

147c Displacement of Fibrinogen from the Air/Aqueous Interface by Dilauroylphosphatidylcholine Lipid

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Fibrinogen (FB) and other serum proteins leak into the aqueous alveolar lining layer due to lung injuries. The adsorption of these serum proteins at the air/aqueous interface can produce higher surface tensions than the pulmonary lipids, and the Acute Respiratory Distress Syndrome (ARDS) can ensue. By having a molecular adsorption mechanism, as compared to a particulate adsorption mechanism of other longer chain lipids, dilauroylphosphatidylcholine (DLPC) lipid can expel FB from the air/aqueous interface at 25 °C, in water or in phosphate-buffered saline, as proven by tensiometry (also at 37 °C), ellipsometry, and infrared reflection–absorption spectroscopy. Moreover, before FB is displaced by DLPC at the interface, there is a substantial initial enhancement in the FB adsorption, consistent with some interaction or binding of DLPC with FB to produce a more hydrophobic protein surface. After the FB molecules have been displaced by DLPC, or when DLPC has already adsorbed at the interface, FB molecules are less favored to adsorb near the DLPC monolayer with the lecithin headgroups facing towards them. The results have implications for possible uses of DLPC lipid in potential lung surfactant formulations in treating patients with ARDS.