

Biomedical
Science

Molecular
Genetics



BMS 301 | Fall 2026-2027

Systemic & Cellular Pathology

**Cell injury, Cell Death , and Adaptation
Mechanisms**



Free Session

A+ is more than just a platform





Introduction to Pathology

STUDY NOTES

DEFINITION

Pathology “is the study of the **structural, biochemical, and functional changes** in cells, tissues, and organs that underlies disease”.

Study of pathology



General pathology



Systemic pathology

The four main aspects

1. Etiology

Causation.

2. Pathogenesis

Biochemical and molecular mechanism.

3. Morphologic changes

The associated structural and functional alterations in cells and organs.

4. Clinical manifestation

Resulting clinical consequences.



1. Etiology

STUDY NOTES

DEFINITION

Etiology is the **initiating cause of a disease.**

The causes of the disease can be grouped into

Genetic

Inherited or acquired **mutations** and **polymorphisms**.

Environmental

- Physical
- Chemical
- Infectious

KEY POINT

The majority of diseases are not caused by one factor (atherosclerosis, cancer, etc.) — they are **multifactorial.**

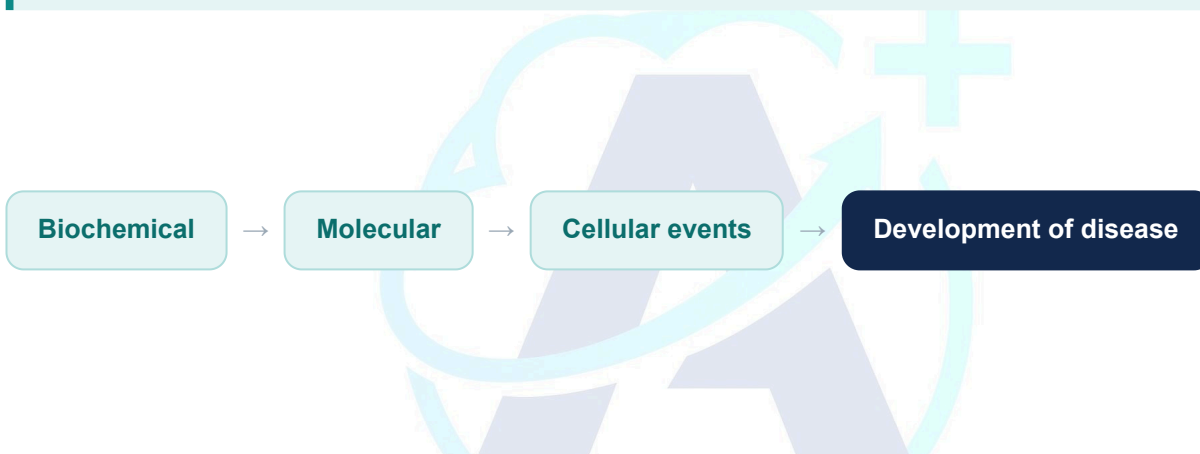


2. Pathogenesis

STUDY NOTES

DEFINITION

The **sequence of biochemical, molecular,** in addition to cellular events that results in the development of the disease.



Omics

Genomics, proteomics and metabolomics explain the **pathogenic mechanism**.

Genomics

Proteomics

Metabolomics

Biomarkers

Disease progression

Therapeutic response



3. Morphologic Changes

STUDY NOTES

Cellular and tissue alteration that are feature of the disease.

CURRENTLY SUPPLEMENTED BY

Genetic alteration analyses

Protein expression analyses



4. Clinical Manifestations

STUDY NOTES

The outcome of the **genetic, biochemical and structural changes**.

Abnormal function



Clinical manifestation



Signs and symptoms

Signs

- Fever (measured)
- Low oxygen levels
- Cough observed by examiner

Symptoms

- "I feel short of breath."
- "I have body aches."
- "I feel tired."



Cellular Responses to Stress

STUDY NOTES

Normal cells have **specific function and structure.**

ADAPTATIONS

Adaptations are **reversible structural and functional modifications** in response to altered physiologic states (such as pregnancy) or certain pathologic stimuli, through which a **new but modified steady state** is established.



