*COMPOSITION OF SURFACE-ACTIVE MATERIAL ISOLATED FROM BEEF LUNG**

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Observations by several investigators suggest that a protein-containing material which possesses unusual surface activity lines the internal surface of the lung[1, 2] and accounts to a large degree for the stability of its fine airspaces.[3] In a recent report, Pattle and Thomas[4] suggest that this surface-active material is a lecithin-protein complex; independently, we have obtained results which indicate that the complex contains these and additional substances.

As the initial step in isolating and purifying this substance, we used the method suggested by Bondurant.[5] Beef lungs were perfused with saline via the pulmonary artery and ventilated with intermittent positive pressure via the trachea. After a short time, a thick white foam poured out of the trachea. The foam was washed with distilled water to remove most of the serum proteins and then dried at 5°C. The dried powder was spread on isotonic saline in a modified Wilhelmy balance,[6] and surface tension was measured during compression and expansion of the surface film. This powder lowered the surface tension to less than 10 dynes/cm when surface area was decreased (Fig. 1, left). The surface tension-area diagram was similar to that seen with saline extracts of minced lung.[3]

Powders prepared from five beef lungs contained 50–70 per cent lipids and 5 per cent nitrogen. The lipids were extracted for 12 hours with equal parts of alcohol and acetone in a Soxhlet apparatus; when spread on isotonic saline and compressed on the balance, they failed to reduce surface tension below 20 dynes/cm. Separation of the major lipid fractions on silicic acid columns yielded about 74 per cent phospholipids, 8 per cent cholesterol, 10 per cent triglycerides, 8 per cent fatty acids, and essentially no cholesterol esters. Fractions containing cholesterol, triglycerides, and fatty acids did not have the unusual surface activity. The phospholipid fraction, however, reduced the surface tension as much as an extract of whole lung; surface tension decreased to 1 to 5 dynes/cm on compression of the surface, and hysteresis occurred on expansion of the film surface (Fig. 1, right).

The surface activity of the phospholipid fraction was inhibited by the other lipid fractions. It is possible that inhibition by certain lipids (or other compounds) may be the cause of the abnormally high surface tension of lung extracts which has been described in some pathological conditions.[7–9]

The surface tension-lowering activity of the phospholipid fraction was preserved under nitrogen for 4 hours but was lost gradually over 2 hours if the film-covered liquid was exposed to room air.

Three pure phospholipids—lysolecithin, sphingomyelin from red blood cells, and synthetic dipalmitoyl lecithin (obtained through the courtesy of Dr. Donald Hanahan of the University of Washington School of Medicine, Seattle)—gave surface tension-area diagrams closely resembling those obtained with whole lung extracts.

In view of available evidence, we suggest the following concept: The formation of a surface film, which is physically stable enough to give near zero surface tension on compression and chemically stable enough to resist oxygen at the interface,

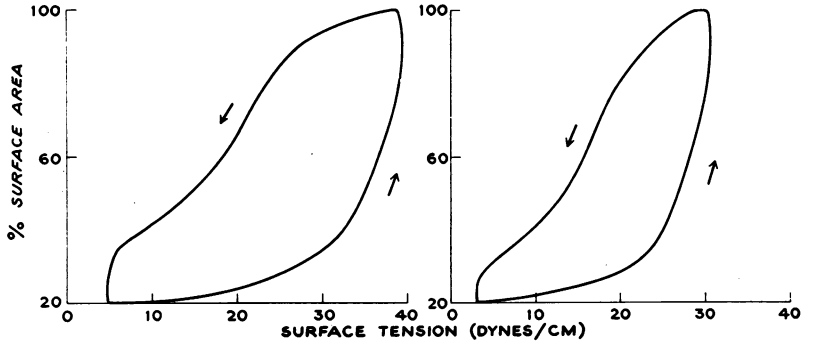

FIG. 1.—Surface tension-area diagrams measured on a modified Wilhelmy balance. Left, dried lung foam; right, lung phospholipids.

requires the high spreading pressure of phospholipids, a matrix of protein, and the antioxidant potential of substances as yet unidentified.

Summary.—Phospholipids isolated from fresh beef lung possess the unusual surface-active properties previously noted in crude lung extracts. However, this activity is gradually lost in air and can be inhibited by other lipid fractions. Certain purified phospholipids from other sources have similar activity.

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