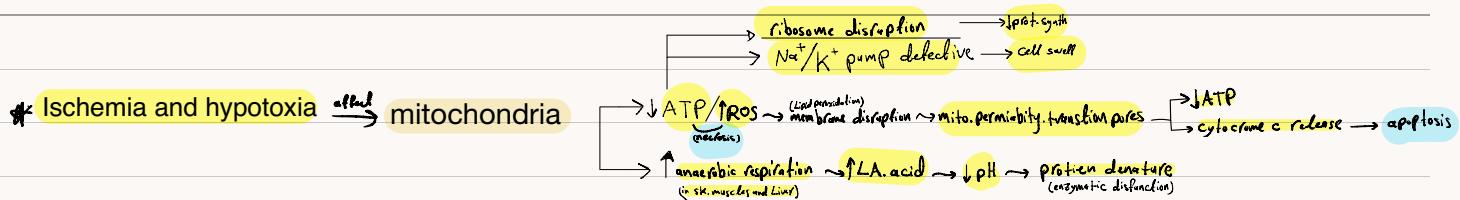


## Cell injury mechanisms



\* Ischemia reperfusion → (reformation blood supply affect mitochondria metabolic ↑ ROS [oxidative stress because of the high metabolic reactions after ischemia] making ROS more than cell capacity of anti-oxidant defense system]

**ROS** → all cell normally in Redox rxns when  $O_2$  reduced to  $H_2O$  (normal amount unless ischemia/reperfusion (oxidative stress))

→ leukocytes ( $O_2 \rightarrow H_2O_2$  (superoxide → Hydrogen peroxide) →  $ClO_3^-$  (for killing microbes and cause inflammation)) / pathogen cause cell necrosis So leukocytes accumulate and cause inflammation

→ ROS effects → lipid peroxidation (membrane disruption) / proteins denaturation / DNA damage



\* **Toxins** → **direct:** ① MgCl<sub>2</sub> bind with SH group of membrane proteins → active transport (permeability) / ② chemotherapeutic agents / ③ microorganism toxins  
**labeled:** CCl<sub>4</sub> → cyclohexene-<sup>14</sup>C SER in Liver converted into free radical (membrane effect in foods (numbers of ER + lysosomes → fatty liver) / mitochondrial membrane → KTP (potassium, DNA)

\* Misfolded proteins → increase when ↓ protein synthesis, ↑ degradation, ↑ chaperon synthesis.  
 (false proopiomelanocortin ( $\alpha$ H<sub>2</sub>) → caspase activation → apoptosis)

diseases → protein deficiency (cystic fibrosis, α1-antitrypsin) / neurodegenerative (Parkinson's)

\* DNA damage  $\xrightarrow{\text{causes}}$  (mutation, radiation, ROS, chem. agent)  $\xrightarrow{\text{p53 activates}}$  stay in G1 or apoptosis  
 $\xrightarrow{\text{p53 mutation}}$  mutation replication  $\rightarrow$  neoplastic changes

