Genetic Relationship between Maximum Hematocrit Values and Hemoglobin Type in Sheep

Abstract. *A significant association between seasonal maximum hematocrit values and hemoglobin type has been shown to exist in sheep. Both characters are known to be under genetic control.*

Recently (*1*, *2*) it was shown that seasonal maximum hematocrit values in normal sheep are under genetic control. In other studies it was demonstrated that hematocrit values, suitably corrected for body weight, are a function of the total volume of circulating red blood cells (*3*). It was also demonstrated that the total volume of the red blood cells is an important factor influencing the outcome of experimental epidemics of disease due to a blood-sucking parasite (*4*).

Hemoglobin type in sheep in the AB system is simply inherited as an allelic pair (*5*). A number of observations suggest that the different gene frequencies in various populations of sheep are the result of selection for one or the other hemoglobin type (*6*). There is evidence that hemoglobin type is correlated with hematocrit value (*7*, *8*) and with blood volume (*8*).

Our purpose here is to report on the classification of the sheep in the flock maintained for parasitological investigations at the New York State Veterinary College with respect both to the best estimate of their hematocrit values (*3*) and to their hemoglobin types. There were 124 ewes, 26 rams, 23 yearlings, and 64 lambs (born in 1963) available for this classification.

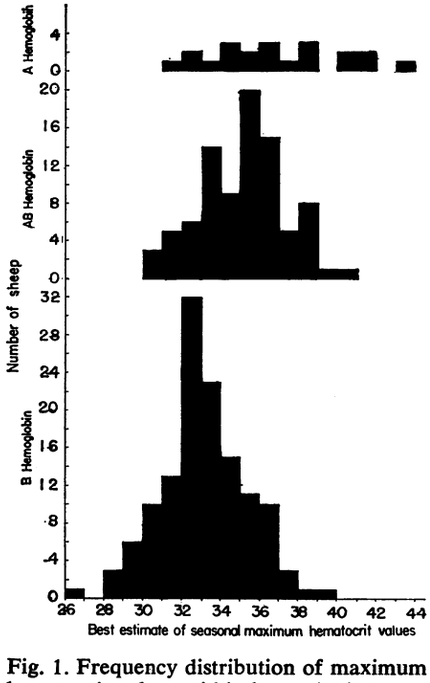

Fig. 1. Frequency distribution of maximum hematocrit values within hemoglobin types.

Figure 1 shows that there is a close relationship between hemoglobin types (*5*) and the best estimates of maximum hematocrit values (*2*). The mean maximum hematocrit values for each hemoglobin type form a series where A is greater than AB, which is greater than B. The differences between these values is highly significant (*9*).

It is therefore of interest that, other things being equal, an animal with a low erythrocyte volume in this flock has a smaller chance of surviving a natural challenge with *Haemonchus contortus* than an animal having a greater erythrocyte volume (*4*), and that animals with hemoglobin A in Australia may show lower fecal egg counts than animals with hemoglobin AB (*10*).

Although no direct experimental data are available, observations on the Veterinary College flock have suggested that low maximum hematocrit values may be associated with mortality subsequent to winter shearing. It is known that sheep breeds indigenous to cold environments tend to have high gene frequencies for hemoglobin A (*5*).

These observations demonstrate that two genetically controlled characters, a phenotype boundary (maximum hematocrit) and a genetic polymorphism (hemoglobin type), are significantly associated in sheep. They may be important in relation to the reaction of an animal to its environment.

J. V. EVANS*
J. H. WHITLOCK
New York State Veterinary College, Cornell University, Ithaca, New York

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* On leave from the Department of Physiology, Univ. of New England, N.S.W., Australia.

