Acute lung injury (ALI) is a disease characterized by severe inflammation and pulmonary edema. There are no cures for ALI. The disease results from ligation of alveolar proinflammatory receptors (PIRs), such as TNFR1 and IL1βR expressed on the alveolar apical membrane. Since the F-actin fence impedes trafficking of these receptors to the cell membrane, our goal is to achieve intracellular delivery of our novel biologic consisting of the actin polymerizing agent, Rac1 in order to enhance F-actin and abrogate alveolar PIR expression. Our studies will entail live confocal microscopy of mouse lungs to determine dynamic quantifications of F-actin expression and the cytosolic Ca2+ in intact alveoli, as well as identification of Ca2+-related signaling pathways that impact actin enhancement. These studies will allow us to explore the efficacy of actin fence enhancement as novel therapy for ALI.