

Immunology I

How the body tells self from non-self — the cells and organs of immunity, what makes a molecule immunogenic, antibody structure and kinetics, and the complement cascade that finishes the job.

Innate vs Acquired Antigens & Epitopes Antibody Structure

Primary/Secondary Response Ag-Ab Interactions Complement

Innate/Acquired

Cells & Organs

Antigens

Antibody Structure

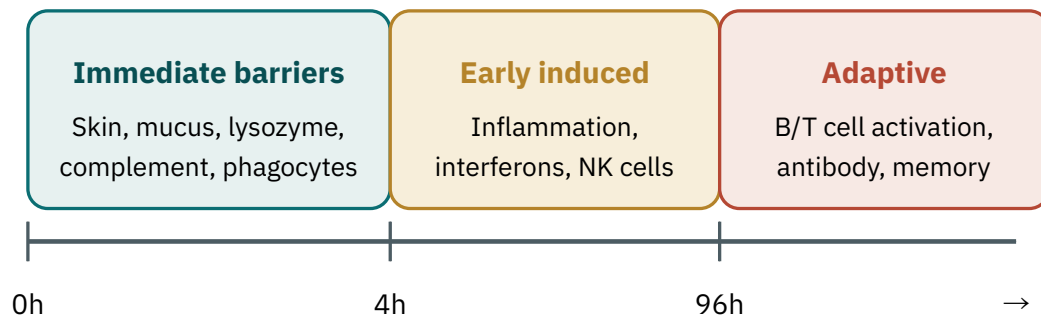
Response Kinetics

1 Innate & Acquired Immunity — Overview

Two systems, working on very different timescales

Feature	Innate immunity	Acquired (adaptive) immunity
Speed	Immediate (minutes–hours)	Delayed (days)
Specificity	Non-specific — recognises broad molecular patterns	Highly specific — recognises particular epitopes
Memory	None	Yes — faster, stronger on re-exposure
Key players	Skin/mucosa, neutrophils, macrophages, NK cells, complement	B & T lymphocytes, antibodies