WHY IT MATTERS

These changes enable the cell to **survive and maintain function.**



Adaptational Mechanisms

STUDY NOTES

SIZE INCREASES

Hypertrophy

Increase in **size of the cell**.

NUMBER INCREASES

Hyperplasia

Increase in **functional activity and number of cells**.

SIZE DECREASES

Atrophy

Decrease in **size and metabolic activity**.

TYPE CHANGES

Metaplasia

Change in **type of cell**.

IF THE STRESS IS ELIMINATED

The cell have the capability to **retain to its normal state** without any consequences.



Cell Injury

STUDY NOTES

DEFINITION

Cell injury is the state when the **adaptive response is exceeded**, cells were exposed to damaging injurious agents, massive loss of nutrients, or the presence of mutations that alter the main functions.

Reversible

Cell injury **can be reversible**.

Irreversible

If the injurious agent **persist** or it was **sever** — leading to **death of the cells**.

PROGRESSIVE IMPAIRMENT

Adaptation**Reversible injury****Cell death**



Cell Death

STUDY NOTES

There are **two pathways** for cell death.

Necrosis

Is the **death of body tissue**. It occurs when too little blood flows to the tissue.

Apoptosis

Programmed cell death.

Cell death



Necrosis



Apoptosis



Causes of Cell Injury

STUDY NOTES

- 1 Physical agents
- 2 Genetic abnormalities
- 3 Oxygen deprivation — hypoxia
- 4 Chemical agents and drugs
- 5 Infectious agents
- 6 Immunologic reaction
- 7 Nutritional imbalance



1. Physical Agents

STUDY NOTES

Mechanical trauma — football tackle, car crash.



Mechanical trauma — **football tackle**



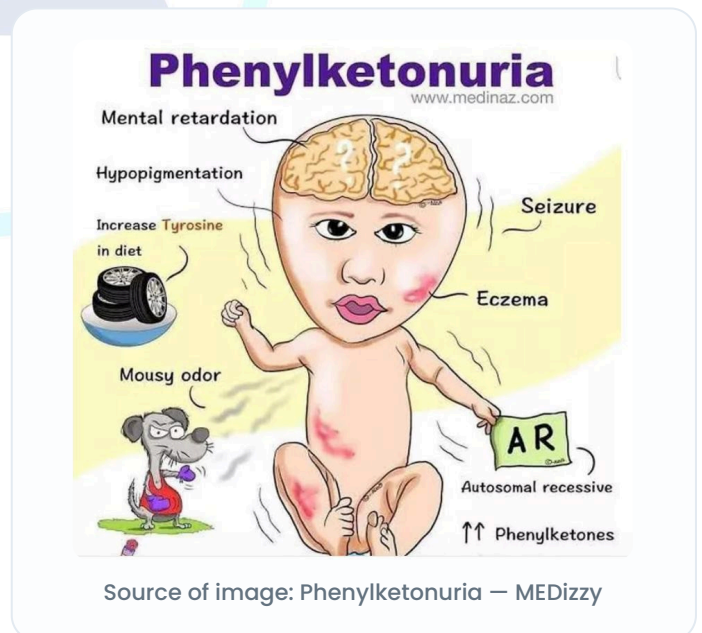
2. Genetic Abnormalities

STUDY NOTES

Mutations: impact **vital enzyme for metabolic function.**

Example — Phenylketonuria (PKU)

- 1 Mutation in the **PAH gene** (Phenylalanine hydroxylase enzyme).
- 2 **Effect:** this enzyme normally breaks down the amino acid phenylalanine.
- 3 **Mutation:** causes loss or reduction of enzyme activity.
- 4 **Consequence:** phenylalanine builds up in the blood → **toxic to the brain.**
- 5 **Condition:** Phenylketonuria (PKU) → leads to intellectual disability, seizures, and developmental problems if untreated.



