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A model for the volume regulatory mechanism of the Airway Surface Layer MICHAEL LANG, MICHAEL RUBINSTEIN, Department of Chemistry, University of North Carolina at Chapel Hill, N.C., C. WILLIAM DAVIS, ROBERT TARRAN, Department of Cell and Molecular Physiology, University of North Carolina at Chapel Hill, N.C., RICHARD BOUCHER, Department of Pulmonary and Critical Care Medicine, University of North Carolina at Chapel Hill, N.C., VIRTUAL LUNG PROJECT COLLABORATION — The airway surface layer (ASL) of a lung consists of two parts: a mucus layer with thickness of about 30 μm in contact with air and a periciliary layer (PCL) of about 7 μm below. Mucus collects dust and bacteria and is swept to throat by beating cilia, while riding on top of PCL. It is important that the thickness of PCL is matched with the length of cilia in order to optimize clearance of mucus. Decrease of PCL thickness would finally lead to an occlusion of the respiratory system. Experiments show that the height of PCL stays constant after removing mucus. When modifying height or composition of this open PCL by removing fluid or adding isotonic solution leads to the same final height of PCL. Thus, there must be a regulatory mechanism, that controls height, i.e. ASL volume. Additional experiments show that mechanical stimulus of the cells like shear leads to an increase of ASL volume, thus, the cell is able to actively adjust this volume. Based on these observations a class of models is introduced that describes the experiments and a specific minimum model for the given problem is proposed.

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