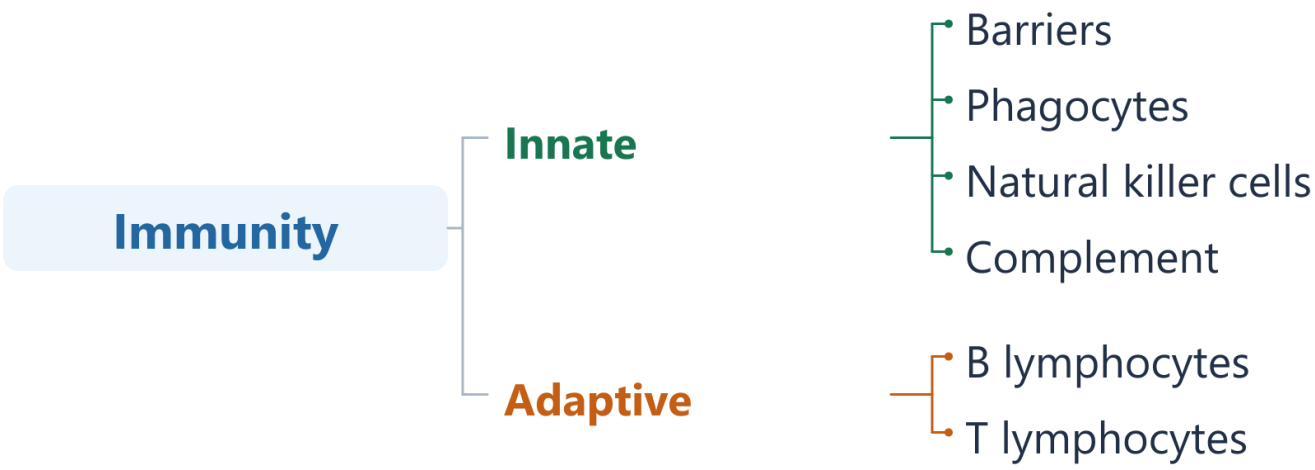
Pathology - Topic 8 - Immunopathology and AIDS

Principles of Immunity

- Immunity is the coordinated protection provided by cells, tissues and soluble mediators against infection and other harmful changes.

Innate and Adaptive Defence



1. Innate Immunity

- **Innate immunity** provides rapid defence through barriers, cells and soluble molecules that recognise broad patterns of danger.
  - Skin, mucosal barriers, phagocytes, natural killer cells and complement are important components.
- Pattern-recognition receptors detect microbial structures and molecules released by damaged cells.
  - Their activation produces inflammation and helps initiate an appropriate adaptive response.

2. Adaptive Immunity

- **Adaptive immunity** uses antigen-specific lymphocytes and can generate long-lasting memory.
  - B lymphocytes produce antibodies, while T lymphocytes coordinate responses or kill infected and abnormal cells.
- A primary response takes time to develop, while memory cells can respond more rapidly after re-exposure.
  - Adaptive immunity works together with innate mechanisms rather than replacing them.

Antigen Recognition and Effector Functions

B Cells and Antibodies

- B-cell receptors bind an antigen and help select the cells that should respond.

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- Activated B cells can become antibody-secreting plasma cells or memory B cells.

➢ Antibodies neutralise microbes or toxins, support opsonisation and activate appropriate complement pathways.

- Different immunoglobulin classes provide different functions and tissue distributions.

Immunoglobulin Classes

Class	Important functions and distribution
IgG	Major serum class in secondary responses; supports opsonisation and placental transfer.
IgM	Prominent early response; pentameric secreted form efficiently activates complement.
IgA	Protects mucosal surfaces; secretory forms occur in saliva, tears, intestinal secretions and breast milk.
IgE	Binds mast cells and basophils and participates in immediate hypersensitivity and anti-parasite responses.
IgD	Functions mainly as a receptor on naive B cells together with IgM.

T Cells and Antigen Presentation

Antigen Recognition and Co-stimulation

- T-cell receptors recognise antigenic peptides presented by HLA molecules.
- Appropriate co-stimulation and cytokine signals help determine whether a productive immune response develops.

Effector and Regulatory T Cells

- CD4 T cells coordinate inflammation and help other immune cells through cytokines and cell contact.
- Different helper-cell responses favour macrophage activation, eosinophilic responses or neutrophil recruitment in different settings.
- CD8 T cells kill appropriate target cells through granule-mediated mechanisms and death-receptor pathways.
- Regulatory T cells help restrain immune activation and maintain tolerance.