Three phases of a host response

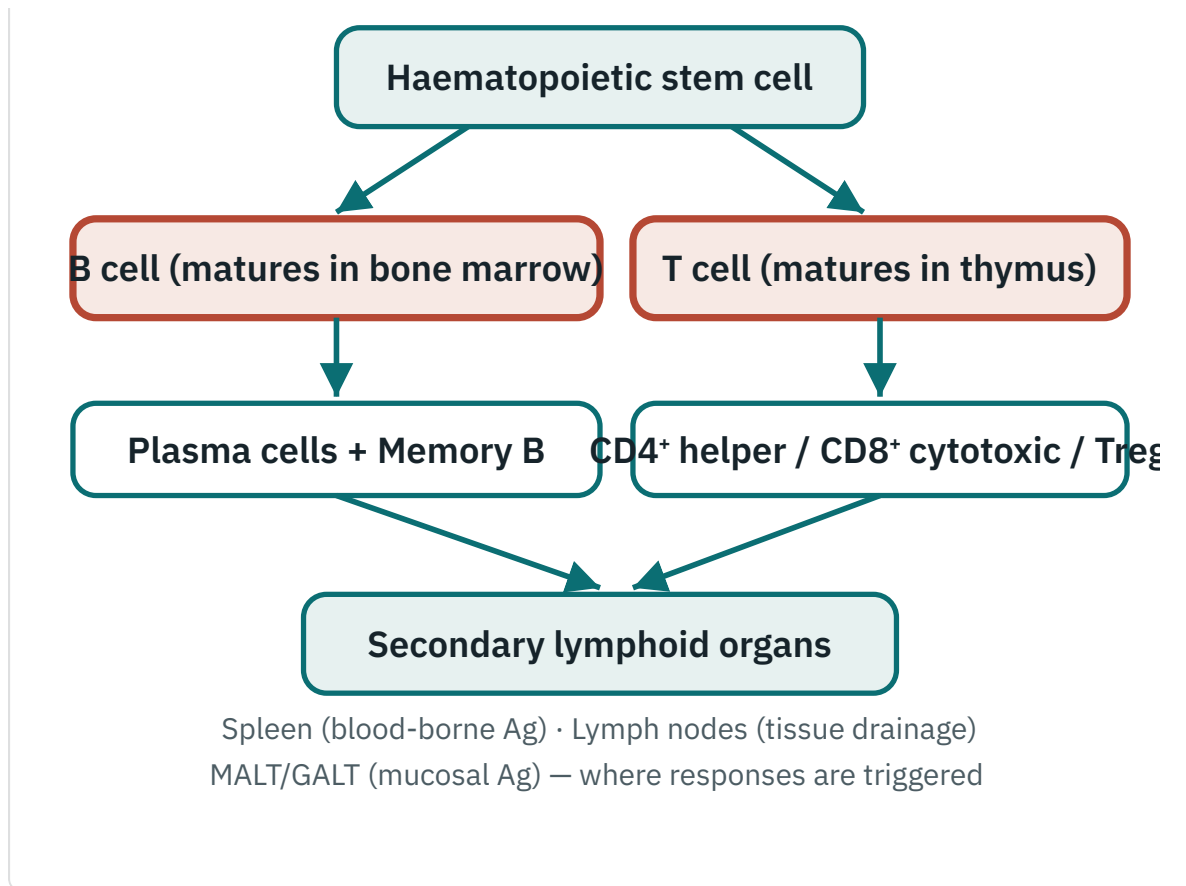


2 Elements of Acquired Immunity

Characteristics, cells, and where they mature and respond

- **Specificity** — each lymphocyte clone recognises one epitope
- **Diversity** — millions of possible receptor specificities, from gene rearrangement
- **Memory** — faster, stronger response on re-encounter
- **Self-limitation** — the response contracts once antigen is cleared
- **Self/non-self discrimination** — tolerance to the body's own antigens

Lymphocyte lineages & where they mature



ANTIGEN-PRESENTING CELLS (APCS)

Dendritic cells (most potent), macrophages, and B cells display processed antigen on MHC II to activate CD4⁺ T helper cells — the critical hand-off between innate detection and adaptive activation.

3 Immunogens & Antigens

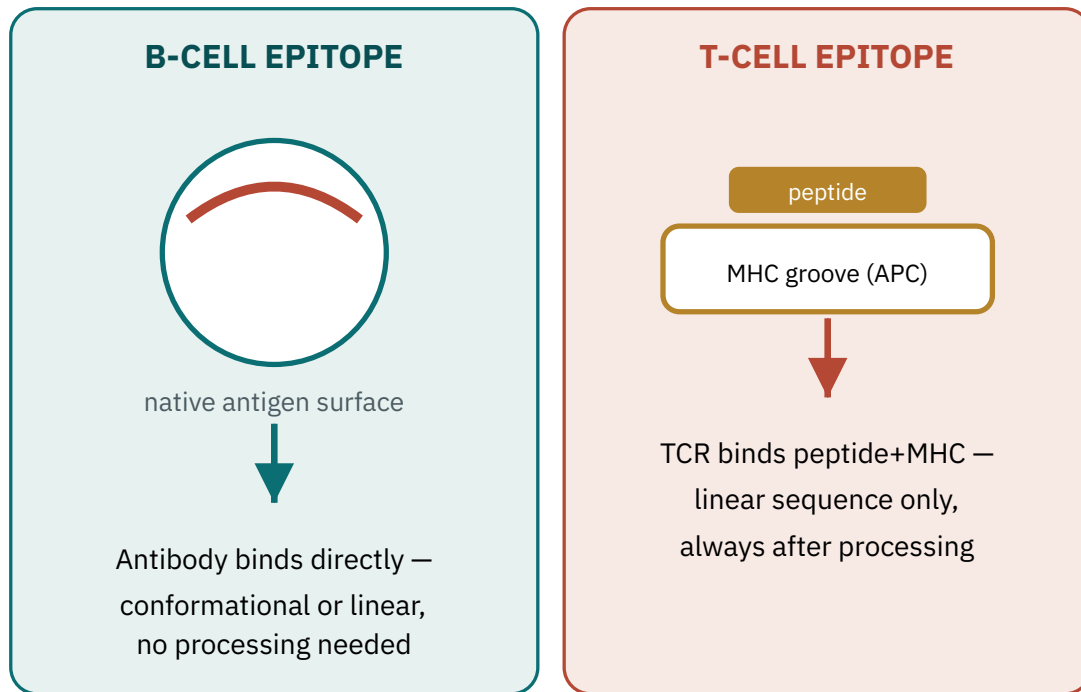
What makes a molecule worth mounting a response against

Every immunogen is antigenic (can bind antibody/TCR), but not every antigen is immunogenic (can trigger a response on its own) — that distinction is the whole point of haptens.

