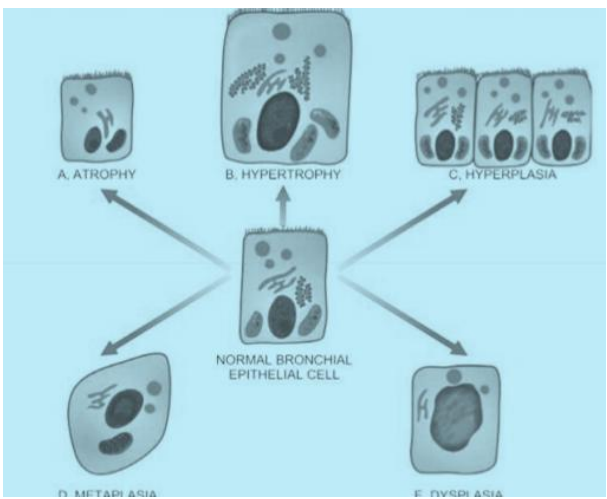
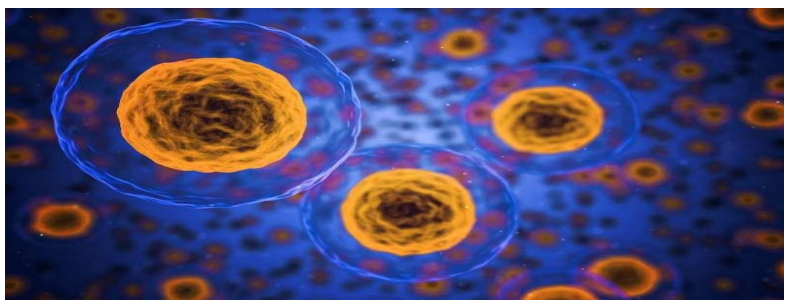
Cellular Adaptations & Subcellular Alterations Part-I





Dr Sajda Khan Gajdhar

Lecture Learning Outcomes:
By the end of this lesson students will be able to:

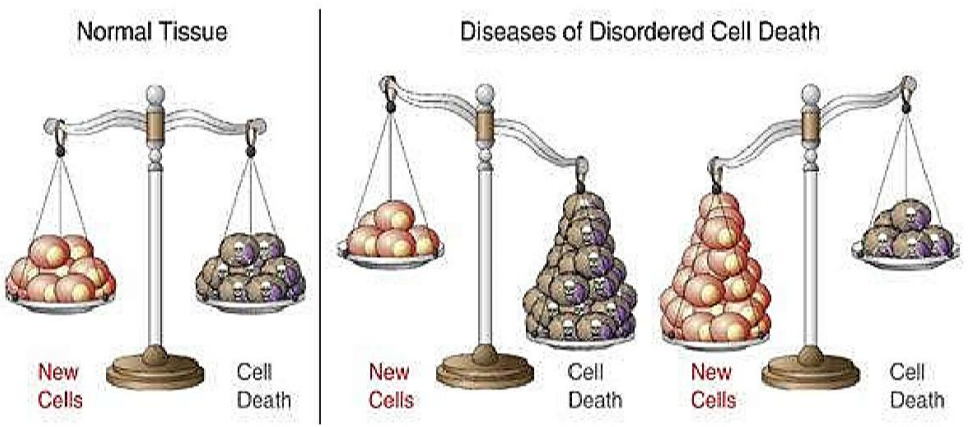
1. Define cellular adaptation and classify its type.
2. Classify and Discuss the types of cellular adaptation
3. Explain hyperplasia, hypertrophy, atrophy and metaplasia with examples.

Homeostasis

Greek – Homeo: same Stasis: stable

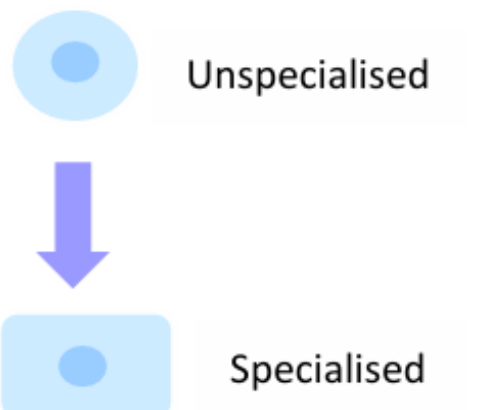
- The "steady state" that cell exists in normally.
- An equilibrium of the cells with their environment for adequate function.
- **When disturbed there is a predisposal for the onset of pathology.**

The cells constantly adjust their structure and function to accommodate changing demands and extracellular stresses



Cellular Differentiation

- The process by which a cell becomes specialised in order to perform a specific function, as in the case of a liver cell, a blood cell, or a neuron (the process of specialisation)
- The characteristics that determine cell type



