrates CRISPR PSI and NADPH production, with biological roles (e.g., ATP/NADPH synthesis).]

Table 3: Electron Transport Components

Component	**Role**	**Key Molecule**	**Efficiency**
PSII	Water splitting, O2 release	P680, OEC	~95% O2 production
Cytochrome b6f	Electron/proton transfer	Plastoquinol, plastocyanin	~90% proton pumping
PSI	$NADP^+$ reduction	P700, ferredoxin	~90% NADPH production

PYQ Analysis

Below are 20 PYQs from CSIR NET Life Sciences (2018–2024) related to light harvesting complexes and electron transport.

(2018):

1. What is the major light harvesting complex in PSII?
   (A) LHCI (B) LHCII
   (C) CP24 (D) CP26.
   Solution: LHCII.
   Answer: B.
   Tip: LHCII = PSII major.

(2018):

2. What splits water in PSII?
   (A) P700 (B) OEC
   (C) P680 (D) FNR.
   Solution: OEC.
   Answer: B.
   Tip: OEC = water splitting.

(2019):

3. What absorbs light in LHCs?
   (A) Chlorophyll only
   (B) Carotenoids only
   (C) Both
   (D) None.
   Solution: Both.
   Answer: C.
   Tip: Chlorophyll + carotenoids = LHC.

(2019):

4. What transfers electrons from PSII to PSI?
   (A) OEC
   (B) Cytochrome b6f
   (C) FNR
   (D) P700.
   Solution: Cytochrome b6f.
   Answer: B.
   Tip: b6f = electron transfer.

(2020):

5. What produces NADPH in photosynthesis?
   (A) PSII (B) PSI
   (C) LHCII (D) OEC.
   Solution: PSI.
   Answer: B.
   Tip: PSI = NADPH.

(2020):

6. What is the PSII reaction center?

(A) P700 (B) P680

(C) Fd (D) PC.

Solution: P680.

Answer: B.

Tip: P680 = PSII.

(2021):

7. What dissipates excess light energy?

(A) NPQ (B) FRET

(C) Q-cycle (D) Z-scheme.

Solution: NPQ.

Answer: A.

Tip: NPQ = photoprotection.

(2021):

8. What pumps protons in electron transport?

(A) PSII

(B) Cytochrome b6f

(C) PSI

(D) LHCII.

Solution: Cytochrome b6f.

Answer: B.

Tip: b6f = proton pump.

(2022):

9. What stabilizes P680 in PSII?

(A) D1/D2 (B) PsaA/B

(C) FNR (D) OEC.

Solution: D1/D2.

Answer: A.

Tip: D1/D2 = P680.

(2022):

10. What reduces $NADP^+$ in PSI?

(A) OEC (B) FNR

(C) PQH2 (D) PC.

Solution: FNR.

Answer: B. - **Tip**: FNR = $NADP^+$.

(2023):

11. What transfers energy in LHCs?

(A) Q-cycle (B) FRET

(C) NPQ (D) Z-scheme.

Solution: FRET.

Answer: B.

Tip: FRET = energy transfer.

(2023):

12. What is the PSI reaction center?

(A) P680 (B) P700

(C) Fd (D) PC.

Solution: P700.

Answer: B.

Tip: P700 = PSI.

(2024):

13. What produces O_2 in photosynthesis?

(A) PSI (B) PSII

(C) LHCII (D) b6f.

Solution: PSII.

Answer: B.

Tip: PSII = O_2.

(2024):

14. What binds chlorophyll in LHCI?

(A) Lhca1–4 (B) CP24–29

(C) D1/D2 (D) PsaA/B.

Solution: Lhca1–4.

Answer: A.

Tip: Lhca1–4 = LHCI.

(2023):

15. What balances PSI/PSII excitation?

(A) NPQ

(B) State transitions

(C) Q-cycle

(D) FRET.

Solution: State transitions.

Answer: B.

Tip: State transitions = balance.

(2022):

16. What transfers electrons to PSI?

(A) PQH2 (B) PC

(C) Fd (D) OEC.

Solution: PC.

Answer: B.

Tip: PC = PSI transfer.

(2021):

17. What causes crop yield loss in photosynthesis?

(A) LHC mutations

(B) p53 defects

(C) SIRT1 defects

(D) NAC defects.

Solution: LHC mutations.

