Membrane Fusion/Fission Yin-Yang in the Pulmonary Surfactant Complexes

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Formation and maintenance by the pulmonary surfactant system of surface active films at the respiratory surface is crucial to stabilize the lung against physical forces acting along the demanding breathing mechanics. For that purpose, surfactant requires the essential participation of small very hydrophobic proteins, SP-B and SP-C. These proteins are assembled by pneumocytes into tightly packed lipid-protein complexes that, once secreted, experiment remarkable structural transformations required for the homeostasis of the alveolar spaces. Lack or dysfunction of SP-B is incompatible with life. This protein assembles into supramolecular complexes able to promote the rapid flow of surface active species towards the interface, and at the same time, the formation of a highly cohesive multilayered structure providing maximal mechanical stability. These actions are associated with the ability of SP-B to promote membrane-membrane interactions and membrane fusion. Surfactant protein SP-C, on the other hand, considered the most hydrophobic protein in proteome, is an small palmitoylated transmembrane peptide producing deep perturbations into surfactant membranes, which end in their fragmentation to form small vesicles of 25-30 nm. These small vesicles are likely uptaken and recycled by pneumocytes and macrophages. The opposed actions of SP-B and SP-C towards membranes is mutually modulated, suggesting that their concerted action is a key feature to sustain pulmonary surfactant performance at the respiratory airspaces.