Requirement	Why it matters
Foreignness	Must be recognised as non-self
High molecular weight	Generally >10 kDa — bigger molecules are more immunogenic

Chemical complexity	Proteins > polysaccharides > lipids/nucleic acids alone
Degradability	Must be processable for T-dependent responses
Dose, route, host genetics	All modulate the strength of the response

B-cell vs. T-cell epitope recognition



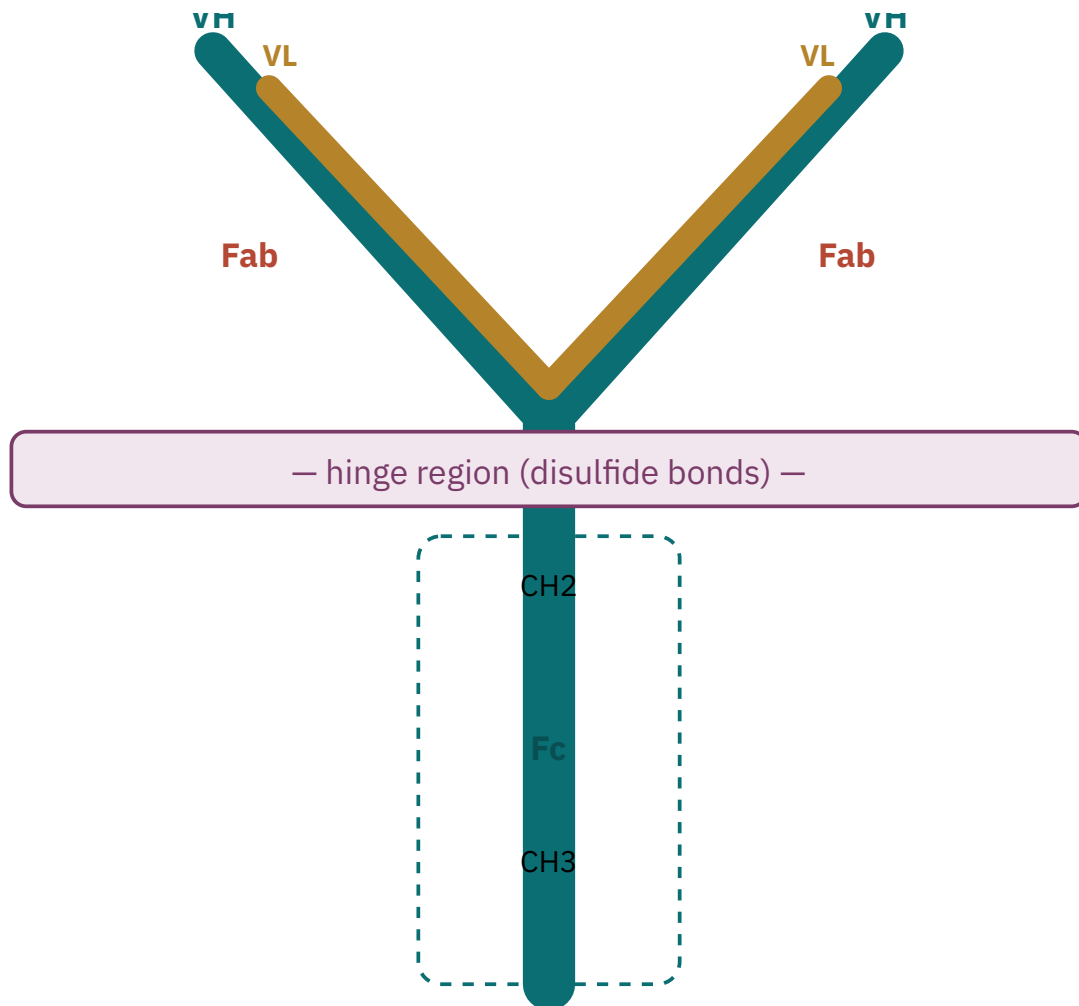
HAPTENS, ADJUVANTS & CROSS-REACTIVITY

A **hapten** (e.g. penicillin, urushiol) is too small to be immunogenic alone but becomes so once conjugated to a carrier protein — the basis of many drug allergies and contact dermatitis. An **adjuvant** (e.g. alum, Freund's) boosts the response to a co-administered antigen via depot effect and innate-immune activation. **Cross-reactivity** occurs when an antibody raised against one antigen also binds a structurally similar one — the mechanism behind molecular mimicry in diseases like rheumatic fever.