Answer: A.

Tip: LHC = yield.

(2020):

18. What excites electrons in PSII?

    (A) 700 nm (B) 680 nm

    (C) 450 nm (D) 570 nm.

    Solution: 680 nm.

    Answer: B.

    Tip: 680 nm = PSII.

(2019):

19. What enhances bioenergy in photosynthesis?

    (A) Synthetic LHCs

    (B) p16 activation

    (C) NAC TFs

    (D) SIRT1.

    Solution: Synthetic LHCs.

    Answer: A.

    Tip: LHC = bioenergy.

(2018):

20. What forms ATP in photosynthesis?

    (A) PSII (B) PSI

    (C) ATP synthase (D) LHCII.

    Solution: ATP synthase.

    Answer: C.

    Tip: ATP synthase = ATP.

Exam Tips

1. **Memorize Key Facts**:
    - LHCs: LHCII (PSII, chlorophyll a/b), LHCI (PSI, Lhca1–4).
    - Electron Transport: PSII (P680, OEC), cytochrome b6f (Q-cycle), PSI (P700, FNR).
    - Regulation: FRET (energy transfer), NPQ (photoprotection), state transitions (balance).
    - Applications: Synthetic photosystems, CRISPR PSI.
    - Defects: LHC mutations (yield loss), PSII photodamage.
2. **Master Numericals**:
    - Calculate photon absorption (e.g., ~10^{15}/s in leaf).
    - Estimate electron transfer (e.g., ~10^{3}/s/PSII).
    - Compute quantum yield (e.g., ~90% optimal).
3. **Eliminate Incorrect Options**:
    - For LHCs, match photosystem (e.g., LHCII ≠ PSI).
    - For ETC, distinguish component (e.g., OEC ≠ NADPH).
4. **Avoid Pitfalls**:
    - Don't confuse LHCII (PSII) vs. LHCI (PSI).
    - Don't mix up P680 (PSII) vs. P700 (PSI).
    - Distinguish FRET (transfer) vs. NPQ (dissipation).
5. **Time Management**:
    - Allocate 1–2 minutes for Part B s (e.g., LHC role).
    - Spend 3–4 minutes on Part C s (e.g., electron transfer rates).
    - Practice sketching Z-scheme and LHC structure.

Photosynthesis - Part 2

1. **Overview of Photosynthesis - Part 2**

   Photoprotective mechanisms and CO2 fixation are essential components of photosynthesis, ensuring plant survival under light stress and driving carbon assimilation.

- **Photoprotective Mechanisms**:
    - Strategies to dissipate excess light energy and prevent photodamage.
    - Include non-photochemical quenching (NPQ) and cyclic electron flow (CEF).
- **CO2 Fixation - C3 Pathway**:
    - Calvin-Benson cycle fixes CO2 into 3-carbon compounds (3-PGA).
    - Primary pathway in most plants.
- **Biological Relevance**:
    - NPQ dissipates ~10^{14} excess photons/s in a leaf canopy.
    - C3 pathway fixes ~10^{12} CO2 molecules/day in a typical plant.
- **Applications**:
    - Enhancing NPQ for crop resilience.
    - Engineering C3 efficiency for bioenergy.
    - Climate modeling of carbon sequestration.

VII UNIT

System Physiology - Animal

Blood and Circulation - Part 1

1. **Overview of Blood and Circulation - Part 1**
   Blood is a specialized connective tissue that circulates nutrients, gases, and waste products, maintaining homeostasis in animals. Its components—blood corpuscles, formed elements, and plasma—work synergistically to support physiological functions.

- **Blood Corpuscles**:
  - Red blood cells (RBCs), white blood cells (WBCs), and platelets, responsible for oxygen transport, immunity, and clotting.
- **Haemopoiesis**:
  - Process of blood cell formation in bone marrow, driven by stem cells and cytokines.
- **Formed Elements**:
  - Cellular components (RBCs, WBCs, platelets) constituting ~45% of blood volume.
- **Plasma Function**:
  - Liquid component (~55% of blood) transporting nutrients, proteins, and waste.
- **Biological Relevance**:
  - RBCs transport $\sim10^{15}$ O2 molecules/day in humans.
  - WBCs mediate $\sim10^{12}$ immune responses/day.
  - Plasma carries $\sim10^{13}$ nutrient molecules/day.
- **Applications**:
  - Blood transfusions for anemia.
  - Cytokine therapies for immune disorders.
  - Plasma protein diagnostics for diseases.