Cellular Proliferation

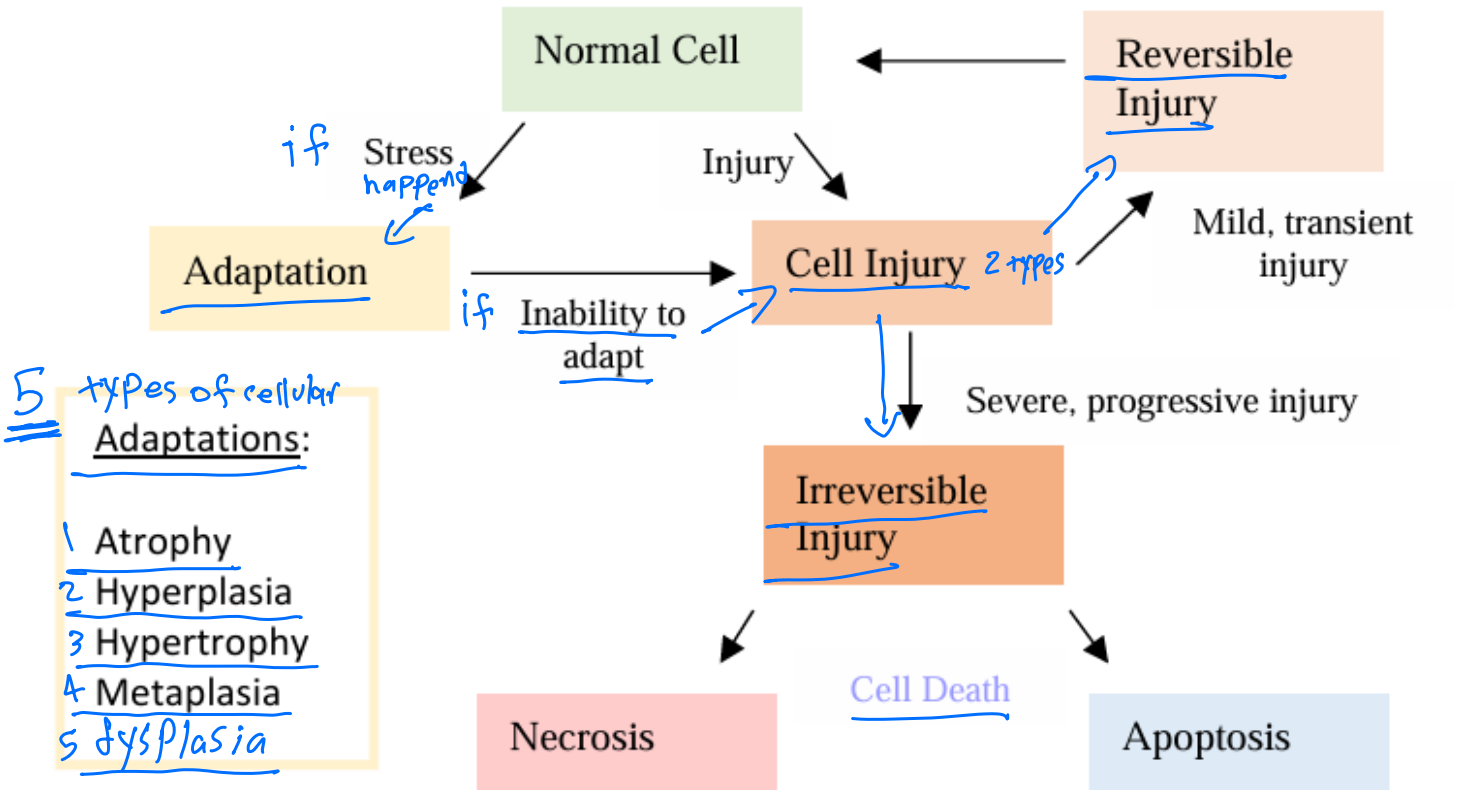
1. **Permanent cells won't divide** – they stay in one part of the cell cycle(Cardiac and skeletal myocytes CNS neurons)
2. **Stable cells** – will move into the cycle when stimulated (Epithelial e.g. liver, kidney, lung, pancreas Smooth muscle cells, fibroblasts, endothelial cells)
3. **Labile cells** – constantly moving through cell cycle. (Epithelial (e.g. skin, GIT, urinary tracts, lining of exocrine ducts)

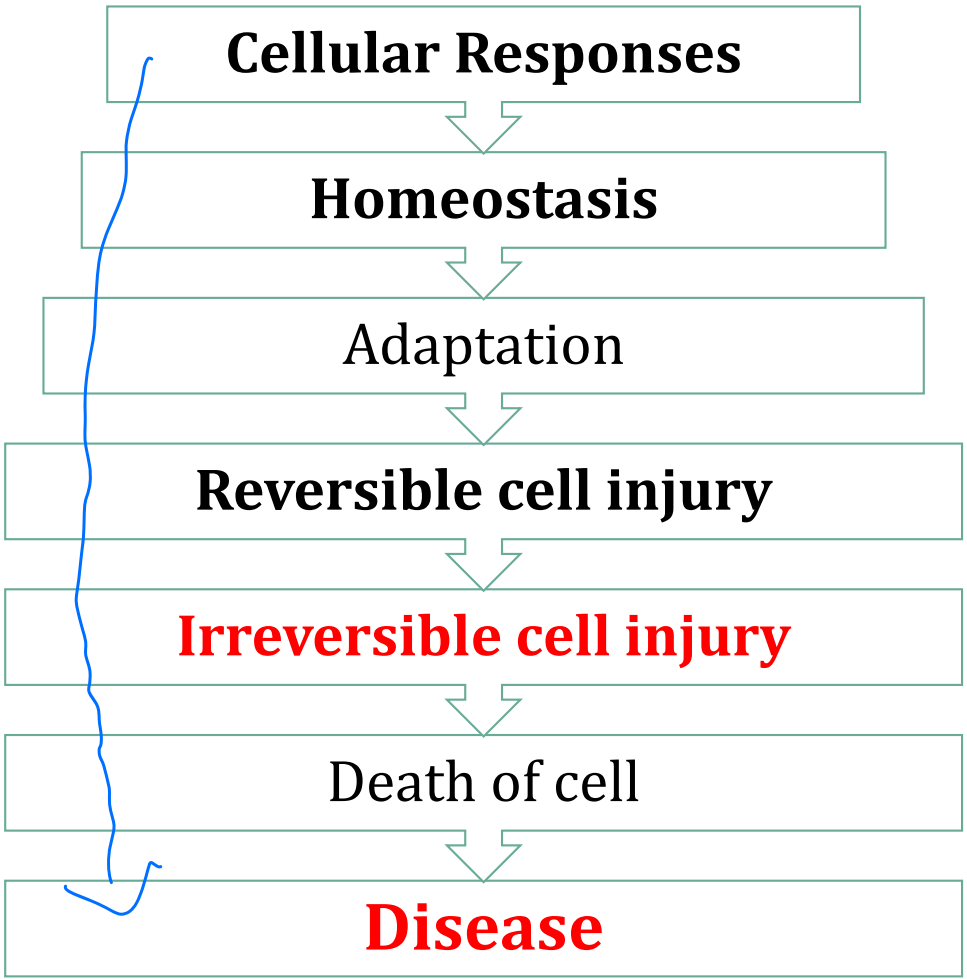
WHAT IS ADAPTATION ?