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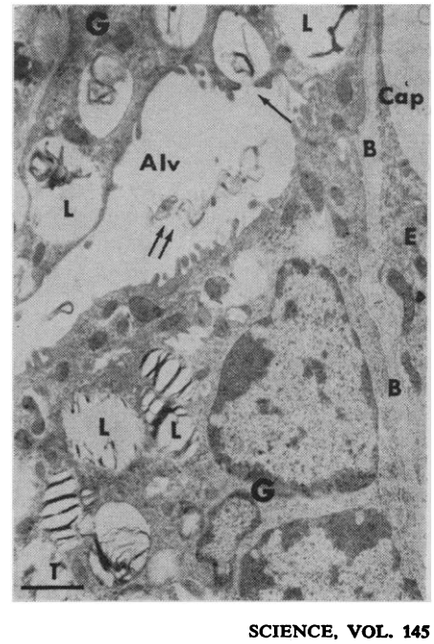
Granular Pneumocytes: Electron Microscopic Evidence of Their Exocrinic Function

Abstract. *The contents of the lamellar bodies of granular pneumocytes are normally released into the alveolar lumen. Exposure of guinea pigs to an atmosphere containing carbon dioxide causes the formation of abnormal lamellar bodies and a significant increase in the pulmonary surface tension. The eventual return to a normal pulmonary surface tension coincides with the formation of normal lamellar bodies.*

The discovery of the surface active agent of the lung (*1*) has prompted several investigators to study the chemical composition and origin of this substance. There appears to remain little doubt about the lipoproteinaceous nature of the surfactant (*2*). The evidence for the cellular site of production or assembly of this substance, or both, is circumstantial, yet it suggests strongly a certain type of transformed mito-



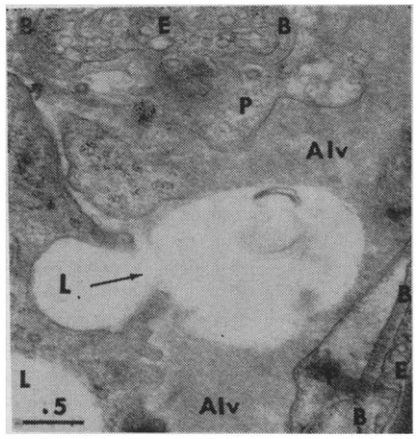


Fig. 2. Lung section of guinea pig exposed to 15 percent CO_2 for 1 day so that it developed uncompensated respiratory acidosis (blood *p*H 7.08). Alveolar lumen (*Alv*) filled with edema fluid, swollen alveolar lining cell (*P*), basement membrane (*B*), and adjacent endothelial cell (*E*). Electron translucent contents of altered lamellar body (*L*) are being discharged (arrow) into the alveolar lumen filled with edema fluid. × 21,000.

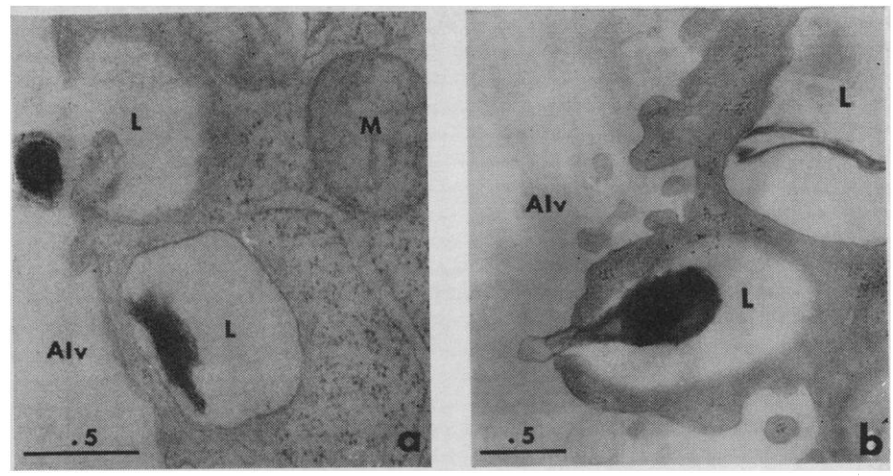


Fig. 3. Lung section of guinea pig exposed to 15 percent CO_2 for 14 days showing recovery-compensated respiratory acidosis (blood *p*H, 7.37). Near normal lamellar bodies (*L*) in the process of discharging their electron opaque contents into the alveolar lumen (*Alv*); mitochondrion (*M*). (*a*) × 21,000; (*b*) × 30,000.