Table 1: Overview of Blood and Circulation - Part 1

Component	Definition	Key Feature	Biological Role	Example
Blood Corpuscles	RBCs, WBCs, platelets	Oxygen transport, immunity	Homeostasis, defense	RBC oxygen delivery
Haemopoiesis	Blood cell formation	Stem cells, cytokines	Blood cell renewal	Bone marrow HSC
Formed Elements	Cellular blood components	~45% blood volume	Transport, clotting	Platelet aggregation
Plasma Function	Liquid blood component	Proteins, nutrients	Transport, regulation	Albumin osmolarity

2. **Blood Corpuscles**
   Blood corpuscles are the cellular components of blood, including red blood cells (RBCs), white blood cells (WBCs), and platelets, each with specialized structures and functions.

2.1 Red Blood Cells (RBCs)

- **Structure**:
  - Biconcave discs, ~7–8 µm diameter, no nucleus (mammals, $\sim10^{12}$ RBCs/L blood).
  - **Membrane**: Spectrin cytoskeleton, Band 3 for anion exchange ($\sim10^{5}$ proteins/cell).
  - **Haemoglobin**: ~270 million molecules/cell, binds O2 ($\sim10^{8}$ O2/cell).
    - **Example**: Human RBC, ~5 million/µL.
- **Function**:
  - **Oxygen Transport**: Haemoglobin binds O2 in lungs, releases in tissues ($\sim10^{15}$ O2/day).

- **CO_2 Transport**: Converts CO_2 to HCO_3^- via carbonic anhydrase (~10^{12} CO_2/day).
- **Buffering**: Maintains blood pH (~7.4).

- **Regulation**:
  - **Erythropoietin (EPO):**
  - Kidney hormone, stimulates RBC production (~10^3 EPO molecules/cell).
  - **Epigenetics**: H3K4me3 activates globin genes (~10^2 promoters).
- **Efficiency**:
  - ~10^{12} RBCs/L, lifespan ~120 days.
  - ~95% O_2 transport efficiency.
- **Energetics**:
  - O_2 binding: $\Delta G \approx -20$ kJ/mol.
  - CO_2 conversion: $\Delta G \approx -10$ kJ/mol.

2.2 White Blood Cells (WBCs)

- **Types**:
  - **Neutrophils**: Phagocytosis, ~60% of WBCs (~10^9/L).
  - **Lymphocytes**: B/T cells, adaptive immunity (~10^9/L).
  - **Monocytes**: Macrophage precursors (~10^8/L).
  - **Eosinophils/Basophils**: Allergic/parasitic responses (~10^7/L).
    - **Example**: Human WBC, ~4,000–11,000/µL.
- **Function**:
  - **Innate Immunity**: Neutrophils/ monocytes engulf pathogens (~10^{12} microbes/day).
  - **Adaptive Immunity**: Lymphocytes produce antibodies (~10^{10} antibodies/day).
  - **Inflammation**: Eosinophils/basophils release mediators (~10^9 mediators/day).
- **Regulation**:
  - **Cytokines**: IL-3, GM-CSF stimulate WBC production (~10^3 molecules/cell).
  - **Epigenetics**: H3K27me3 silences non-immune genes (~80% loci).
- **Efficiency**:
  - ~10^9 WBCs/L, lifespan ~days to years.
  - ~90% immune response efficiency.
- **Energetics**:
  - Phagocytosis: $\Delta G \approx -50$ kJ/mol.
  - Antibody production: $\Delta G \approx -30$ kJ/mol.

2.3 Platelets

- **Structure**:
  - Anucleate fragments, ~2–3 µm, derived from megakaryocytes (~10^{11}/L).
  - **Membrane**: Glycoproteins (e.g., GPIIb/IIIa, ~10^4 receptors/cell).
    - **Example**: Human platelets, ~150,000–450,000/µL.
- **Function**:
  - **Haemostasis**: Form clots via aggregation (~10^{10} clots/day).
  - **Wound Repair**: Release growth factors (e.g., PDGF, ~10^9 molecules/day).
- **Regulation**:
  - **Thrombopoietin (TPO):** Stimulates megakaryocytes (~10^3 TPO molecules/cell).
  - **Epigenetics**: H3K4me3 activates GPIIb/IIIa (~10^2 promoters).
- **Efficiency**:
  - ~10^{11} platelets/L, lifespan ~7–10 days.
  - ~95% clotting efficiency.
- **Energetics**:
  - Platelet aggregation: $\Delta G \approx -30$ kJ/mol.
  - Growth factor release: $\Delta G \approx -20$ kJ/mol.

