

# Cellular Adaptations and Accumulations

- Adaptations: Reversible changes in cells responding to environmental changes
- Can be physiologic (normal stimulation) or pathologic (stress response)

## Types of Adaptations



### Hypertrophy

- Occurs in cells with limited division capacity
- Can be physiologic (e.g., uterus during pregnancy/ hypertrophy. Adult muscle cells) or pathologic (e.g., cardiac hypertrophy)

### Hyperplasia

- Increase in cell number
- Occurs in cells capable of replication
- Types: Hormonal (e.g., breast tissue growth) and Compensatory (e.g., liver regeneration)

### Atrophy

### Metaplasia

### Pathologic hyperplasia

- Caused by excessive hormonal or growth factor stimulation. Normally, after a normal menstrual period. disturbance in this balance increased estrogenic stimulation → endometrial hyperplasia, (a common cause of abnormal menstrual bleeding). → Benign prostatic hyperplasia is by androgens → Certain viral infections

The hyperplastic process remains controlled

## Cardiac Hypertrophy Mechanisms

- Two types of signals drive cardiac hypertrophy [1]:
  1. **Mechanical triggers** (e.g., stretch)
  2. **Soluble mediators** (growth factors & adrenergic hormones)
- **Signal Transduction Process [1]:**
  - Stimuli → Signal transduction pathways → Gene induction → Protein synthesis
- **Cellular Changes [2]:**
  - Increased protein and myofilament synthesis
  - Enhanced force generation per contraction
  - Adaptation to increased work demands
- **Protein Modifications [2]:**
  - Switch from adult to fetal/neonatal contractile proteins
  - Replacement of  $\alpha$ -myosin heavy chain with  $\beta$ -myosin heavy chain
  - Results in slower, more energy-efficient contractions

An adaptation to stress such as hypertrophy can progress to functionally significant cell injury if the stress is not relieved

THE HIGH  
YIELD



## Types of Adaptations

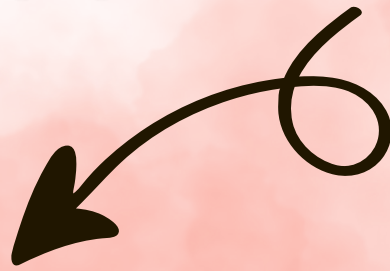


Hypertrophy

Hyperplasia

Atrophy

Metaplasia



### Shrinkage of cell size

- Causes: decreased workload, loss of innervation, diminished blood supply, etc.
- Results from decreased protein synthesis and increased degradation

→ In many situations, atrophy also is associated with autophagy

### Replacement of one adult cell type with another

- Occurs as a protective mechanism against stress
- Examples: Respiratory epithelium changes in smokers, esophageal changes in chronic reflux

→ The influences that induce metaplastic change in an epithelium, if persistent, may predispose to malignant transformation.

# Intracellular Accumulations

- Cells may accumulate abnormal amounts of substances, which can be harmless or cause injury
- Main pathways of abnormal intracellular accumulations:
  - Inadequate removal and degradation
  - Excessive production of endogenous substances
  - Deposition of abnormal exogenous materials

## Types of Accumulations

### Fatty Change (Steatosis)

- Accumulation of triglycerides in parenchymal cells
- Common in liver, also occurs in heart, skeletal muscle, and kidney
- Caused by toxins, protein malnutrition, diabetes, obesity, or anoxia

### Glycogen

- Excessive accumulation associated with abnormal glucose or glycogen metabolism
- Occurs in poorly controlled diabetes and glycogen storage diseases

### Lipofuscin

- Insoluble brownish-yellow granular material
- Accumulates with aging or atrophy in various tissues
- Marker of past free radical injury

### Cholesterol and Cholesteryl Esters

- Tightly regulated in cellular metabolism
- Can accumulate in phagocytic cells due to increased intake or decreased catabolism
- Important in atherosclerosis

### Carbon

- Exogenous pigment common in urban environments
- Inhaled and phagocytosed by alveolar macrophages
- Causes anthracosis in lungs and lymph nodes

### Melanin

- Endogenous brown-black pigment synthesized by melanocytes
- Protects against UV radiation

### Hemosiderin

- Hemoglobin-derived golden yellow to brown pigment
- Accumulates with excess iron
- the iron can be unambiguously identified by the Prussian blue histochemical reaction
- Can lead to hemosiderosis or hemochromatosis