GENERAL PATHOLOGY

L-5 CELL INJURY

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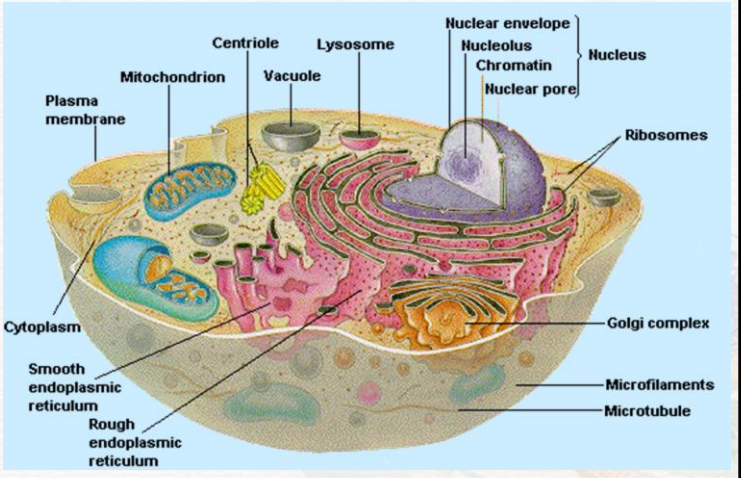

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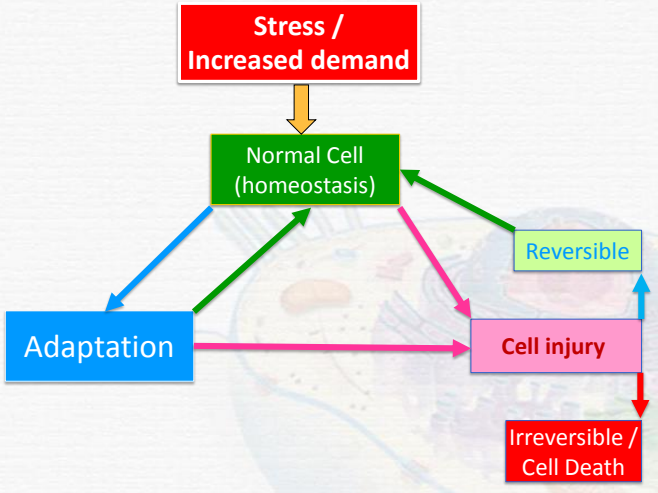

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CELL INJURY

- No adaptive response or adaptive capacity is exceeded
- "Any change that results in a loss of the ability to maintain normal or adapted homeostatic state" -- Cell Injury.

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Severity of Cell Injury

Dependent on

- Severity and type of stress — **Acid – strong / weak**
- Blood Supply and Nutrition — **Single / double**
- Metabolic state of cell — **Active / resting**
- Previous state of cell — **Adaptive / normal**
- Duration of insult
- Type of Cell involved

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intensity of insult & duration of insult } **Hot Water**

1 sec at 160^0
2 min at 122^0
45 min at 109^0

Type of cell involved — **Hypoxia**

Brain Cells	4-5 min
Renal Cells	~20 min
Cardiac muscles	~ 20-30 min
Skeletal muscle	~2-3 hrs

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Cellular Expressions in Response to Stress

- **Reversible Injury**
  1. Cell swelling
  2. Fatty Change
- **Irreversible Injury**
  1. Necrosis
     - Coagulative
     - Liquefactive
     - Caseative
     - Fat necrosis
     - Zenker's Necrosis
  2. Apoptosis

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Cell Injury - Causes

A Generalized List

- Oxygen
- Chemical agents and drugs
- Infectious organisms
- Immune reactions
- Genetic derangements
- Nutritional imbalances
- Physical agents
- Aging

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- **Oxygen deprivation**
  - *Hypoxia*, or decreased O_2 in the blood,
  - *Ischemia* is a decrease or loss of blood supply,
  - Carbon monoxide (CO) poisoning wherein CO binds so avidly to hemoglobin that O_2 has no binding site.
- **Chemical agents**
  - Everything in moderation: ↑↑↑ Blood glucose → shift of H_2O into blood → hyperosmolar coma.
  - Poisons: Alter membrane permeability or enzyme function.
  - Organic and inorganic environmental agents.
  - Therapeutic agents/drugs.

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- **Infectious agents**
  - Viruses.
  - *Chlamydia.*
  - *Rickettsia.*
  - Bacteria.
  - Fungi.
  - Parasites.

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- **Immune reactions**
  - *Anaphylaxis.*
  - *Loss of tolerance* by T cells
- **Genetic defects**
  - Single amino acid substitution (hemoglobin S in sickle cells).
  - Enzyme deficiencies.
  - Anatomic malformations

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- Sickle cell disease
- The sickling occurs because of a mutation in the hemoglobin gene "HbSS"
- sickle-shaped red blood cells that obstruct capillaries and restrict blood flow to an organ, resulting in ischemia.

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- **Nutritional imbalances**
  - Protein-calorie.
  - Vitamins.
  - Excess.
    - Type 2 diabetes mellitus.
    - Atherosclerosis.
- **Physical agents**
  - Trauma
  - Temperature
  - Radiation
  - Electricity
  - Atmospheric pressure

- **Aging**
  - Cellular senescence.
  - Degeneration.
  - Poor healing.

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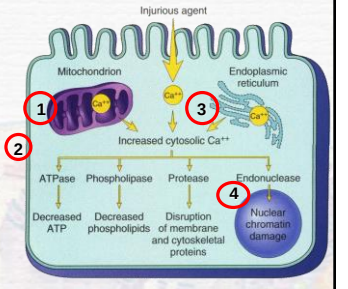
CELL INJURY

- Vulnerable intracellular systems
  1. ATP generation.
     - Mitochondrial aerobic respiration.
  2. Cell membrane integrity.
     - Ionic homeostasis.
     - Osmotic homeostasis.
  3. Protein synthesis.
     - Cell structure and function.
  4. Integrity of the genetic apparatus.
     - Access to DNA.
     - Interactions with DNA.
     - DNA replication.
     - DNA repair mechanisms.



- **Injury at one focus often has a cascade effect**
- **Morphological changes occur only after critical biochemical (molecular) damage**

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