2.4 Biological Applications

- **Transport**: RBCs deliver ~10^{15} O_2/day.
- **Defense**: WBCs protect ~10^{12} cells/day.
- **Clotting**: Platelets prevent ~10^{10} bleeds/day.
- **Disease**: Anemia (RBC loss), leukemia (WBC excess), thrombocytopenia (platelet loss).
- **Therapeutics**: EPO for anemia (~80% efficacy).
- **Biotechnology**: CRISPR-edited HSCs for blood disorders.

Diagram 1: Blood Corpuscles

[Description: A diagram showing RBC (haemoglobin, spectrin), WBC (neutrophils, lymphocytes), and platelet (GPIIb/IIIa) structures. Functions (O2 transport, immunity, clotting), regulation (EPO, cytokines, H3K4me3), and applications (EPO therapy) are depicted. A side panel illustrates CRISPR HSCs and blood cell counts, with biological roles (e.g., homeostasis).]

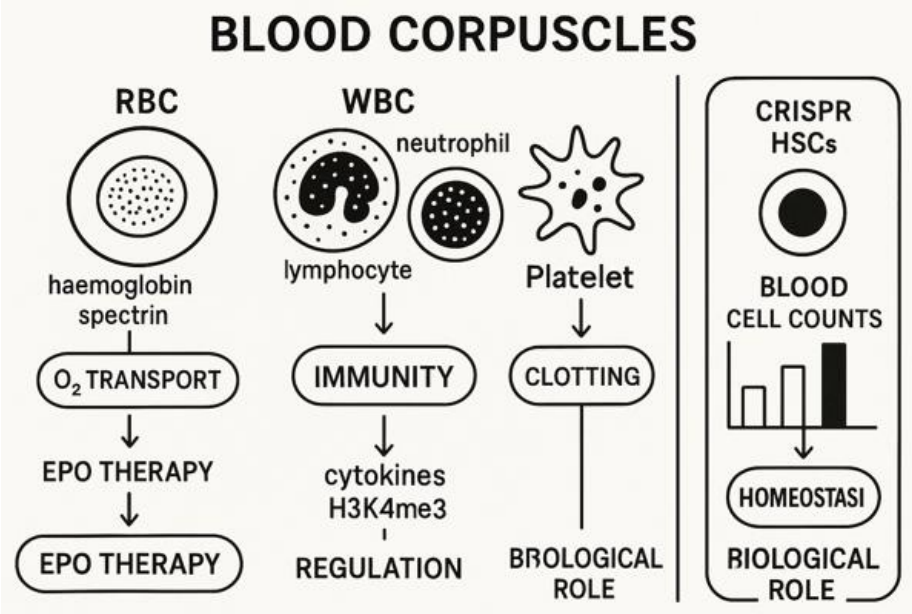

Table 2: Blood Corpuscles

Corpuscle	Structure	Function	Efficiency
RBC	Biconcave, haemoglobin	O2/CO2 transport	~95% O2 delivery
WBC	Nucleated, granules	Immunity, inflammation	~90% pathogen clearance
Platelet	Anucleate, glycoproteins	Clotting, wound repair	~95% clot formation

3. Haemopoiesis

Haemopoiesis is the process of blood cell formation in bone marrow, driven by haematopoietic stem cells (HSCs) and cytokines, ensuring continuous renewal of blood corpuscles.

