YOUR MENTOR GURU

CSIR NET Life Sciences & Biotechnology — Unit 4

Cell Communication and Cell Signaling

Study & revision companion | Topic explanations, core facts, and active-recall questions

How to use this guide

Read each topic, explain the key mechanism in your own words, then answer the self-check prompts without looking at the notes. Use your detailed textbook or class notes for full derivations and additional examples.

Topic overview

1. 1. Receptors and Second Messengers
2. 2. MAPK, PI3K and JAK–STAT Pathways
3. 3. Cell Adhesion and Communication
4. 4. Innate Immunity and Complement
5. 5. Adaptive Immunity and Antigen Presentation
6. 6. Hypersensitivity, Vaccines and Immunodeficiency
7. 7. Host–Pathogen Interactions and Cancer

1. Receptors and Second Messengers

GPCRs catalyze GDP–GTP exchange on heterotrimeric G proteins; Gαs stimulates adenylyl cyclase while Gαi often inhibits it.

cAMP activates PKA; IP_3 releases Ca^{2+} from ER stores and DAG helps activate PKC.

Receptor tyrosine kinases often dimerize and autophosphorylate, creating docking sites for signaling proteins.

Signal termination involves GTP hydrolysis, phosphatases, messenger breakdown and receptor internalization.

GPCRs, receptor kinases and intracellular receptors detect signals. cAMP, Ca^{2+}, IP_3 and DAG relay information.

When solving a question, distinguish the underlying mechanism from its observable outcome. Compare the normal situation with the altered condition, and check whether the evidence supports a direct effect or only an association. The central distinction to retain is: signal amplification allows a few activated receptors to affect many downstream proteins.

Self-check: Explain the mechanism or relationship above; identify one common misconception; predict the effect of changing a key variable.

2. MAPK, PI3K and JAK–STAT Pathways

The Ras–Raf–MEK–ERK cascade conveys many mitogenic signals to transcriptional targets.

PI3K produces PIP_3, recruiting PH-domain proteins including AKT; PTEN reverses this lipid signal.

Cytokine receptors frequently recruit JAK kinases, which phosphorylate STAT proteins that dimerize and regulate transcription.

Feedback loops and phosphatases shape signaling duration and amplitude.

MAPK and PI3K pathways regulate proliferation, survival and differentiation; feedback and phosphatases limit signaling.

When solving a question, distinguish the underlying mechanism from its observable outcome. Compare the normal situation with the altered condition, and check whether the evidence supports a direct effect or only an association. The central distinction to retain is: a downstream constitutively active protein can bypass an upstream block.

Self-check: Explain the mechanism or relationship above; identify one common misconception; predict the effect of changing a key variable.

3. Cell Adhesion and Communication

Cadherins mediate calcium-dependent cell–cell adhesion and link to the actin cytoskeleton through catenins.

Integrins connect extracellular matrix proteins to cytoskeletal and signaling networks.

Gap junction channels formed by connexins permit passage of small molecules between adjacent animal cells.

Tight junctions regulate paracellular permeability and help maintain epithelial polarity.

Cadherins mediate many cell–cell contacts; integrins connect cells to extracellular matrix; gap junctions transfer small molecules.

When solving a question, distinguish the underlying mechanism from its observable outcome. Compare the normal situation with the altered condition, and check whether the evidence supports a direct effect or only an association. The central distinction to retain is: integrins support bidirectional signaling.

Self-check: Explain the mechanism or relationship above; identify one common misconception; predict the effect of changing a key variable.

4. Innate Immunity and Complement

Pattern-recognition receptors detect microbial or damage-associated patterns and initiate innate responses.

Classical, lectin and alternative complement pathways converge on C3 cleavage.

C3b promotes opsonization; C3a and C5a promote inflammation, with C5a a potent chemoattractant.

C5b–C9 assemble the membrane attack complex; phagocytes use Fc and complement receptors to recognize targets.

Phagocytes, complement and pattern-recognition receptors respond rapidly to pathogens.

When solving a question, distinguish the underlying mechanism from its observable outcome. Compare the normal situation with the altered condition, and check whether the evidence supports a direct effect or only an association. The central distinction to retain is: c3b supports opsonization and C5a promotes inflammation.