4 Antibody Classification, Structure & Functions

One basic Y-shaped unit, five isotypes, many jobs

The basic immunoglobulin unit



Fab = antigen binding (VH+CH1 / VL+CL) · Fc = effector functions

Isotype	Structure	Key features
IgG	Monomer	~75% of serum Ig; crosses placenta; dominant in secondary response; opsonises, activates complement
IgA	Dimer (secretory)	Mucosal defence — saliva, tears, breast milk; poor complement activator
IgM	Pentamer	First antibody made; most efficient complement activator (10 sites); too large for placenta
IgD	Monomer, membrane-bound	Co-expressed with IgM on naïve B cells as the B-cell receptor
IgE	Monomer	Lowest serum level; binds mast cell/basophil Fc receptors; immediate hypersensitivity, anti-helminth

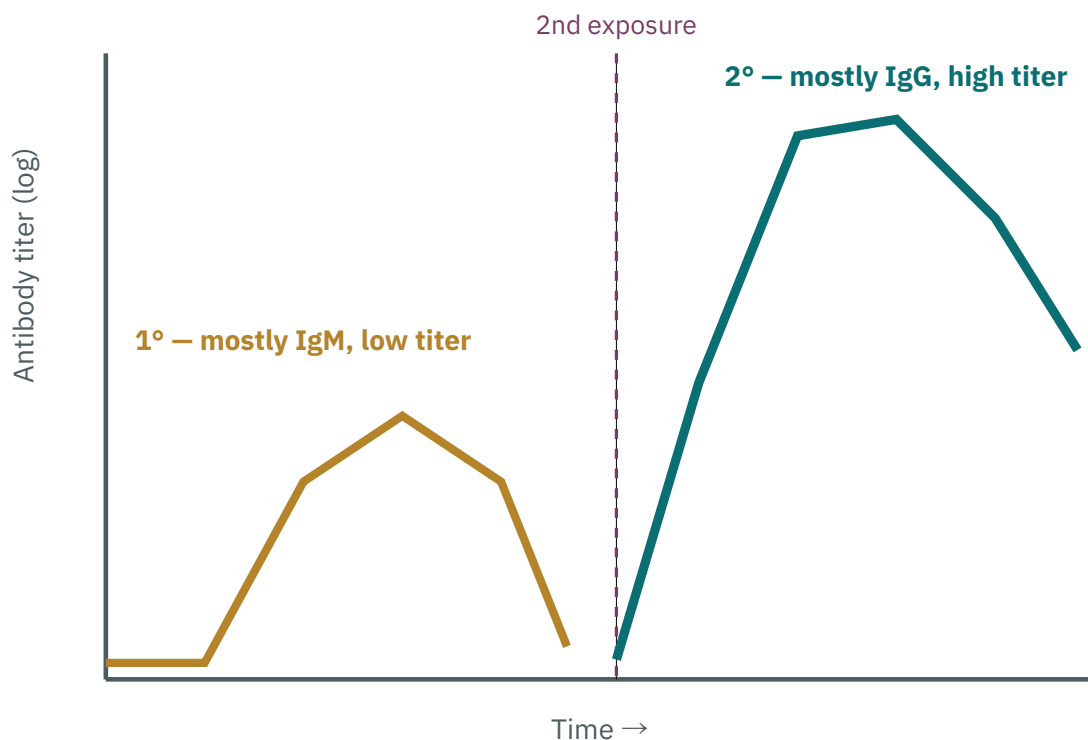
EFFECTOR FUNCTIONS, IN SHORT

Neutralisation (blocking toxins/viruses) · Opsonisation (coating for phagocytosis) · Complement activation · ADCC (antibody-dependent cell-mediated cytotoxicity) · Agglutination/precipitation of antigen.