adjusment

- Adaptations are reversible changes in the size, number, phenotype, metabolic activity, or functions of cells in response to changes in their environment.

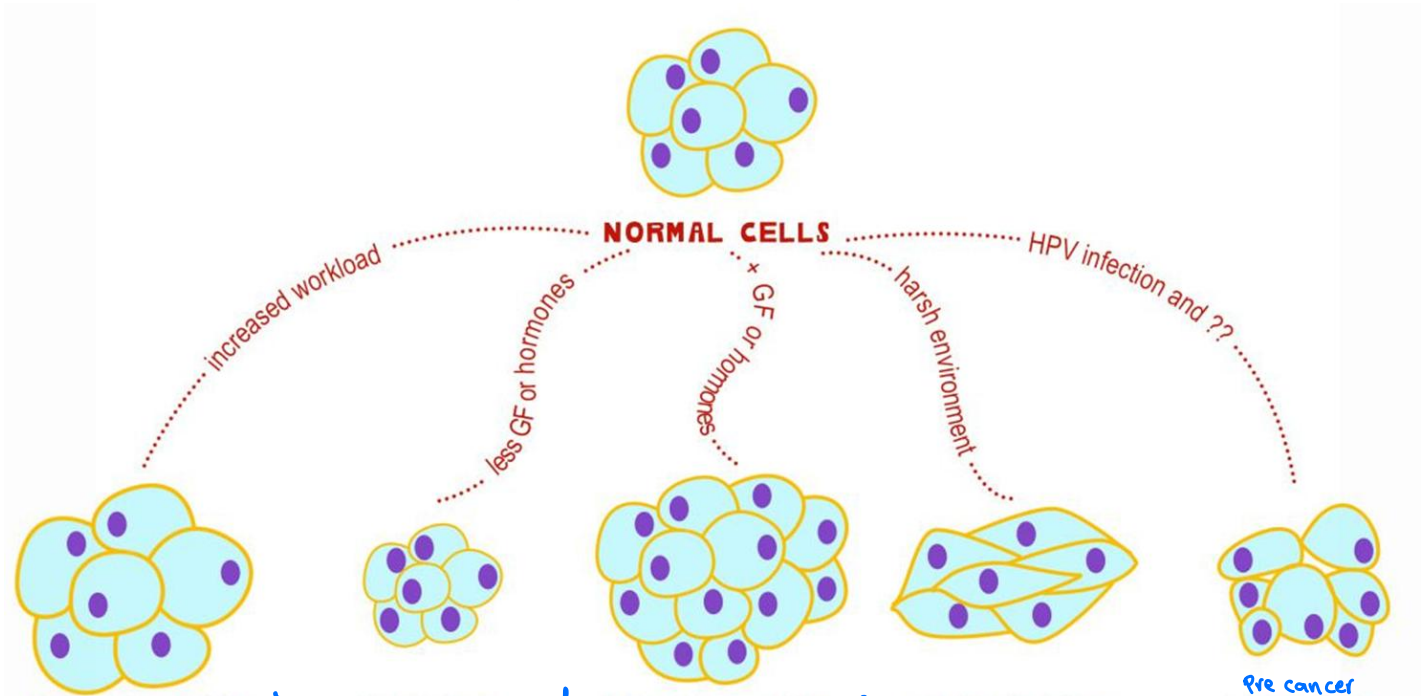
Response to cell stress/injury



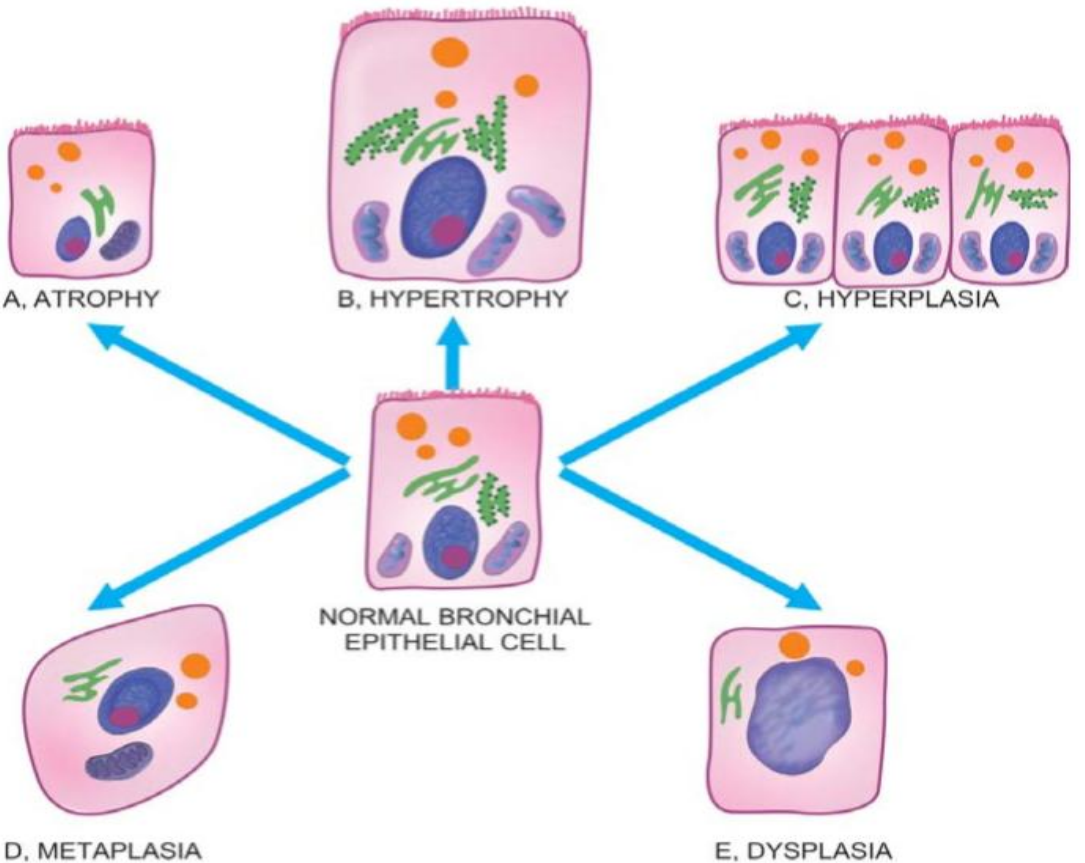

Cellular Responses
Homeostasis
Adaptation
Reversible cell injury
Irreversible cell injury
Death of cell
Disease

Classification

types of cellular adaptation



How do the cells makes these adjustments?



Cellular Adaptations

Cellular adaptations is **achieving a new steady state** (adjustments the cells make) in order for the cells **to stay viable** and to do their function (adverse environmental changes)