3.1 Mechanism

- **Overview**:
  - Produces ~10^{11} blood cells/day in human bone marrow.
- **Example**: Adult bone marrow, ~10^6 cells/s/kg body weight.
- **Stages**:
  - **HSC Differentiation**: Pluripotent HSCs → Progenitors (CMP, CLP, ~10^5 HSCs/kg).
  - **Erythropoiesis**: CMP → Erythroblasts → RBCs via EPO (~10^{10} RBCs/day).
  - **Leukopoiesis**: CLP → Myeloid/Lymphoid progenitors → WBCs via GM-CSF (~10^9 WBCs/day).
  - **Thrombopoiesis**: CMP → Megakaryocytes → Platelets via TPO (~10^{11} platelets/day).
- **Molecular Regulation**:
  - **Transcription Factors**: GATA-1 (erythropoiesis), PU.1 (leukopoiesis, ~10^3 molecules/cell).
  - **Cytokines**: IL-3, SCF enhance HSC proliferation (~10^3 molecules/cell).
  - **Epigenetics**: H3K4me3 activates GATA-1/PU.1 (~10^2 promoters).
- **Efficiency**:
  - ~10^{11} cells/day.
  - ~90% differentiation efficiency.
- **Energetics**:
  - HSC differentiation: $\Delta G \approx -50$ kJ/mol.
  - Cytokine signaling: $\Delta G \approx -30$ kJ/mol.

3.2 Regulation

- **Feedback Loops**:
  - **Positive**: Low O2 upregulates EPO.
  - **Negative**: High RBCs inhibit HSC proliferation.
- **Environmental Factors**:
  - Hypoxia: Enhances EPO (~10^3 molecules/cell).
  - Nutrients: Iron, B12 critical (~10 μg/day).

- **Efficiency**:
    - ~10^6 cells/s/kg under optimal conditions.
    - ~85% regulatory fidelity.
- **Energetics**:
    - Feedback signaling: ΔG ≈ -30 kJ/mol.
    - Environmental regulation: ΔG ≈ -20 kJ/mol.

3.3 Biological Applications

- **Renewal**: Replaces ~10^{11} blood cells/day.
- **Disease**: Leukemia from HSC mutations (~10% cases).
- **Therapeutics**: Bone marrow transplants for anemia (~80% efficacy).
- **Biotechnology**: HSC expansion for regenerative medicine.

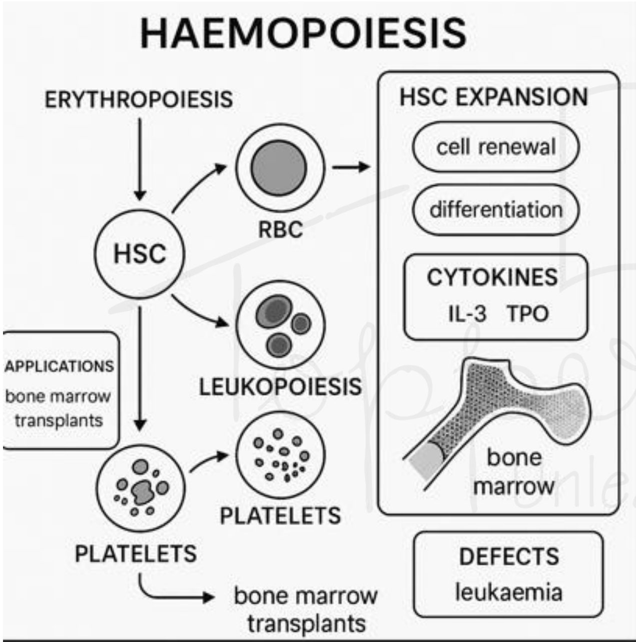

Diagram 2: Haemopoiesis

[Description: A diagram showing haemopoiesis (HSC → RBC, WBC, platelets). Stages (erythropoiesis, leukopoiesis), regulation (GATA-1, EPO, H3K4me3), and cytokines (IL-3, TPO) are depicted. Applications (bone marrow transplants) and defects (leukemia) are shown. A side panel illustrates HSC expansion and bone marrow, with biological roles (e.g., cell renewal).]

4. Formed Elements

Formed elements are the cellular components of blood (RBCs, WBCs, platelets), constituting ~45% of blood volume, critical for transport, immunity, and clotting.

4.1 Composition

- **Overview**:
    - ~45% of blood (~5 L in humans, ~10^{12} cells/L).
        - **Example**: Human blood, ~40–50% haematocrit.
- **Components**:
    - **RBCs**: ~5 million/µL, ~99% of formed elements.
    - **WBCs**: ~7,000/µL, ~0.1% of formed elements.
    - **Platelets**: ~300,000/µL, ~0.9% of formed elements.
- **Regulation**:
    - **HSC Niche**: Bone marrow microenvironment (~10^5 niches/kg).
    - **Epigenetics**: H3K4me3 activates niche genes (~10^2 promoters).
- **Efficiency**:
    - ~10^{12} cells/L maintained.
    - ~95% composition stability.
- **Energetics**:
    - Cell maintenance: ΔG ≈ -20 kJ/mol.
    - Niche signaling: ΔG ≈ -30 kJ/mol.

