ADAPTATION
Lecture-3

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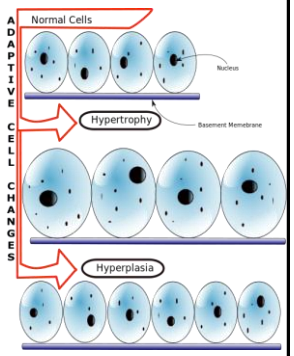
HYPERPLASIA

- Increase in the number of cells in an organ or tissue
- Increase in tissue or organ size
  - physiologic
  - pathologic

In both situations, cellular proliferation is stimulated by growth factors

the hyperplastic process remains controlled; if the signals that initiate it stops, the hyperplasia disappears.



http://en.wikipedia.org/wiki/File:Hyperplasia_vs_Hypertrophy.svg

Physiologic

- Occurs due to a normal stressor.
  - increase in the size of mammary tissue near and after parturition
  - increase in size of of endometrium during pregnancy,
  - and liver growth after partial resection

Role of growth factors and cytokines, e.g., *Hyperplasia of bone marrow cells* producing red blood cells in animals living at high altitude. This is stimulated by increased production of the growth factor erythropoietin

Pathologic

- Occurs due to an abnormal stressor.
  - growth of adrenal glands due to production of ACTH by a pituitary adenoma,
  - proliferation of endometrium due to prolonged estrogen stimulus.
- Pathologic hyperplasia may precede the development of malignancy/neoplasia/cancer
- In certain viral infections; for example, papilloma viruses cause skin warts and mucosal lesions composed of masses of hyperplastic epithelium.

HYPERPLASIA

- After partial hepatectomy, Hepatocytes begin to divide by 12 hours,
- It takes about 1 to 2 weeks to complete,
- After restoration of the liver mass, cell proliferation is "turned off" by various growth inhibitors.

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HYPERPLASIA

Factors driving compensatory hyperplasia

- HGF released from non-parenchymal cells binds with c-Met receptor present on hepatoctyes
- TGF-α and EGF are also mitogenic for hepatocytes
- IL-6 and TNF-α are produced early in hepatic regeneration, and are necessary for the proliferative response

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HYPERPLASIA

Resolution of compensatory hyperplasia

- TGF-β is an important inhibitor, which is also produced by non-parenchymal cells in the liver

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HYPERPLASIA

Concept of reversibility

- Removal of hormonal stimulation
- Removal of inciting cause

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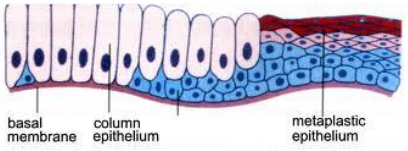
METAPLASIA

- **One differentiated (Highly Specialized) cell type replaced by another differentiated (Less Specialized) cell type.**
- **As a response to Better Withstand the adverse environment**
- **Occurs mostly due to chronic irritation, including inflammation.**
- **Cigarette smokers**

  Columnar → Stratified squamous epithelium.
- **Vitamin-A deficiency; Squamous metaplasia of prostate gland**



a model of squacmous epithelial metaplasia

- Metaplasia is usually an adaptive response to persistent injury.
- **Genetic reprogramming**
  - Reprogramming of epithelial stem cells from one epithelial cell type to another
  - Reprogramming of mesenchymal stem cells (pluri-potent) so that they differentiate along a different pathway
  - Due to the action of cytokines, growth factors, etc and the subsequent activation of
    - tissue-specific genes

- **In most of the cases,**

  Proliferation of germinal cells or stem cells
- **In fewer cases, direct transformation of differentiated cells**
- **Metaplasia is usually fully reversible.**
  - If the noxious stimulus is removed (e.g., when one stops smoking), the metaplastic epithelium eventually returns to normal.

Metaplasia

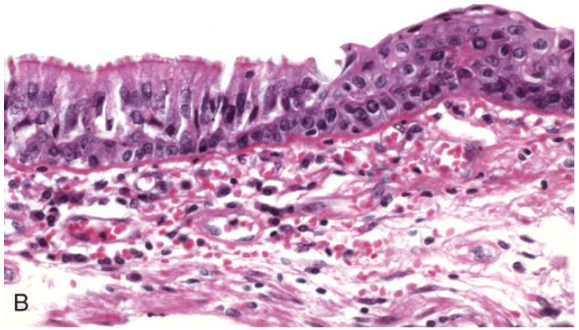
- ***Microscopically***
  - **In mesenchymal metaplasia**

    Fibrous tissue ---- bone or cartilage.
  - **Osseous metaplasia in cartilage**

    Costal cartilages
  - **Osseous metaplasia in soft tissues,**

    In scars and in areas of dystrophic calcification

Metaplasia



Squamous metaplasia in smokers

Metaplasia

- **Grossly**
  - –Difficult to appreciate grossly