PHYSIOLOGIC

are responses to normal stimulus or demands of mechanical stress

PATHOLOGIC

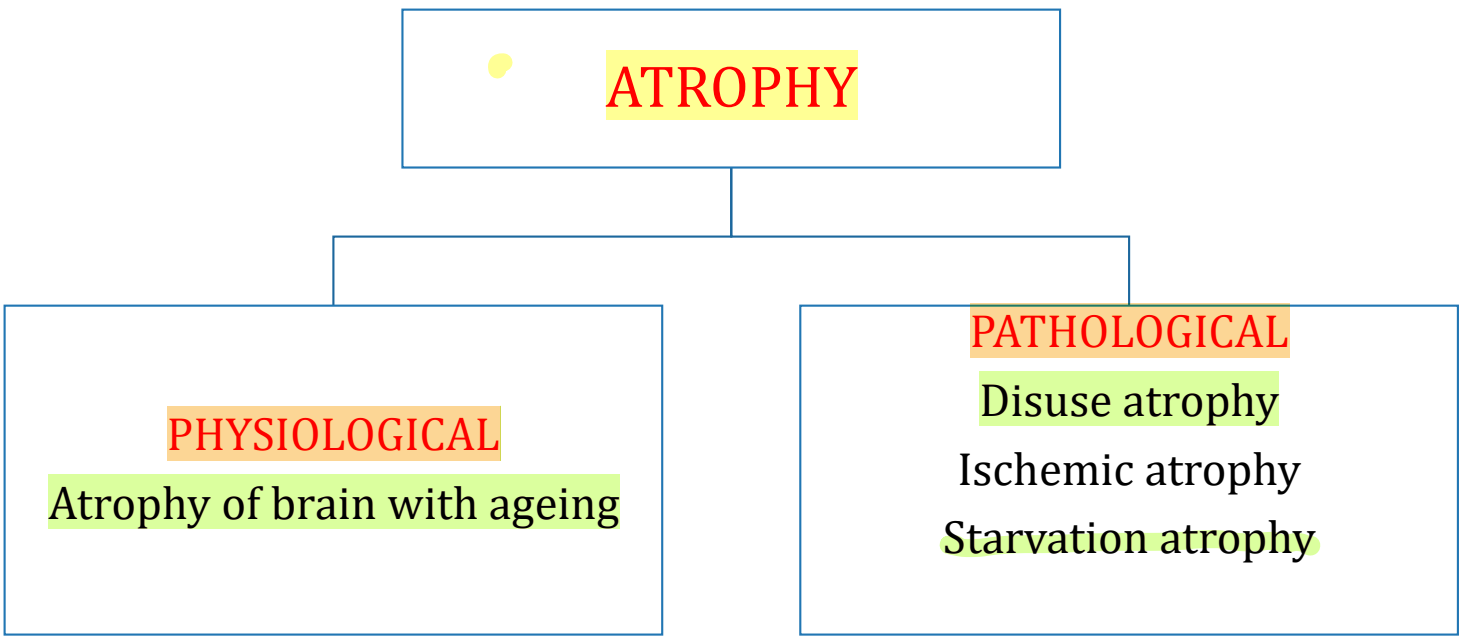
are responses in which cells modify their structure and function to escape injury, but at the expense (loss) of normal function

Cellular Adaptations are reversible on removal of stress

Atrophy

- Reduction in the number and size of a cell or tissue or an organ or its parts. 
- Cells become smaller in order to survive.
- Reduction is size due to individual cells undergoing a combination of autophagy and apoptosis or just apoptosis in ‘old’ cells
- Atrophic cells may have diminished function.