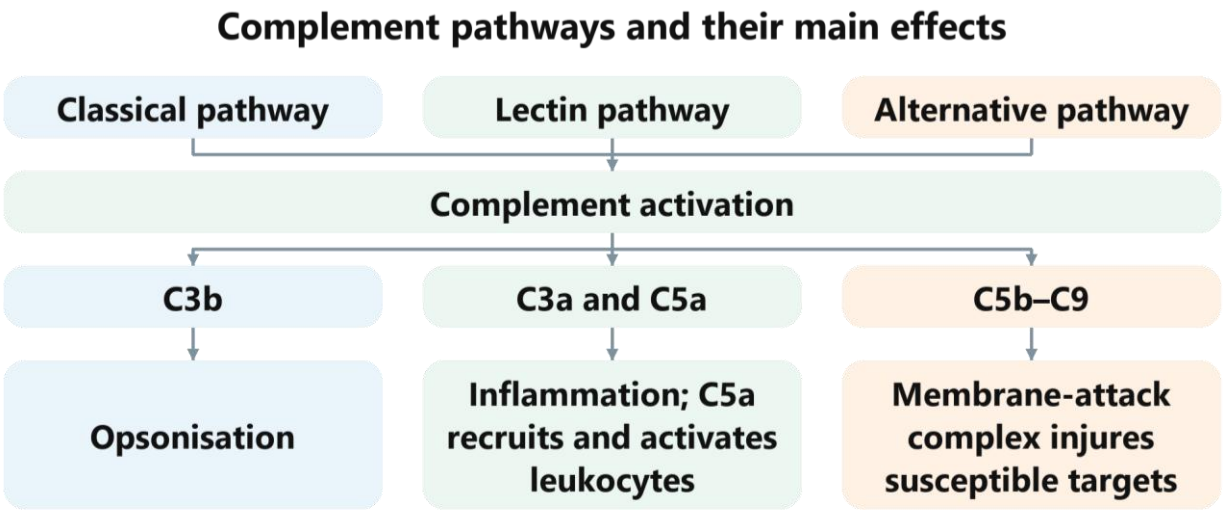
Helper T-Cell Functions

- Th1 responses promote macrophage activation through interferon-gamma and support defence against intracellular pathogens.

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- Th2 responses promote IgE-associated and eosinophilic reactions through cytokines including IL-4, IL-5 and IL-13.
- Th17 responses recruit neutrophils and support epithelial defence, while follicular helper T cells assist germinal-centre B-cell responses.

Complement



Complement pathways and their main effects

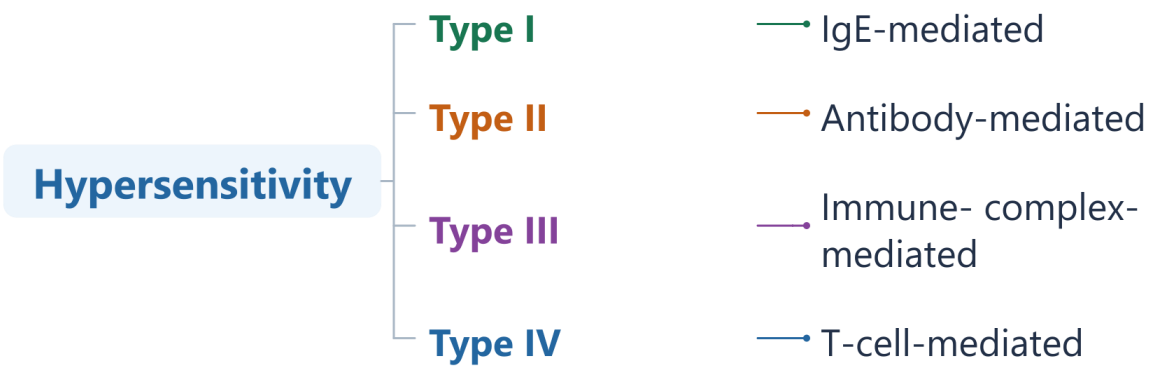
- The classical, alternative and lectin pathways activate complement through different initiating signals.
  - C3b promotes opsonisation, while C3a and C5a stimulate inflammatory reactions and C5a also recruits and activates leukocytes.
  - The C5b–C9 membrane-attack complex can injure susceptible target membranes.
- Complement deficiency can impair microbial defence or clearance of immune complexes, depending on the affected component.

Hypersensitivity Reactions

- **Hypersensitivity** is tissue injury caused by an excessive or inappropriate immune response.

