

Q: What is the central unifying concept of oxidative stress in disease?

A: A shared pathophysiological mechanism in which redox imbalance drives inflammation, mitochondrial dysfunction, and tissue injury across multiple organ systems.

Q: What is redox dysregulation in disease contexts?

A: A sustained failure of antioxidant systems to control reactive species, leading to chronic signaling distortion and molecular damage.

Q: What is the mitochondria-centered disease concept in oxidative stress?

A: Mitochondria act as both primary ROS sources and amplifiers of cellular injury, linking metabolism to disease progression.

Q: What is oxidative inflammation coupling?

A: A bidirectional loop where ROS activate inflammatory pathways and inflammation further increases ROS production.

Q: What is endothelial redox dysfunction?

A: Loss of nitric oxide signaling due to oxidative inactivation, resulting in impaired vascular tone and vascular disease.

Q: What is lipid peroxidation cascade pathology?

A: Chain-reaction oxidation of membrane lipids leading to structural instability and bioactive aldehyde formation.

Q: What is protein redox misfolding in disease?

A: Oxidative modification of proteins causing loss of function, aggregation, and proteostasis overload.

Q: What is oxidative DNA damage as a disease driver?

A: Accumulation of mutations and genomic instability due to incomplete repair of ROS-induced lesions.

Q: What is ferroptosis in disease pathology?

A: Iron-dependent cell death driven by lipid peroxide accumulation and failure of glutathione-dependent detoxification.

Q: What is mitochondrial failure syndrome in oxidative disease?

A: Bioenergetic collapse caused by ROS-induced damage to electron transport chain components.

Q: What is redox signaling importance in chronic disease?

A: Conversion of physiological ROS signaling pathways into persistent pathological activation states.

Q: What is antioxidant depletion syndrome conceptually?

A: Exhaustion of enzymatic and non-enzymatic buffering systems under chronic oxidative load.

Q: What is compartmentalization failure in oxidative pathology?

A: Loss of spatial restriction of ROS, leading to widespread molecular damage beyond intended signaling domains.

Q: What is inflammatory redox amplification?

A: Self-sustaining cycle where cytokines increase ROS production and ROS further activate inflammatory transcription.

Q: What is metabolic–redox coupling in disease?

A: Interdependence of energy metabolism and ROS production where metabolic dysfunction directly increases oxidative stress.

Q: What is proteostasis collapse in oxidative disease?

A: Failure of chaperones and degradation systems due to oxidative overload and protein misfolding accumulation.

Q: What is vascular redox injury?

A: Structural and functional damage to blood vessels driven by oxidative modification of endothelial and smooth muscle cells.

Q: What is oxidative stress threshold theory?

A: Concept that disease onset occurs when ROS generation exceeds adaptive and repair capacity thresholds.

Q: What is redox network failure?

A: System-wide breakdown of coordinated antioxidant, signaling, and repair mechanisms.

Q: What is the modern integrative disease model of oxidative stress?

A: A systems-level interaction of mitochondrial dysfunction, inflammation, metabolic imbalance, and proteostatic failure forming a self-reinforcing pathological network.