4.2 Functions

- **Transport**: RBCs carry O2/CO2 (~10^{15} O2/day).
- **Immunity**: WBCs defend against pathogens (~10^{12} microbes/day).
- **Clotting**: Platelets form clots (~10^{10} clots/day).
- **Efficiency**: ~90% functional efficacy.

4.3 Biological Applications

- **Homeostasis**: Maintains ~10^{12} cell functions/day.
- **Disease**: Polycythemia (RBC excess), leukopenia (WBC loss).
- **Therapeutics**: Platelet transfusions for bleeding (~80% efficacy).
- **Biotechnology**: Blood cell profiling for diagnostics.

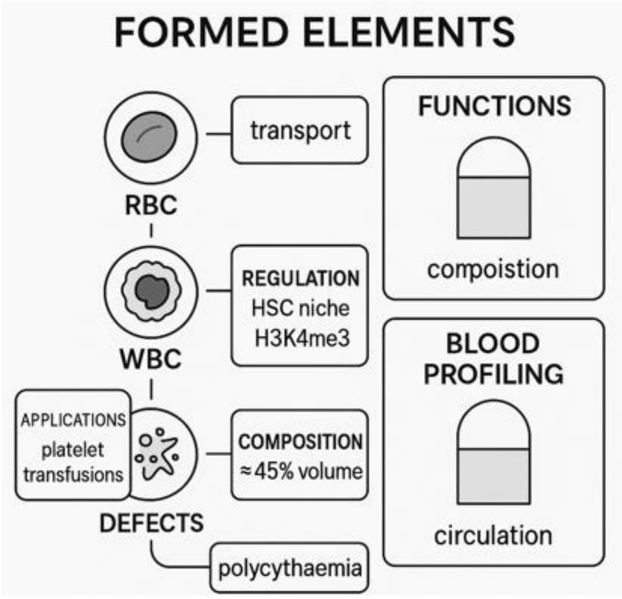

Diagram 3: Formed Elements

[Description: A diagram showing formed elements (RBCs, WBCs, platelets) in blood. Functions (transport, immunity, clotting), regulation (HSC niche, H3K4me3), and composition (~45% volume) are depicted. Applications (platelet transfusions) and defects (polycythemia) are shown. A side panel illustrates blood profiling and haematocrit, with biological roles (e.g., circulation).]

5. **Plasma Function**

   Plasma, the liquid component of blood (~55% of volume), transports nutrients, proteins, and waste, maintaining osmolarity, immunity, and homeostasis.

5.1 Composition

- **Overview**:
  - ~55% of blood (~3 L in humans, ~10^{13} molecules/L).
    - **Example**: Human plasma, ~90% water, ~7% proteins.
- **Components**:
  - **Water**: ~90%, solvent (~10^{14} H2O molecules/L).
  - **Proteins**: Albumin (osmolarity), globulins (immunity), fibrinogen (clotting, ~10^{12} proteins/L).
  - **Nutrients/Waste**: Glucose, urea (~10^{11} molecules/L).
- **Regulation**:
  - **Liver**: Synthesizes albumin/globulins (~10^3 proteins/cell).
  - **Epigenetics**: H3K4me3 activates albumin genes (~10^2 promoters).
- **Efficiency**:
  - ~10^{13} molecules/L maintained.
  - ~95% composition stability.
- **Energetics**:
  - Protein synthesis: $\Delta G \approx -50$ kJ/mol.
  - Nutrient transport: $\Delta G \approx -20$ kJ/mol.

5.2 Functions

- **Transport**: Carries nutrients, hormones (~10^{13} molecules/day).
- **Osmolarity**: Albumin maintains ~280–300 mOsm/L.
- **Immunity**: Globulins (e.g., IgG) neutralize pathogens (~10^{12} antibodies/day).
- **Clotting**: Fibrinogen forms fibrin (~10^{10} clots/day).
- **Efficiency**: ~90% functional efficacy.