chondrion usually called the lamellar body (mitochondrial transformation, plasmosome) of the granular pneumocyte (large alveolar lining cell, alveolar epithelial cell) (*3*, *4*). But direct evidence for the discharge of these lamellar bodies from the granular pneumocytes into the alveolar space is still lacking, as pointed out recently by Klaus *et al.* (*4*) and Pattle (*5*). In this report we present evidence for the exocrinic function of these pneumocytes, as observed with the electron microscope.

We recently obtained evidence that the lamellar bodies of granular pneumocytes are the site of origin of the surfactant. (i) It was shown that in CO_2-induced hyaline membrane disease in guinea pigs (*6*) the time course of changes of pulmonary surface tension and lamellar bodies paralleled each other (*7*).

During the development of hyaline membranes, which was limited to the uncompensated phase of respiratory acidosis, the average minimum surface tension rose from a normal of 5.0 dyne/cm to 21.0 dyne/cm and normal lamellar bodies disappeared. (ii) Electron microscopic observations showed the discharge of the lipoproteinaceous (electron opaque) contents of lamellar bodies from the granular pneumocyte into the alveolar space as well as the discharge of the abnormal electron translucent contents of pathologic lamellar bodies in carbon dioxide-induced hyaline membrane disease.

Figures 1 to 3 show electron micrographs of lung sections from guinea pigs. Although the direction of movement of the contents of the lamellar bodies could be in question in Figs. 1 and 3, which represent the normal state and recovery state of carbon dioxide intoxication, there can be no doubt that the pathological contents of the lamellar body in Fig. 2 is being discharged from within the granular pneumocyte into the alveolar space filled with edema fluid.

The minimal surface tension of the lung from which the picture in Fig. 1 was obtained was 5 dyne/cm; it was 21 dyne/cm in the lung shown in Fig. 2, and 8 dyne/cm in Fig. 3.

Histochemical studies for acid phosphatase activity, after incubation of lung slices in a modified Gomori medium (*8*), did not reveal reaction products in the lamellar bodies when observed with the light and electron microscope. The observed release process of lamellar bodies can thus not be considered to be of lysosomal origin.

These findings provide evidence for the secretion of a lipid-rich (electron opaque) substance from the granular pneumocyte into the alveolar lumen. In the uncompensated phase of carbon dioxide intoxication, the contents of the lamellar bodies are changed into a very electron-translucent material which is associated with a rise in minimal surface tension indicating a lack of surfactant. These observations, particularly the return to normal surface tension and normal appearance of electron opaque lamellar bodies during the compensated phase of CO_2-induced respiratory acidosis, lend further support for the identification of the lamellar bodies as the source of the pulmonary surfactant.

Fig. 1 (left). Section of lung of control guinea pig (blood *p*H 7.41). Niche of an alveolar lumen (*Alv*) between two adjacent granular pneumocytes (*G*) which are attached to basement membrane (*B*), Part of capillary lumen (*Cap*) and endothelial cell (*E*) in right margin. Numerous electron opaque lamellar bodies (*L*) are present, the lumen of one is in direct communication (arrow) with the alveolar lumen (*Alv*). Lamellar electron opaque material in the alveolar lumen (double arrows). × 8000

K. BENSCH
Department of Pathology,
Yale School of Medicine,
New Haven, Connecticut
K. SCHAEFER
Physiology Division, U.S.
Navy Medical Research Laboratory,
New London, Connecticut
M. E. AVERY
Department of Pediatrics,
Johns Hopkins Medical School,
Baltimore, Maryland

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