Cause of atrophy



Mechanism:

1. Decreased protein synthesis
2. Increased protein degradation in the cells

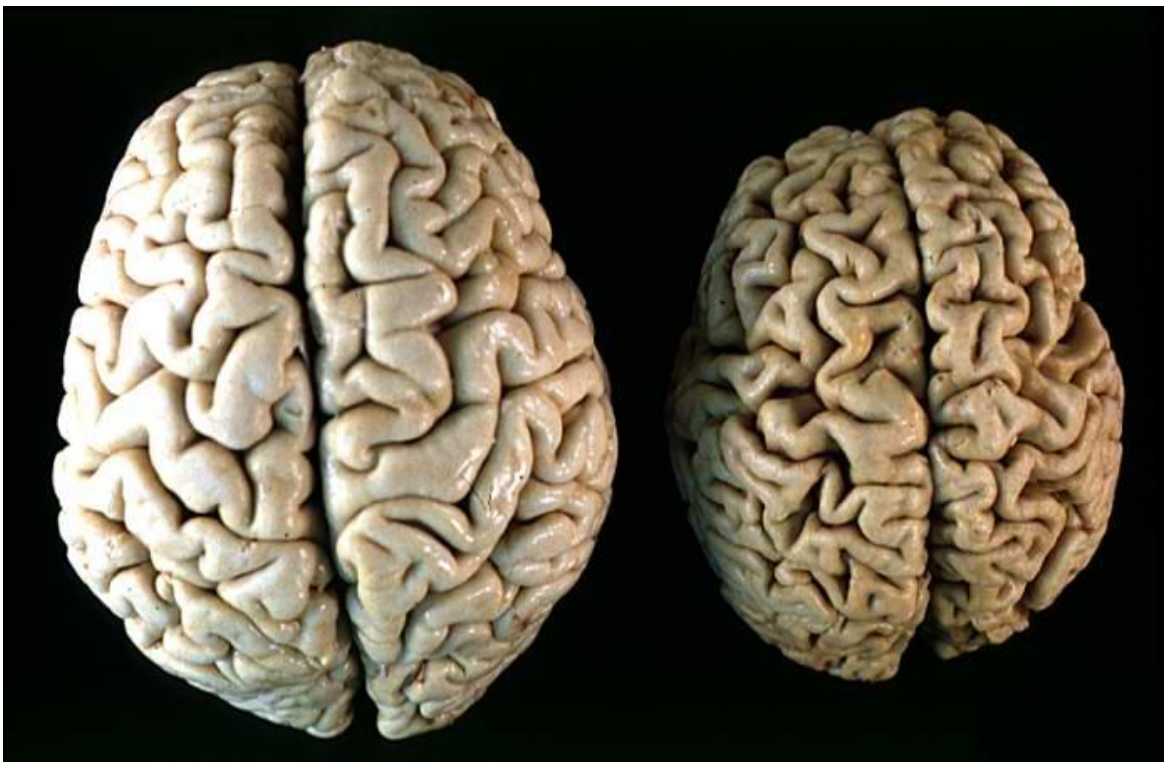
Morphology of atrophy

- **GROSS:** The organ is small and shrunken

- **MICROSCOPY:** The cells are smaller in size due to reduction in cell organelles

Brain atrophy

This gross photograph shows a normal brain (left) and a brain from a geriatric patient (right). Note the decreased size, the narrowed gyri, and the widened sulci of the brain



Pathological

Disuse atrophy

it will become smaller after fracture

less blood supply

Ischemic atrophy

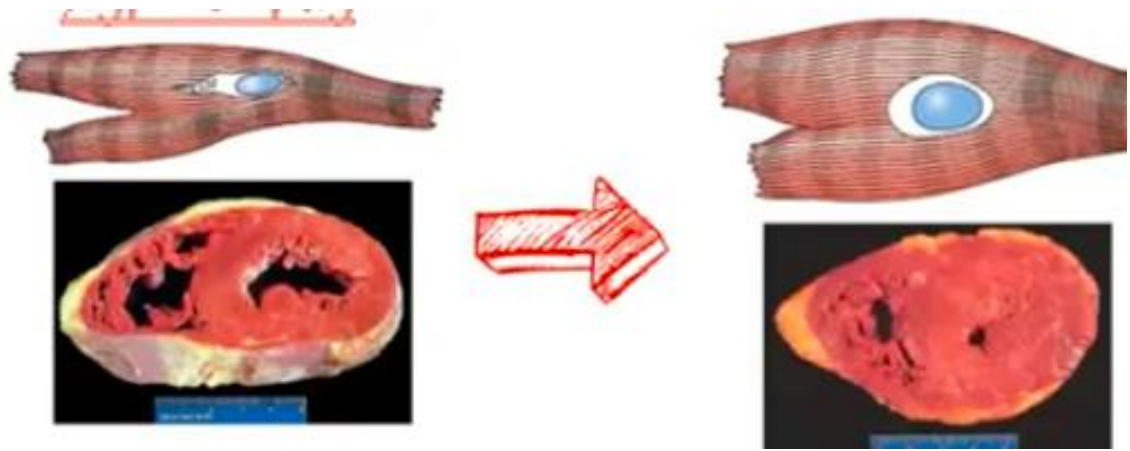
shrink due to less blood supply in left kidney