PAH mutation



Loss of enzyme activity



Phenylalanine build-up



PKU



3. Oxygen Deprivation — Hypoxia

STUDY NOTES

DEFINITION

Hypoxia is a condition where cells and tissues are **deprived of adequate oxygen supply**, which is essential for ATP production (aerobic oxidative respiration).

What cause hypoxia?

Reduced blood flow

Ischemia.

Inadequate oxygenation of blood

Sever blood loss, **anemia**.

HYPOXIA MAY LEAD TO

Adaptation

/

Reversible injury

/

Death of cell



4. Chemical Agents & Drugs

STUDY NOTES

EVEN THE ORDINARY

Glucose, salt, oxygen at high level is toxic.

POISONS

Arsenic, cyanide, mercury may damage cell in short period of time (minutes to hours).

ENVIRONMENTAL & OCCUPATIONAL

Insecticides**Herbicides****Carbon monoxide****Asbestos**

Occupational hazards.



Where in daily life might people be exposed to **carbon monoxide** and **asbestos**, and why are these exposures harmful?



5. Infectious Agents

STUDY NOTES

Starting from **viruses** to **tapeworms**.

Viruses

Bacteria

Fungi

Parasites

Tapeworms

How it cause cell injury is **diverse**.



6. Immunologic Reaction

STUDY NOTES

Against self

Autoimmune disease — immune reaction against self antigen.

Against external agents

Immune reaction against external agents such as **viruses** and **environmental substances** may also cause cell and tissue injury.



7. Nutritional Imbalance

STUDY NOTES

Protein calorie deficiencies are the main cause of cell injury; common in lower income population.

SHORTAGE

Nutritional shortage

Anorexia nervosa — the body adapt to starvation.

EXCESS

Nutritional excess

Obesity → diabetes and cancer.

BALANCED

Balanced diet.

IMBALANCE

High in lipid → **atherosclerosis**.



Class Activity

STUDY NOTES

CELLULAR RESPONSE DIAGRAM

You have **8 minutes** to draw a diagram showing how cells respond to stress and injury. Include these elements:

Normal healthy cell

Left side of the diagram.

Stressful agents

Hypoxia, toxins, trauma, infection.

Cell tries to adapt

- Atrophy (shrinks)
- Hypertrophy (enlarges)
- Hyperplasia (multiplies)
- Metaplasia (changes type)

If adaptation fails → cell death

- Necrosis (uncontrolled)
- Apoptosis (programmed)

USE ARROWS TO SHOW PROGRESSION

Normal



Stress



Adaptation



Death



To Summarise...

STUDY NOTES

PATHOLOGY

4

Main aspects

- Etiology — causation
- Pathogenesis — mechanism
- Morphologic changes and clinical manifestation

ETIOLOGY

2

Groups of causes

- Genetic — mutations, polymorphisms
- Environmental — physical, chemical, infectious
- Most diseases are multifactorial

ADAPTATION

4

Adaptational mechanisms

- Hypertrophy — cell size increases
- Hyperplasia — activity and number increase
- Atrophy and metaplasia

CELL INJURY

2

Reversible or irreversible

- Adaptive response is exceeded
- Reversible injury is recoverable
- Irreversible if the agent persist or was sever

CELL DEATH

2

Pathways

- Necrosis — death of body tissue
- Occurs when too little blood flows to the tissue
- Apoptosis — programmed cell death

CAUSES

7

Causes of cell injury

- Physical agents, genetic abnormalities, hypoxia
- Chemical agents and drugs, infectious agents
- Immunologic reaction, nutritional imbalance

THE WHOLE PICTURE

Normal cell



Adaptation



Reversible injury



Cell death



References

STUDY NOTES

TEXTBOOK

Kumar, V., Abbas, A. K., C Aster, J. C. (2021). *Robbins and Cotran pathologic basis of disease* (10th ed.). Elsevier.

IMAGE CREDIT

Source of image: Phenylketonuria — MEDizzy.

LECTURE

Systematic and Cellular Pathology — HMG 399. Week 1, Second Class, 3rd of September 2026. Dr. Alaa Siddig.