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PERMEABILITY OF WATER VAPOR THROUGH DIPALMITOYL LECITHIN (DPL) MONOLAYERS. S.R. Turner*, W.S. Lynn*, M. Litt*, K.H. Kilburn, M.A. Passero*, D.J. Hurst* and H. Yasuda*. Duke University, Durham, N.C. and Research Triangle Institute, N.C.

DPL is the principal surface active lipid in lung alveoli. The observation that compressed monolayers of insoluble surfactants such as cetyl alcohol and palmitic acid retard the transport of water vapor, oxygen and CO₂ had led us to study whether DPL monolayers exhibit similar behavior. Our apparatus consists of a 5 x 10 cm enclosed teflon trough across which a low velocity stream of dry He is passed. A thermal conductivity detector measures water content of the exit gas, which is a function of evaporation rate. Monolayers spread on the trough can be compressed to 15% of the original area and re-expanded during permeability determination. Our results confirm that cetyl alcohol and palmitic acid monolayers retard water transport by a factor of two or more, when compressed to minimal possible surface tension of about 20 dyne/cm. On the other hand, DPL monolayers, compressed to zero dyne/cm surface tension exhibit no detectable difference in evaporation rate compared to a clean surface. DPL is thus unique among insoluble surfactants in attaining the highest possible surface pressure (zero tension) with no effect on water permeability, a combination of properties ideally suited to alveolar function.

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PROTEIN SYNTHESIS BY LUNG: INFLUENCE OF STARVATION ON AMINO ACID INCORPORATION INTO PROTEIN. K. Dickie* and D. Massaro. V.A. - G.W. University Medical Center, Washington, D.C., 20422

We studied the effect of food deprivation on the in-vitro synthesis by lung of total protein and of protein in a surface active fraction. Rabbits were either allowed food and water ad libitum (C) or deprived of food but allowed water for 72 hrs (FD) prior to sacrifice. Lung slices were incubated with leucine-U-¹⁴C at 40°. We measured radioactivity in crude protein and in the protein found in a surface active fraction obtained from lung. Free leucine was also measured in lung tissue.

We found that FD was associated with a marked decrease in leucine-¹⁴C incorporation into crude protein. This was not due to a difference in the free leucine content between C and FD rabbits. The decrease in leucine-¹⁴C incorporation into lung protein in food deprived rabbits was reversed 40 min. after the intraperitoneal administration of tryptophane. Leucine-¹⁴C incorporation into protein of a surface active lung fraction was also decreased. Food deprivation was associated with a fall in the ratio of lung RNA to DNA.

We conclude that FD decreases protein synthesis by lung and that this may be relevant to the effect of FD on lung mechanics and surface activity. (Supported in part by the American Thoracic Society).

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ISOLATION AND IDENTIFICATION OF THE PROTEINS FROM CANINE SURFACE ACTIVE MATERIAL. Richard J. King*, Daniel J. Klass*, Elias Gikas*, and John A. Clements*. Cardiovascular Research Institute, University of California, San Francisco, California, 94122.

We purify canine pulmonary surface active material (SAM) by centrifugation in density gradients (King and Clements, Fed. Proc. 29: 661, 1970), precipitate its proteins in ethanol/diethyl ether (1:3) at -15°C, and dissolve them in 0.1% SDS solution, pH 9.1. The proteins are resolved into three fractions by Sephadex G-200 gel filtration in 0.1% SDS; these are identified by immunochemical and electrophoretic techniques as principally IgG, albumin, and a non-plasma component. The third component contains phosphorus, and the protein to phosphorus ratio is 5.5. It has an electrophoretic mobility in polyacrylamide gels greater than the plasma proteins, and can be stained in these gels with both Coomassie blue and PAS. We estimate its molecular weight at 10-11,000 by gel electrophoresis in 0.1% SDS. This material gives a reaction of partial identity with antiserum directed against the non-serum components of SAM, showing that it contains one or more of the antigenic determinants of the material which we locate at the alveolar surface by immunofluorescence (Klass, King, Clements, Fed. Proc. 30: 619, 1971).

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INTERACTION OF HUMORAL AGENTS AND REGULATION OF AIRWAY SMOOTH MUSCLE RESPONSES (ASMR). James S. Douglas*, Richard B. Helgeson*, and Arend Bouhuys. John B. Pierce Foundation and Yale Univ. Lung Research Center, New Haven, Conn. 06510.

ASMR to aerosolized or intravenous histamine (H) in spontaneously breathing unanesthetized guinea pigs can be reduced by treatment with mepyramine, atropine, hexamethonium, bretylium and phentolamine. Guanethidine, dichloroisoproterenol, pronethanol and propranolol increase ASMR. These drug effects may be explained by implicating cholinergic and adrenergic reflexes but the assumption of different reflexes with mutually opposing actions is unattractive. A simpler explanation is that responses to exogenous stimuli are modified, at the cellular level, by endogenous neural and humoral agents. Such interaction has been demonstrated *in vitro* using an isolated superfused trachea (ST) with intact innervation. H (1 µg) and methacholine (1 µg) contracted the trachea giving a rise in intratracheal pressure. Superfusion of ST with theophylline (100 µg/ml) reduced these responses. When ST returned to baseline conditions after H (1 µg), responses to nerve stimulation or methacholine were enhanced for up to 35 minutes. Superfusion of ST with subthreshold doses of H caused a 100% increase in responses to methacholine or nerve stimulation. These data suggest that different humoral agents may interact at a common intracellular pathway thus modifying ASMR. One such possible site of interaction is the formation and metabolism of 3'5' cyclic AMP. Supported in part by USPHS HE-14534 and HE-14179.

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ALVEOLAR SURFACE AREA, SURFACE TENSION & COMPLIANCES OF ENZYME TREATED CAT LUNGS. M.J. Fisher* & K.C. Weber. ALFORD, NIOSH, USPHS, HEW & Dept. of Physiology & Biophysics, W.Va. U. Med. Ctr., Morgantown, W.Va. 26506 & Dept. of Anesthesiology, U. of Alabama Med. Sch., Birmingham, Alabama 35233

Cats were given single or multiple intratracheal injections of papain (3 to 30 mg. total) and/or phospholipase-C (0.2 to 3 mg total). The alveolar surface area and area surface tension relationships for the enzyme treated cat lungs (obtained from gas and liquid pressure-volume data one week to 4 months after the last injection) were not significantly different from those of the control cats, indicating the absence of any gross tissue destruction by the enzymes. This indicates the cat lung is highly resistant to papain induced emphysema in contradistinction to the rapid induction of emphysema in the rat lung by papain (Gross, P., Arch. Environ. Hlth: 50-58, 1965). This observation was supported by histological data from cat and rat lungs. Compliances of the papain treated cat lungs were increased during gas deflation at small lung volumes and decreased during gas and liquid deflations at large lung volumes. Phospholipase-C had only a slight effect on lung compliances. Lung radiographs revealed that within 1/2 hour after the intratracheal injections of papain, a pneumonic infiltrate was present within the terminal bronchioles and alveolar spaces indicating some lung changes. The lung radiographs returned to normal within 5 days. (Supported in part by Grants # NGR 47-001-048 NASA and # 70-AG-9C West Virginia Heart Association.)