Postdoctoral Fellow Description
Zasadzinski Lab, Department of Chemical Engineering and Materials Science
University of Minnesota
Joseph A. Zasadzinski, PI
zasad008@umn.edu
612-626-2957

Brief Description of the Position:
The Zasadzinski group is looking to hire a postdoctoral fellow to work on an ongoing NIH sponsored project examining the effects of interfacial rheology on the development of acute respiratory distress syndrome (ARDS). ARDS is a pulmonary disease with a 40% mortality rate that effects over 300,000 people in the US each year. The underlying causes of the disease are not well understood, but we believe that ARDS may be explained by changes to the interfacial properties of the lung surfactant (LS) that occur during lung inflammation. The Laplace pressure difference, \( \Delta P = 2\gamma/R \), between the inside and outside of the curved, air-liquid interface in the alveoli suggests that the interconnected alveoli in the lung of different radii, \( R \), are at best metastable if the surface tension, \( \gamma \) is constant. Any variation in \( R \) would cause \( \Delta P \) to increase in the smaller alveolar and decrease in the larger alveoli, which is known as the “Laplace Instability.” However, healthy lung surfactant causes \( \gamma \) to decrease with decreasing alveolar interfacial area sufficiently to arrest the Laplace Instability. The variation of surface tension with interfacial area is known as the dynamic dilatational modulus, \( \varepsilon(\omega) = A(\omega)(\partial\gamma/\partial A) \), in which \( \omega \) is the breathing frequency. If the amount or quality of lung surfactant changes during disease, the dilatational modulus and surface tension can change, and if \( \partial(\gamma)/\partial R = (2\varepsilon - \gamma)/R^2 < 0 \) or \( (2\varepsilon - \gamma) < 0 \), the Laplace Instability will be triggered, which can cause the alveoli to collapse and fill with fluid, one of the symptoms of Acute Respiratory Distress Syndrome (ARDS). Hence, the dilatational modulus, and how it depends on monolayer composition, morphology, interfacial curvature, and the dynamic changes in lung area are essential factors controlling lung stability. Lysolipid concentrations, produced by degradation of bacterial and/or viral lipids by phospholipase A\(_2\) during the immune response, increase during lung inflammation. Preliminary work in the Zasadzinski group has shown that these lysolipids increase \( \gamma \) while simultaneously decreasing \( \varepsilon \). A unique custom built confocal microscope/capillary pressure microtensiometer is available in the Zasadzinski lab to study the interfacial dilatational and shear rheology of LS and develop structure-function relationships in LS monolayers by simultaneous imaging of the monolayer with confocal microscopy. We actively collaborate on interfacial adsorption dynamics with Dr. David Morse and Dr. Cari Dutcher at the U of M, Todd Squires at UCSB, and we have access to natural and synthetic lung surfactants and proteins prepared by Alan Waring at UCLA. We hope to elucidate the underlying cause of ARDS by visualizing the interface during bubble expansion and compression.

The position requires a Ph. D. in chemical or biochemical engineering, mechanical engineering, physics, biophysics, chemistry, or a related field. As a salaried Postdoctoral Associate, you qualify for health, dental and life insurance programs. This appointment provides vacation benefits as described here [https://policy.umn.edu/hr/academicvacation](https://policy.umn.edu/hr/academicvacation). You can review a summary of benefits available to you in this position at the following link: [https://hr.umn.edu/Benefits/UM-M-Employment-Benefits](https://hr.umn.edu/Benefits/UM-M-Employment-Benefits). Should you have questions about your benefits, please contact benefits@umn.edu or by calling 612-624-8647.