5 Kinetics of Antibody Responses

Why booster doses work

Primary vs. secondary response



	Primary response	Secondary response
Lag phase	Long (several days)	Short
Dominant isotype	IgM first, then some IgG	Mostly IgG (memory cells pre-switched)
Magnitude & affinity	Lower	Higher — affinity maturation via somatic hypermutation

Duration	Shorter	Longer
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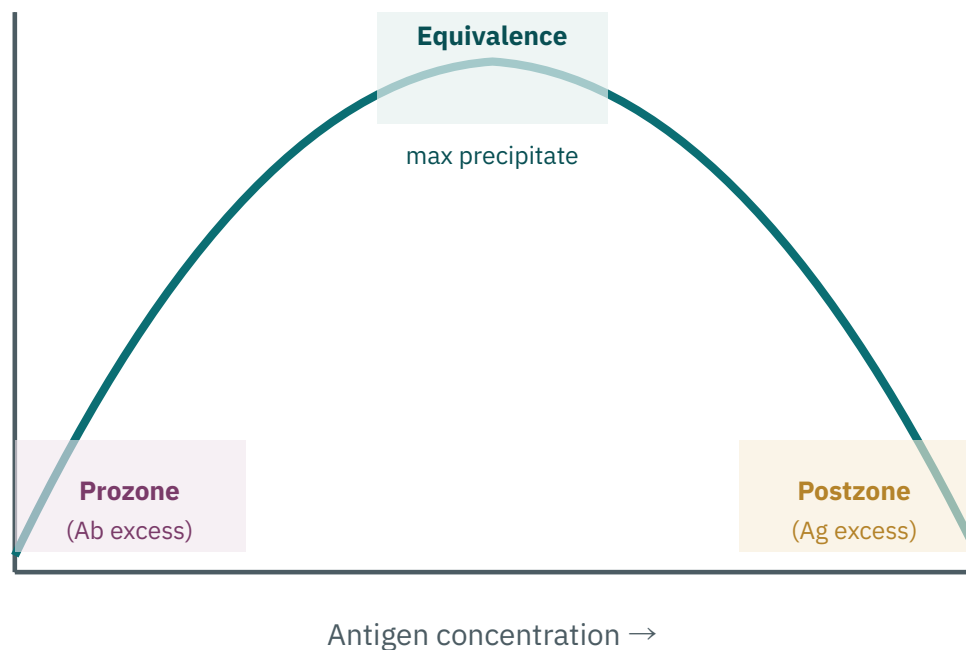
6 Antigen–Antibody Interactions

Non-covalent, reversible — but the proportions matter

Binding is entirely non-covalent — hydrogen bonds, electrostatic, van der Waals, and hydrophobic forces — which is exactly why it's reversible and why the ratio of antigen to antibody changes what you can observe.

- **Association constant (K_a) / Affinity** — strength of binding between one antigenic determinant and one antibody combining site; $K_a = [Ab-Ag] / [Ab][Ag]$
- **Avidity** — the overall binding strength of a multivalent antibody for a multivalent antigen; greater than the sum of individual affinities (IgM: modest affinity per site, but very high avidity from 10 sites)

