Acute Respiratory Distress Syndrome (ARDS), a common and highly fatal disease that is characterized by an inflammatory exudate in the air spaces in the lungs. ARDS can develop after many different insults including sepsis, trauma, or pneumonia.

Alveolar macrophages and neutrophils have been implicated in ARDS development.

Following exposure to hyperoxia, rats develop ARDS characterized by rapid increases in neutrophils, protein, and LDH concentration in their lung lavages that occur after 52 hours and before death at 66 hours.

Alveolar macrophages have two phenotypes. The M1 phenotype induces a pro-inflammatory response. The M2 phenotype induces an anti-inflammatory response.

The effect of hyperoxia on M1/M2 phenotypes and its relationship to ARDS development is unknown.

Hyperoxia Exposure
Collected Bronchoalveolar Lavage (BAL) fluid from control Sprague Dawley (SD) rats and SD rats exposed to hyperoxia; pressurized at sea level with flow of 100% oxygen for 52-54 hours. Cell pellet and BAL supernatant was isolation via centrifugation.

M1/M2 Phenotype
Used flow cytometry to determine whether the alveolar macrophage phenotype was M1 or M2. Used CD11b and Forward Side Scatter to characterize macrophages. Used CD163 to identify the M2 phenotype and CD80 to identify the M1 phenotype. Each primary antibody was conjugated to a distinct fluorochrome.

Protein Concentration
Performed a Bicinchoninic Acid (BCA) assay to determine the concentration of protein in the BAL. Used Sigma-Aldrich Kit.

LDH Levels
Cytotoxicity Assay was used to determine amount of LDH in control and hyperoxia exposed rats. Used Promega CytoTox 96 Non-Radioactive Cytotoxicity Assay Kit.

Lung Lavage Cell Counts
Amount of alveolar macrophages and amount of neutrophils in the lung lavages of control and hyperoxia exposed rats were determined through cell counts using a hemocytometer.

Hypothesis
An alveolar macrophage phenotype switch from M2 to M1 is induced in rats exposed to hyperoxia.

Hyperoxia induces ARDS.

Hyperoxia induces an influx of neutrophils into the lungs.

Hyperoxia changes alveolar macrophage phenotype from M2 to M1.

Our results suggest that hyperoxia shifts alveolar macrophage phenotype from M2 to M1, a change that potentially contributes to ARDS development.

Investigating alveolar macrophages under a spectrum of oxygen conditions.

Investigating how to prevent the phenotype switch from M2 to M1 to potentially impede ARDS development.


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