Starvation atrophy

HYPERTROPHY

INCREASE IN THE SIZE OF THE CELLS

ENLARGEMENT OF THE ORGAN OR TISSUE,



- It is mostly seen in cells that cannot divide, such as **skeletal muscle** (pumping iron), and **cardiac muscle** (hypertension). (permanent cells).
- **No new cell division** in **hypertrophy.**
- Dividing cell ---------- Hypertrophy + Hyperplasia
- Non-dividing cell--------Hypertrophy
- These changes usually revert to normal if the cause is removed.

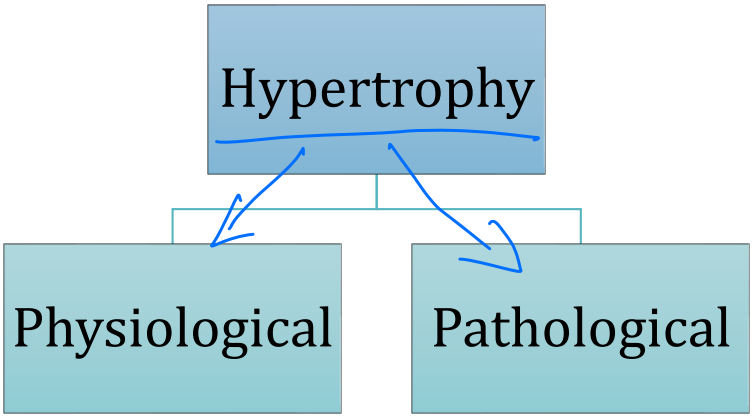
Cause : of hypertrophy

- Increased functional demand **or**

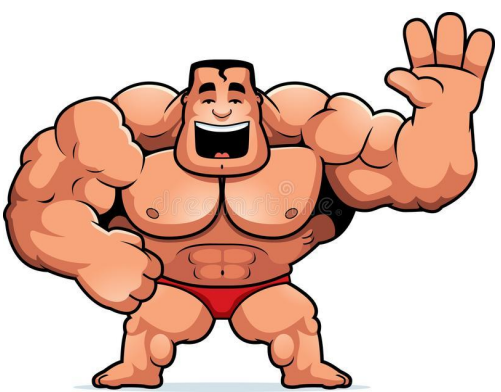
  By stimulation of hormones and growth factors

Mechanisms :

- Increased production of cellular proteins.



- Most common stimulus for hypertrophy: Increased work load



Types of hypertrophy

Physiologic

- Muscles hypertrophy in body builders

Pathologic

Hypertrophy of cardiac muscle

Hypertrophy of Smooth muscle

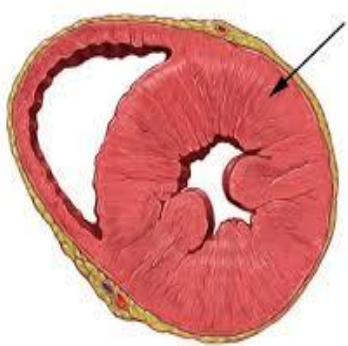
Hypertrophy of skeletal muscle

* Compensatory Hypertrophy (kidney)

Body builder skeletal muscle.

CARDIAC HYPERTROPHY

Hypertrophy of cardiac muscle may occur. Left ventricle hypertrophy (LVH) seen in Hypertension, Patient
Aortic valve disease



This image demonstrates left ventricular hypertrophy, represented by the abnormally thick muscular wall of the left ventricle (arrow) relative to the right (left side of the image).

Myocardial hypertrophy.

- Cross-section of the heart of a patient with long- standing hypertension
- Shows pronounced, concentric left ventricular hypertrophy

Compensatory hypertrophy

one kidney not function so the other kidney will increase in size

In an organ when the contralateral organ is removed e.g. kidney (there is also hyperplasia)

Hyperplasia

- Hyperplasia is an **increase in the number of parenchymal cells**.
- Abnormally increased rate of cell division or failure of apoptosis – results in tissue/organ getting bigger
  decrease in death cells
- Hyperplasia may sometimes co-exist with hypertrophy.
- Hyperplasia persists only till the stimulus is present **(different from neoplasia)**

It's reversible, but cancer is not

Physiologic hyperplasia

reversible

Hormonal hyperplasia – e.g., Enlarged size of the uterus in pregnancy.

Pathologic hyperplasia

reversible

Chronic injury/irritation: lead to hyperplasia , especially in skin or oral mucosa (Excess granulation tissue during wound healing or formation of skin warts)

METAPLASIA

- Metaplasia is a **reversible change in which one adult cell type (epithelial or mesenchymal) is replaced by another adult cell type**
- It usually in response to abnormal stimuli and often reverts back to normal on removal of stimulus.
- **Types of metaplasia:**
    1. Epithelial
    2. Mesenchymal

- Metaplasia of epithelial cells in the conductive region of the airways:
- Original cell → sustained stress → metaplasia
- Adaptation is useful as it **allows for cell survival,** however, some functions are lost

