

CELLULAR ADAPTATIONS

lec no. 1

Adaptive Mechanisms (all are REVERSIBLE)

1) Hypertrophy ($\uparrow$ cell size = $\uparrow$ organ size = $\uparrow$ function)

- pure (non-dividing cells), mixed (dividing cells)
- more organelles & proteins = $\uparrow$ size
- causes:
 - hormonal stimulation $\rightarrow$ ex. uterine smooth muscle
 - growth factor stimulation
 - increased functional demand $\rightarrow$ ex. cardiac hypertension in aortic stenosis

2) Hyperplasia ($\uparrow$ cell number = $\uparrow$ organ size)

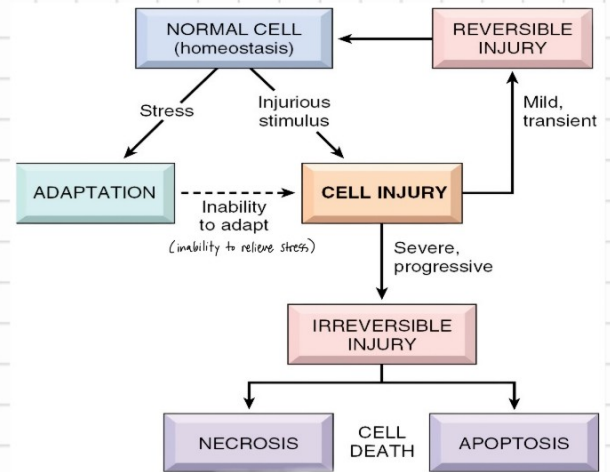
- tissues with proliferative ability
- pure, mixed
- pathologic + mutations = cancer (irreversible)
- causes:
 - hormonal stimulation (normal / excessive)
 - compensatory
 - viral infections

3) Atrophy ($\downarrow$ cell size = $\downarrow$ organ size = $\downarrow$ function)

- Mechanisms:
 - $\downarrow$ protein synthesis
 - $\uparrow$ degradation
 - $\uparrow$ autophagy
- causes:
 - decreased workload $\rightarrow$ immobilization
 - loss of innervation
 - diminished blood supply (ischemia)
 - inadequate nutrition
 - loss of endocrine stimulation $\rightarrow$ ex. menopause
 - aging (sterile atrophy)

4) Metaplasia (change cell type)

- reprogramming stem cells
- increased risk of cancer
- better coping, less functional
- causes:
 - smoking $\rightarrow$ squamous metaplasia, bronchi
 - Vit A deficiency $\rightarrow$ squamous metaplasia, bronchi or any site lined with differentiated epithelium
 - gastroesophageal reflux disorder (GERD) $\rightarrow$ stratified squamous $\rightarrow$ intestinal (columnar) epithelium with goblet cells to cope with acidity



Cell Injury

- Causes:
 - Oxygen Deprivation $\rightarrow$ atherosclerosis may cause ischemia which leads to hypoxia/apoxia (most common)
 - Chemical Agents $\rightarrow$ pesticides/insecticides/glucose (diabetic cell death)
 - Infectious Agents $\rightarrow$ pathogens: viruses/bacteria/fungi/protozoa
 - Immunologic Reactions $\rightarrow$ autoimmune/allergic/microbes
 - Genetic factors (chromosomal abnormalities)
 - $\rightarrow$ DOWN Syndrome
 - Nutritional Imbalances $\rightarrow$ Over-nutrition (obesity) $\rightarrow$ Under-nutrition (atrophy)
 - Physical Agents $\rightarrow$ burns/electrical shocks/any physical trauma (falling,...)

* ALL EXAMPLES ARE IN THE TABLE BELOW *

CASE	ADAPTATION	PHYSIOLOGIC VS PATHOLOGIC	DRIVING CAUSE	NOTES
Cardiac hypertension & aortic stenosis	Hypertrophy (pure)	pathologic	increased functional demand (pressure overload)	
Skeletal muscles (athletes)	Hypertrophy (pure)	physiologic	increased functional demand (mechanical stress)	
Uterine smooth muscle enlargement (pregnancy)	Hypertrophy (mixed)	physiologic	hormonal stimulation ($\uparrow$ estrogen)	
Breast enlargement (puberty/pregnancy/ lactation)	Hyperplasia (mixed)	physiologic	hormonal stimulation	puberty $\rightarrow$ estrogen, growth hormone, ... pregnancy/lactation $\rightarrow$ estrogen progesterone prolactin, ...
Liver after partial resection	Hyperplasia (pure)	physiologic	compensatory hyperplasia (growth factors)	
Endometrial Hyperplasia	Hyperplasia (pure)	pathologic	excessive hormonal stimulation ($\uparrow$ estrogen)	* May develop into endometrial carcinoma
Benign prostatic Hyperplasia (BPH)	Hyperplasia (pure)	pathologic	excessive hormonal stimulation ($\uparrow$ androgen)	
Warts	Hyperplasia (pure)	pathologic	viral infection (human papillomavirus, HPV)	
Decreased uterus size after Menopause	Atrophy	physiologic	loss of endocrine stimulation ($\downarrow$ estrogen)	
Denervation injury	Atrophy	pathologic	loss of innervation	
Chronic ischemia	Atrophy	pathologic	diminished blood supply	
Chronic nutrition deprivation/Malnutrition	Atrophy	pathologic	inadequate nutrition	
Squamous Metaplasia	Metaplasia	pathologic	Smoking (bronchi)	
Squamous Metaplasia	Metaplasia	pathologic	Vit A deficiency	
Gastroesophageal Metaplasia	Metaplasia	pathologic	Chronic GERD	
* extra example				
Proliferative phase of menstrual cycle (endometrial cell proliferation)	Hyperplasia (pure)	physiologic	hormonal stimulation ($\uparrow$ estrogen)	