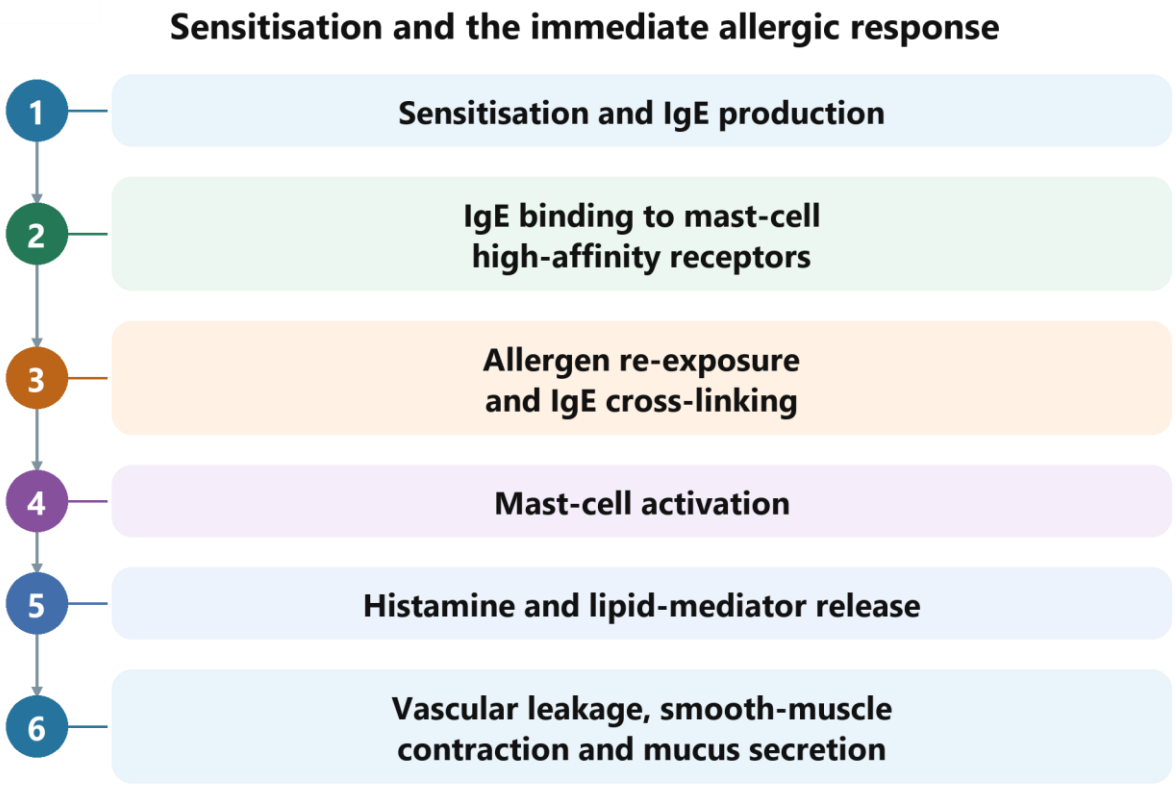
How Protective Responses Cause Injury

- The classical four-type classification groups reactions by their main mechanism, although some diseases involve more than one type.



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1. Type I: Immediate Hypersensitivity



- In type I hypersensitivity, an allergen stimulates an IgE-associated response in a sensitised person.
    - During sensitisation, helper-cell signals promote IgE production, and IgE binds high-affinity receptors on mast cells.
- Re-exposure allows the allergen to cross-link cell-bound IgE and activate the mast cell.
    - Histamine and newly formed lipid mediators cause vascular leakage, smooth-muscle contraction and mucus secretion.
    - Examples include allergic rhinitis, some forms of asthma and systemic anaphylaxis.
- Anaphylaxis can produce widespread vasodilation, oedema and airway compromise rather than a reaction confined to one site.

Late-Phase Tissue Injury

- The late phase depends on recruited eosinophils and other cells and their cytokines, and it can persist after the initial mast-cell products have declined.
- Recurrent eosinophil and epithelial injury can produce tissue remodelling, particularly in chronic allergic airway disease.

2. Type II: Antibody-Mediated Injury or Dysfunction

- Type II hypersensitivity involves antibodies directed against antigens on cells or extracellular structures.
    - The antibodies can promote complement activation, inflammation or phagocytic removal of the target.

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- Autoimmune haemolytic anaemia illustrates antibody-associated red-cell destruction.
    - Anti-basement-membrane disease illustrates antibody binding to a relatively fixed tissue antigen.
- An antibody may alter a receptor's function without destroying the cell.
    - In Graves disease, antibodies stimulate the TSH receptor, while in myasthenia gravis antibodies impair neuromuscular transmission through several receptor-associated mechanisms.

3. Type III: Immune-Complex Injury

- Type III hypersensitivity occurs when antigen–antibody complexes deposit in tissue and activate inflammatory mechanisms.
    - Complement activation and leukocyte recruitment can injure vessels, glomeruli, joints and other sites.
- Systemic lupus erythematosus and serum sickness are important examples.
    - A local Arthus reaction is a classic local immune-complex response.
- The site and severity depend on complex formation, clearance, vascular conditions and other host factors.

Tissue Morphology

- Small-vessel injury may show fibrinoid necrosis, neutrophils and fragmented nuclear debris.
- Glomerular immune-complex deposits commonly produce a granular immunofluorescence pattern, in contrast with the linear pattern of classic anti-GBM disease.
- Complement consumption can lower circulating complement levels during an active systemic immune-complex process.

4. Type IV: T-Cell-Mediated Injury

- Type IV hypersensitivity is mediated by T cells rather than by circulating antibodies.
    - CD4-cell cytokines recruit and activate inflammatory cells, while CD8 cells can directly kill target cells.
- Allergic contact dermatitis and the tuberculin skin response illustrate delayed T-cell-mediated reactions.
    - Persistent T-cell stimulation can contribute to granuloma formation.
- The onset is usually delayed compared with an immediate mast-cell reaction because cellular recruitment and activation take time.

Delayed Response and Direct Cytotoxicity

- A tuberculin reaction typically reaches its greatest local response after approximately 48–72 hours and contains activated T cells and macrophages.

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