A. EPITHELIAL METAPLASIA.

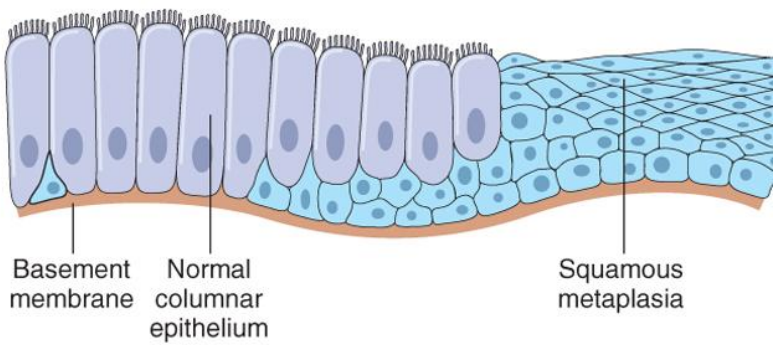
in smokers

- Epithelial is the more common type, usually the cell becomes less specialized.

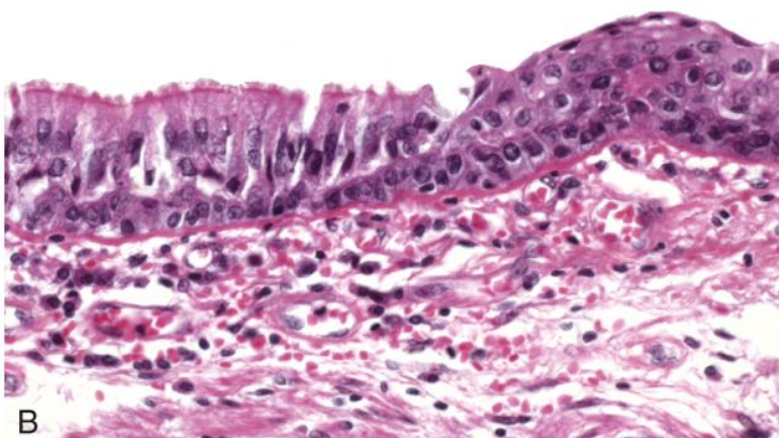
1. **Sqamous metaplasia:** original epithelium replaced by squamous epithelium.
   - Bronchi of smokers- pseudo stratified ciliated columnar to squamous epithelium.

2. **Columnar metaplasia:** original epithelium replaced by columnar epithelium.
   - Barrett esophagus: the squamous cells of eosphagus replaced by columnar

Epithelial metaplasia is exemplified by the squamous change that occurs in the **respiratory epithelium in habitual chronic cigarette smokers**



A



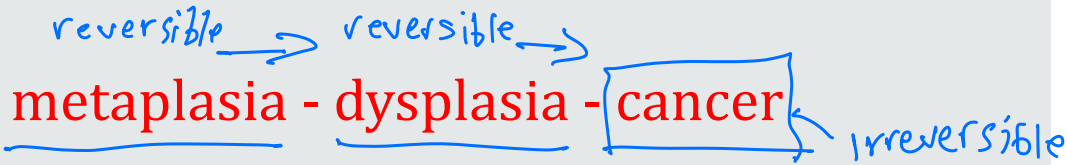
B

The normal ciliated columnar epithelial cells of the trachea and bronchi are focally or widely **replaced by stratified squamous epithelial cells.**

The rugged stratified squamous epithelium may be able **to survive the noxious chemicals in cigarette smoke** that the more fragile specialized epithelium would not tolerate.

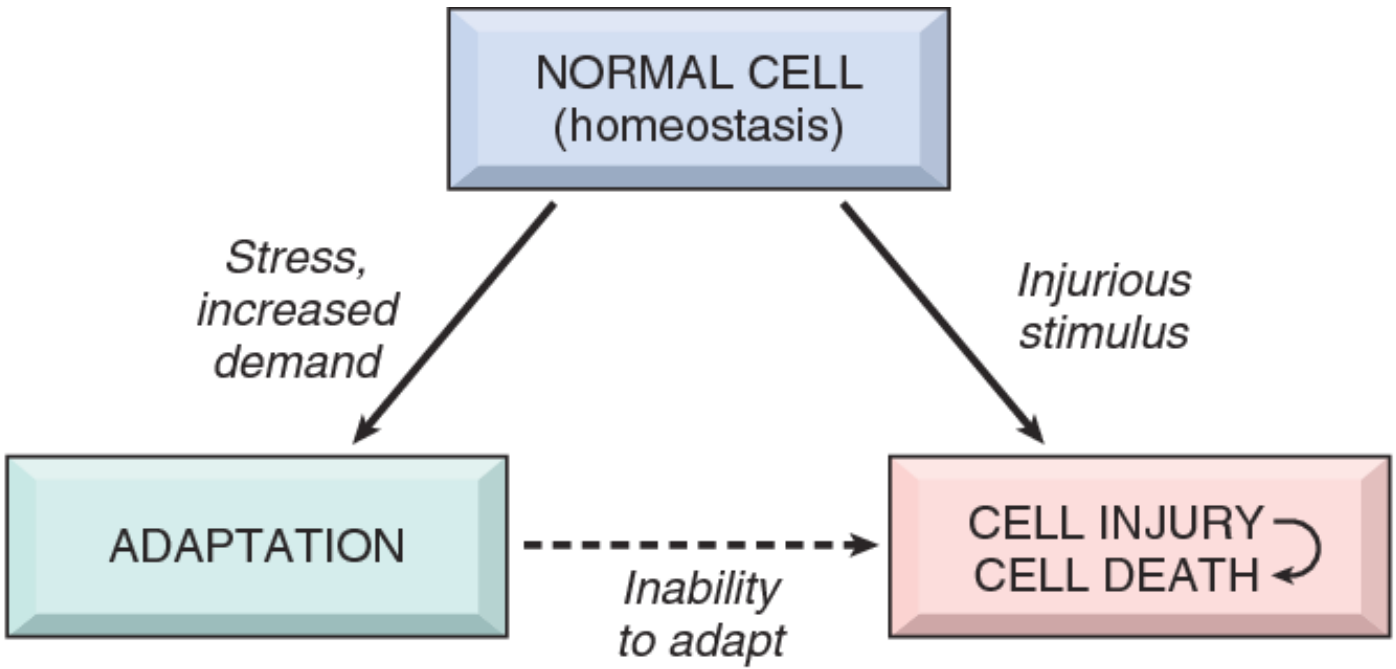
- Changed cell type → change function → results in coughing
- **Take away stressor** → reversible • Metaplasia and hyperplasia increases risk of cancer