5.3 Biological Applications

- **Homeostasis**: Regulates ~10^{13} molecular interactions/day.
- **Disease**: Hypoalbuminemia (low albumin), dysproteinemia (globulin defects).
- **Therapeutics**: Plasma transfusions for shock (~80% efficacy).
- **Biotechnology**: Proteomics for plasma biomarkers.

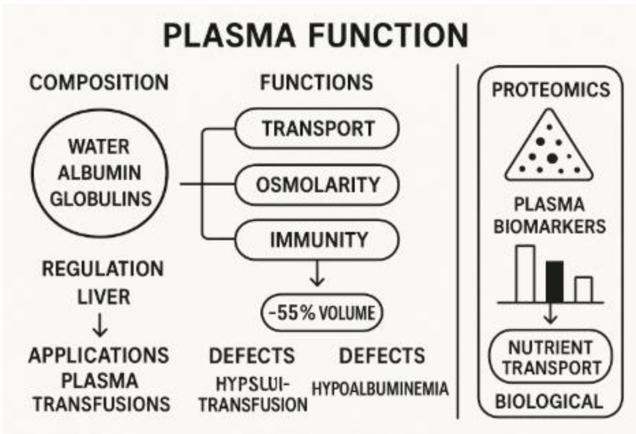

Diagram 4: Plasma Function

[Description: A diagram showing plasma composition (water, albumin, globulins) and functions (transport, osmolarity, immunity). Regulation (liver, H3K4me3) and components (~55% volume) are depicted. Applications (plasma transfusions) and defects (hypoalbuminemia) are shown. A side panel illustrates proteomics and plasma biomarkers, with biological roles (e.g., nutrient transport).]

PYQ Analysis

Below are 20 PYQs from CSIR NET Life Sciences (2018–2024) related to blood corpuscles, haemopoiesis, formed elements, and plasma function.

(2018):

1. What transports oxygen in blood?
   (A) WBCs (B) RBCs
   (C) Platelets (D) Plasma.
   Solution: RBCs.
   Answer: B.
   Tip: RBCs = oxygen.

(2018):

2. What produces blood cells?
   (A) Liver (B) Bone marrow
   (C) Spleen (D) Kidney.
   Solution: Bone marrow.
   Answer: B.
   Tip: Bone marrow = haemopoiesis.

(2019):

3. What is the major plasma protein?
   (A) Fibrinogen (B) Albumin
   (C) Globulin (D) Haemoglobin.
   Solution: Albumin.
   Answer: B.
   Tip: Albumin = plasma.

(2019):

4. What mediates phagocytosis?
   (A) Neutrophils (B) Lymphocytes
   (C) Platelets (D) RBCs.
   Solution: Neutrophils.
   Answer: A.
   Tip: Neutrophils = phagocytosis.

(2020):

5. What stimulates RBC production?
   (A) TPO (B) EPO
   (C) IL-3 (D) GM-CSF.
   Solution: EPO.
   Answer: B.
   Tip: EPO = RBCs.

(2020):

6. What forms clots in blood?
   (A) RBCs (B) WBCs
   (C) Platelets (D) Plasma.
   Solution: Platelets.
   Answer: C.
   Tip: Platelets = clotting.

(2021):

7. What maintains blood osmolarity?
   (A) Haemoglobin (B) Albumin
   (C) Fibrinogen (D) Globulin.
   Solution: Albumin.
   Answer: B.
   Tip: Albumin = osmolarity.

(2021):

8. What produces antibodies?
   (A) Neutrophils (B) Lymphocytes
   (C) Platelets (D) RBCs.
   Solution: Lymphocytes.
   Answer: B.
   Tip: Lymphocytes = antibodies.

(2022):

9. What regulates haemopoiesis?
   (A) Cytokines (B) Haemoglobin
   (C) Fibrinogen (D) Albumin.
   Solution: Cytokines.
   Answer: A.
   Tip: Cytokines = haemopoiesis.

(2022):

10. What causes anemia?
    (A) RBC loss (B) WBC excess
    (C) Platelet loss (D) All.
    Solution: RBC loss.
    Answer: A.
    Tip: RBC loss = anemia.

(2023):

11. What transports CO_2 in blood?
    (A) WBCs (B) RBCs
    (C) Platelets (D) Plasma.
    Solution: RBCs.
    Answer: B.
    Tip: RBCs = CO_2.