Precipitin curve – the zone of equivalence



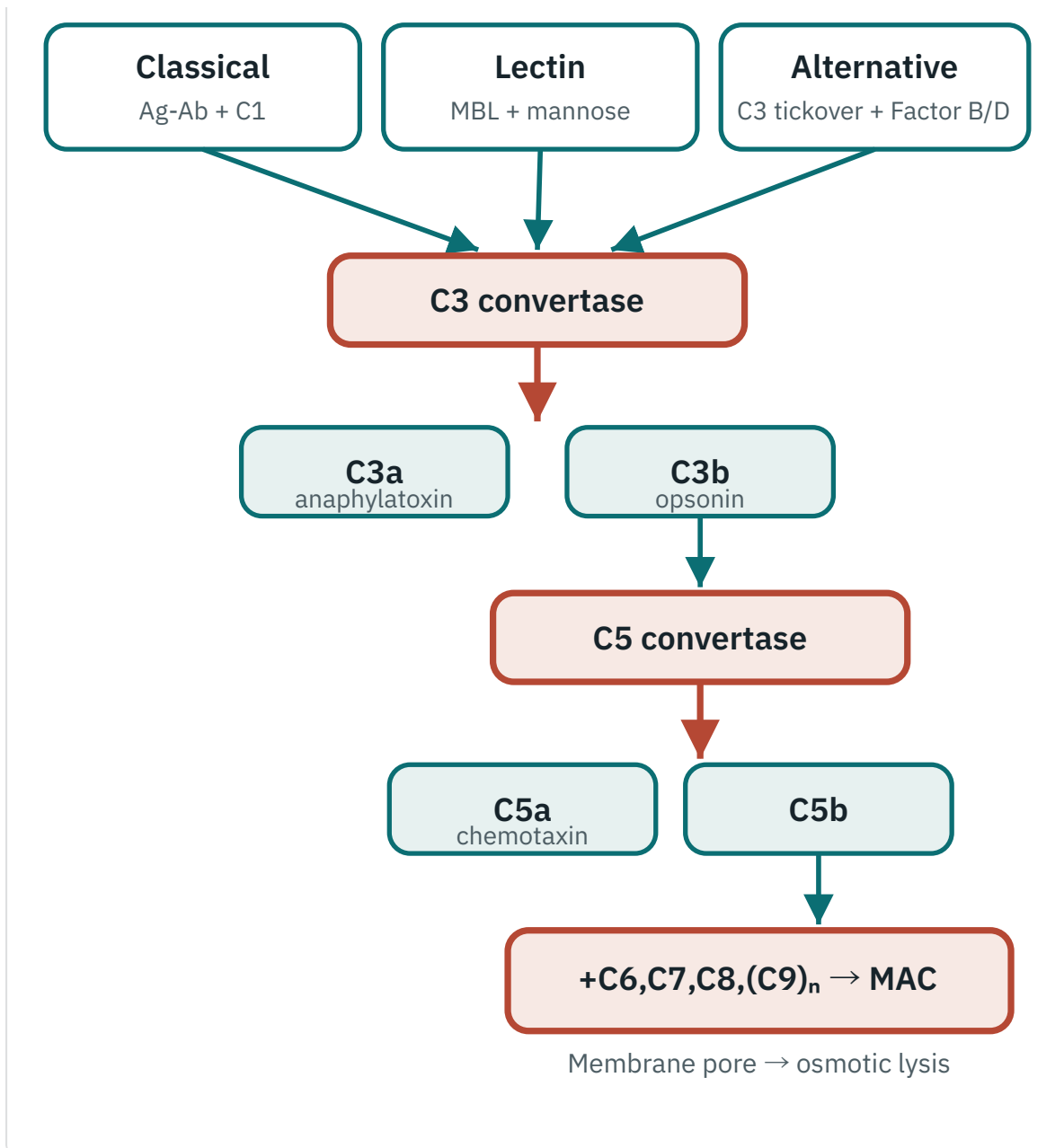
Reaction	Antigen form	Basis	Example use
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Precipitation	Soluble	Lattice formation at antibody:antigen equivalence	Ouchterlony double diffusion, radial immunodiffusion
Agglutination	Particulate (cells, beads)	Cross-linking of particles — more sensitive, needs less antibody	Blood typing, Widal test, latex agglutination

7 Complement System

~30 plasma proteins, three starting points, one shared ending

Three activation pathways converge on C3



Function	Mediator
Cell lysis	Membrane attack complex (C5b-9)
Opsonisation	C3b
Inflammation / chemotaxis	C3a, C5a (anaphylatoxins)
Immune complex clearance	C3b-coated complexes via RBC CR1

DEFICIENCY STATES

C1 inhibitor deficiency → hereditary angioedema. Terminal component (C5–C9)

deficiency → recurrent *Neisseria* infections. C3 deficiency → severe, recurrent pyogenic infections. Regulatory proteins (DAF, Factor H, Factor I, CD59) normally protect host cells from complement's own destructive potential.

8 Exam Corner

Practice question set — end-semester format

B.Sc. Biochemistry — End-Semester Examination

Paper: Immunology I

Full Marks: 30

Time: 1.5 Hrs

GROUP A — LONG ANSWER (ANSWER ANY TWO)

1. Describe the structure of an immunoglobulin molecule and discuss the classification, distribution, and functions of the five antibody isotypes. **10**
2. Compare the kinetics of the primary and secondary antibody responses with a suitable diagram, and explain the immunological basis of vaccination. **10**
3. Describe the classical, alternative, and lectin pathways of complement activation and discuss the biological functions of complement. **10**

GROUP B — SHORT NOTES (ANSWER ANY TWO)

- a. Requirements of immunogenicity; haptens and adjuvants **5**
- b. Difference between B-cell and T-cell epitopes **5**
- c. Affinity vs. avidity; precipitation vs. agglutination **5**