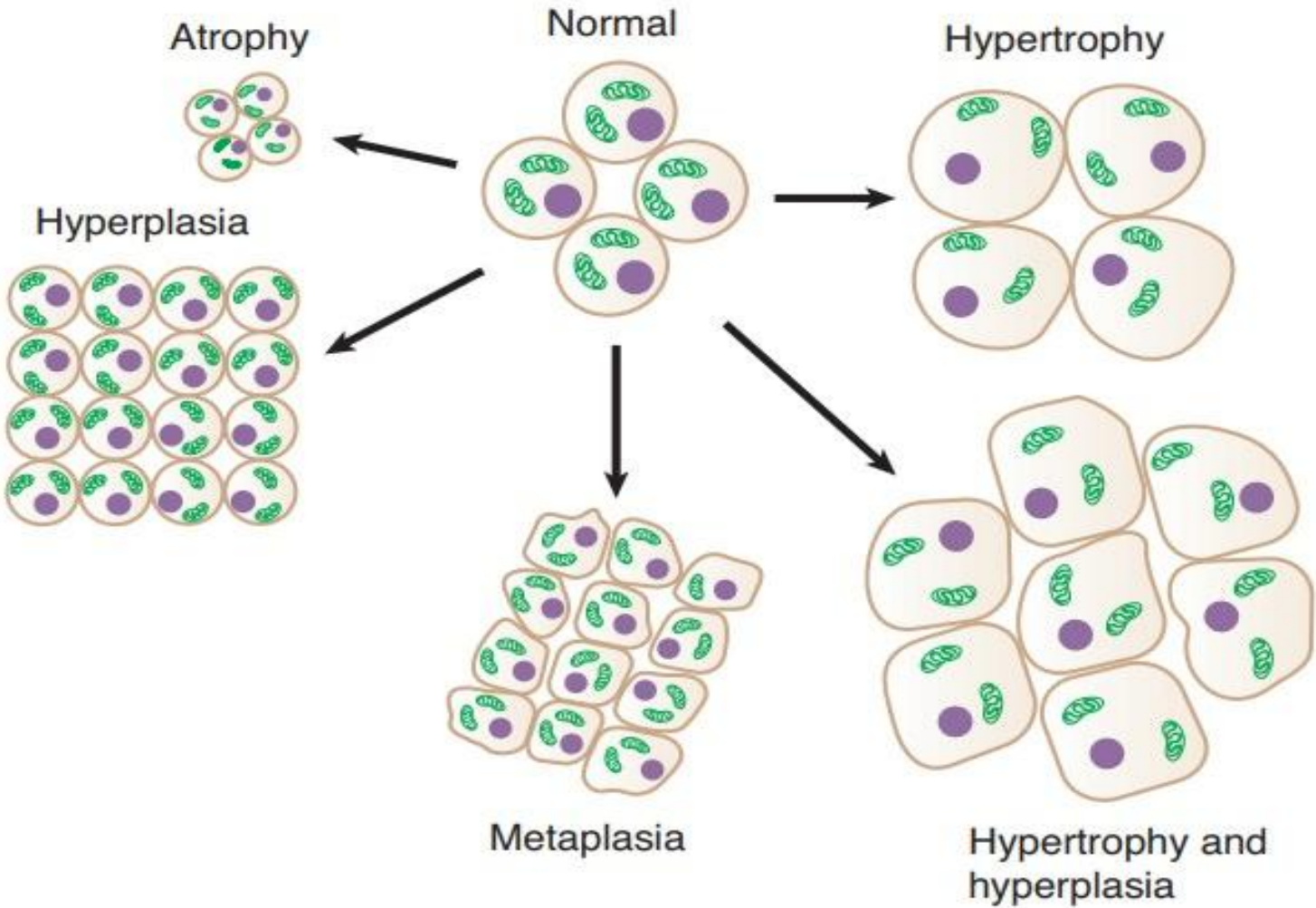
If abnormal stimuli is persistent,



e.g. lung cancer in smokers

Summary of lecture




Atrophy
Normal
Hypertrophy
Hyperplasia
Metaplasia
Hypertrophy and hyperplasia

Essential Learning Resource

- **Harsh Mohan: Essential Pathology for Dental Students** (with Practical Pathology). 5th ed; 2017; Jaypee Brothers Medical Publishers
- **Harsh Mohan: Textbook** of Pathology. 7th ed; 2014; Jaypee Brothers Medical Publishers
- **Kumar: Robbins Basic** Pathology. 10th ed; 2017; Elsevier

Thank you