Abstract Submitted
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Pulmonary surfactant and macrophages studied at the air/liquid interface revealed by Brewster angle microscopy (BAM) DANA-MARIE TELESFORD, HEATHER ALLEN, Department of Chemistry & Biochemistry, The Ohio State University, TRACY CARLSON, Department of Veterinary Biosciences, The Ohio State University, LARRY SCHLESINGER, Department of Microbial Infection & Immunity, The Ohio State University — The alveolus is lined with a complex mixture of lipids and proteins called pulmonary surfactant (PS) that lower surface tension at the alveolar air/liquid interface. The surface area of the lung for a 70 kg adult human at total lung capacity is $\sim$70 m^2. The large surface area and the direct exposure to the environment with every inhalation make this organ more susceptible to invasion by viruses, bacteria, and small particles. The most abundant cell recovered in human lung lavage is the alveolar macrophage which accounts for 85% of the total. The primary function of the alveolar macrophage is to defend the lung against invasion, but also in the clearance of surfactant components in the lung. Quintero and Wright,[1] in an in vitro study observing alveolar macrophage metabolism of two lipid components dipalmitoyl phosphatidylglycerol (DPPG) and dipalmitoyl phosphatidylcholine (DPPC), noted that DPPG was removed at a faster rate. The mechanism by which this process takes place is not fully understood and our aim is to investigate the interactions of macrophages with different lipids using Brewster angle microscopy. Preliminary studies suggest that THP-1 differentiated macrophages do not significantly perturb DPPC and DPPG monolayers and research utilizing alveolar macrophages is underway. The effect of PS SP-A and SP-D is also discussed.

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