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https://mcw.marquette.edu/biomedical-engineering/computational-lung-physiology-lab/index.php
Oxidative stress, inflammation, and cell death are common pathways in the pathogenesis of ALI/ARDS.

Acute inflammation results in damage to the alveolar-capillary barrier, leading to alveolar edema and severe impairment of oxygenation.

Injury to the pulmonary capillary endothelium is the primary cause of increased-permeability pulmonary edema in ALI/ARDS.
Animal Models of Human ALI/ARDS

- Animal models have been developed to evaluate the time course, severity and pathophysiological mechanisms of ALI/ARDS.

- Two well-established rat models (direct insult):
  - Exposure to 100% O₂ (hyperoxia).
  - Treatment with intratracheal endotoxin (lipopolysaccharide, LPS).

- Both models reproduce the cardinal features of clinical ALI/ARDS:
  - Bilateral infiltration
  - Increased microvascular permeability
  - Low-pressure edema
  - Hypoxemia
  - Endothelial cell death
Preliminary results using DSSQ in lungs from rats exposed to room air (normoxia) or high oxygen (hyperoxia) as a model of human ARDS.

Change in emission signal (525 nm, counts)

Time (min)

0 10 20 30 40

-6e+5 -4e+5 -2e+5 0 2e+5 4e+5 6e+5 8e+5
Isolated Perfused Rat Lung Preparation

15% O₂, 6% CO₂

Pump

Reservoir

Rat lung
Preliminary results (DSSQ reduction) in plasma from rats exposed to room air (normoxia) or high oxygen (hyperoxia) as a model of human ARDS

DSSQ (50 mM) in plasma from normoxic and hyperoxic rats (Nrf2 WT, Nrf2 homo, NOX4 homo)
EX 485 nm, EM 525 nm