Self-check: Explain the mechanism or relationship above; identify one common misconception; predict the effect of changing a key variable.

5. Adaptive Immunity and Antigen Presentation

B cells recognize native antigens through BCRs; conventional αβ T cells recognize peptide–MHC complexes.

MHC I generally presents endogenous peptides to CD8 T cells; MHC II generally presents endosomal peptides to CD4 T cells.

V(D)J recombination creates receptor diversity; activation-induced cytidine deaminase supports class switching and somatic hypermutation in B cells.

Primary responses are slower; memory cells support faster, often stronger secondary responses.

B cells make antibodies; T cells recognize peptides displayed by MHC molecules. Antibody diversity involves V(D)J recombination.

When solving a question, distinguish the underlying mechanism from its observable outcome. Compare the normal situation with the altered condition, and check whether the evidence supports a direct effect or only an association. The central distinction to retain is: mHC I generally presents to CD8 cells; MHC II to CD4 cells.

Self-check: Explain the mechanism or relationship above; identify one common misconception; predict the effect of changing a key variable.

6. Hypersensitivity, Vaccines and Immunodeficiency

Type I hypersensitivity involves IgE and mast-cell degranulation; type II involves antibody-mediated injury.

Type III involves immune-complex deposition; type IV is predominantly T-cell mediated.

Vaccines promote antigen-specific immune memory; adjuvants enhance innate activation and antigen presentation.

Immunodeficiency may be inherited or acquired and can affect humoral, cellular or innate compartments.

Immune memory underlies vaccination; hypersensitivity, autoimmunity and immunodeficiency involve different failures of immune control.

When solving a question, distinguish the underlying mechanism from its observable outcome. Compare the normal situation with the altered condition, and check whether the evidence supports a direct effect or only an association. The central distinction to retain is: type I hypersensitivity is commonly IgE-mediated.

Self-check: Explain the mechanism or relationship above; identify one common misconception; predict the effect of changing a key variable.

7. Host–Pathogen Interactions and Cancer

Pathogens exploit adhesion, invasion, immune evasion and intracellular survival mechanisms.

Oncogenes gain growth-promoting function; tumor suppressors lose restraints on proliferation or genome integrity.

RB regulates G1/S progression through E2F; p53 responds to stress by regulating arrest, repair and apoptosis.

Metastasis requires invasion, dissemination and colonization of distant tissues.

Microbes use attachment, entry and immune-evasion strategies; infection depends on host and pathogen factors.

When solving a question, distinguish the underlying mechanism from its observable outcome. Compare the normal situation with the altered condition, and check whether the evidence supports a direct effect or only an association. The central distinction to retain is: cell entry does not guarantee productive infection.

Self-check: Explain the mechanism or relationship above; identify one common misconception; predict the effect of changing a key variable.

Active-recall practice

1. Explain the essential principles of 1. Receptors and Second Messengers and give one experimental or real-world application.

2. Explain the essential principles of 2. MAPK, PI3K and JAK–STAT Pathways and give one experimental or real-world application.

3. Explain the essential principles of 3. Cell Adhesion and Communication and give one experimental or real-world application.

4. Explain the essential principles of 4. Innate Immunity and Complement and give one experimental or real-world application.

5. Explain the essential principles of 5. Adaptive Immunity and Antigen Presentation and give one experimental or real-world application.

6. Explain the essential principles of 6. Hypersensitivity, Vaccines and Immunodeficiency and give one experimental or real-world application.

7. Explain the essential principles of 7. Host–Pathogen Interactions and Cancer and give one experimental or real-world application.

Final revision checklist

- 1. Receptors and Second Messengers
- 2. MAPK, PI3K and JAK–STAT Pathways
- 3. Cell Adhesion and Communication
- 4. Innate Immunity and Complement
- 5. Adaptive Immunity and Antigen Presentation
- 6. Hypersensitivity, Vaccines and Immunodeficiency
- 7. Host–Pathogen Interactions and Cancer

Further study

Use the corresponding unit notes, topic-wise detailed lessons, MCQs, practice tests and full-length mock tests on yourmentorguru.com. This compact PDF is a revision companion, not a replacement for full syllabus textbooks.