



PHARMACOLEARN

It's Important to Know!

BP204T — Pathophysiology

B.Pharm Semester 2 · Unit 1 — Cell Injury, Inflammation and Repair

Unit I: Cell Injury, Inflammation and Repair

Homeostasis and feedback

Homeostasis is maintenance of a relatively stable internal environment. Negative feedback opposes a change, while positive feedback reinforces a change until a defined event is completed. Disease can result when regulation fails, a stress exceeds adaptive capacity or an abnormal response becomes harmful.

Causes and mechanisms of cell injury

Cell injury can result from oxygen deprivation, physical agents, chemicals, infections, immune reactions, genetic defects or nutritional imbalance. Early injury may be reversible when the cause is removed. Persistent or severe injury can cause irreversible damage and cell death.

Important mechanisms include ATP depletion, mitochondrial dysfunction, loss of membrane integrity, calcium influx, oxidative stress, protein misfolding and DNA damage. Ribosome injury can reduce protein synthesis. Nuclear damage can lead to chromatin change and loss of genetic control.

Adaptation and cell death

Hypertrophy is increased cell size. Hyperplasia is increased cell number. Atrophy is reduced cell size or tissue mass. Metaplasia is replacement of one mature cell type by another better able to tolerate stress. Dysplasia is disordered growth and may be associated with a premalignant change.

Apoptosis is regulated cell death with cell shrinkage and fragmentation into membrane-bound bodies. Necrosis involves cell swelling, membrane rupture and inflammation. The morphology and biochemical pathways differ, though some diseases can involve both processes.

Inflammation and repair

Acute inflammation begins with changes in blood flow and vascular permeability, followed by leukocyte recruitment. Selectins support rolling, integrins support firm adhesion and chemotactic signals guide migration. Mediators include histamine, prostaglandins, leukotrienes, cytokines and

complement fragments.

Chronic inflammation involves persistent injury, macrophages, lymphocytes, tissue destruction and repair occurring together. Repair uses regeneration when viable cells and supporting tissue remain, or fibrosis when damage is extensive. Wound healing proceeds through haemostasis, inflammation, proliferation and remodelling.

Unit I: Frequently asked questions

Q: What is reversible cell injury?

A: It is cell dysfunction that can return toward normal when the damaging stimulus is removed before irreversible membrane, mitochondrial or nuclear damage occurs.

Q: How does apoptosis differ from necrosis?

A: Apoptosis is regulated cell death with limited inflammation, while necrosis commonly involves membrane rupture and inflammation.

Q: What are the main stages of wound healing?

A: They are haemostasis, inflammation, proliferation and remodelling.

Unit I: Key takeaways

- Homeostasis depends on coordinated control systems.
- ATP loss, calcium imbalance and oxidative stress are important injury mechanisms.
- Cells adapt through hypertrophy, hyperplasia, atrophy and metaplasia.
- Apoptosis is regulated, while necrosis commonly causes inflammation.
- Inflammation and repair can restore tissue or produce fibrosis.

Unit I: Clinical correlation

Clinical correlation: inflammation. Redness, heat, swelling, pain and loss of function are classic clinical signs of inflammation.