A REPORT ON THE SURFACTANT SYSTEM OF THE LUNG

by

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I. INTRODUCTION

In the past 10-15 years, basic biological research has progressed greatly in defining the pulmonary surfactant system. The reason for specialized focus on this system is because it plays a particularly significant role in the survival of newborn infants. The lack of pulmonary surfactant is intimately linked to the respiratory distress syndrome (RDS) in infants which is also called the hyaline membrane disease (HMD). This disease appears to be the cause of 30% of all neonatal deaths and 50-70% of premature infant deaths (Crofton and Douglas, 1969). According to Northway and Daily (1972), 25% of "liveborn premature infants," and 1-2% of all newborn infants develop this disease. Because this disease accounts for such a large portion of infant deaths, scientists are involved in research which may allow better treatment and perhaps prevention of this disease.

The basic cause of this disease is not known. However, it appears as though the "maturity" of the pulmonary surfactant system at birth is an important factor in this disease. From the statistics cited above, it appears as if prematurity predisposes an infant to develop this disease. As will be shown in this report, in premature animals the surfactant system is not fully developed. However, treatment with corticosteroids appears to accelerate the development of the system and may prolong survival of animals (Avery, 1973).

Avery (1973) constructed a table from various sources showing things which are known for certain, or are probable, or are possible in relation to this disease. A few things which are known with certainty will be mentioned here. The disease occurs near the time of birth, and death or recovery occurs in 3-5 days. Reduced lung compliance (elasticity), low systemic blood pressure, reduced effective pulmonary blood flow, and cyanosis are additional characteristics. Pathological observations include regions of atelectasis (collapse) of alveoli, and formation of hyaline membranes. The hyaline membrane lines the alveoli and is made up of "sloughed cell debris in a protein matrix" (Crofton and Douglas, 1969). These membranes contain fibrins (Avery, 1973). The etiology of the disease is definitely linked to surfactant deficiency (Avery, 1973). Evidence for this comes from the "effectiveness of continuous distending airway pressure" (Avery, 1973). These and other clinical and pathological symptoms are characteristic of this disease. The general consensus appears to be that alveolar collapse due to a lack of pulmonary surfactant is a primary factor of the disease. Bates et al. (1971) (citing various sources) gave possible causes of this disease which include "asphyxia, hypoperfusion of the pulmonary vasculature, or a fibrinolytic-enzyme defect."

This report will focus on the pulmonary surfactant system itself. Four basic topics will be covered. The first topic will mainly define the system which includes the

composition, structure, and synthesis of surfactant. Secondly, the discussion will center on the type II cell and model systems. The third topic will deal with varicus treatments of animals and/or lung tissues in order to determine what agents or conditions might control surfactant synthesis and secretion. A discussion of proposed research will be the fourth topic. Due to the rather broad scope of this paper, an attempt will be made to cover significant points about this subject. The reader should keep in mind that this report, as with any publication, is limited by the author's biases toward certain information and is limited to this author's knowledge of the field. Even though some portions of this subject are covered superficially, the report should provide a thorough background for anyone interested in studying the pulmonary surfactant system.