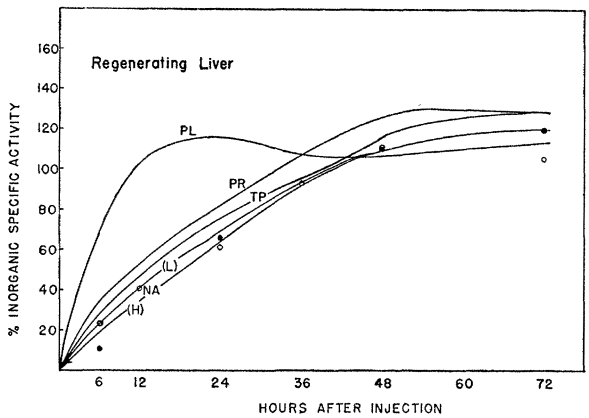

FIG. 2. Regenerating rat liver. Specific activities of phosphorus fractions at various time intervals after P^{32} injection, expressed as per cent. of the liver inorganic phosphorus specific activity. The symbols are the same as in Fig. 1.

of magnitude which can be accounted for by the synthesis of nucleic acid in the formation of new cells. The "protein residue" here apparently has the same rate of turnover as in resting liver, indicating little if any relation to growth. The increased rate of turnover in the "total protein" is therefore due wholly to the higher uptake by its nucleic acid component, confirming Marshak's interpretation on nuclei.[2]

Our results serve to point out the discrepancies between the results of Hahn and Hevesy[1] on nucleic acid and those of Marshak,[2] Tuttle *et al.*[3] and Kohman and Rusch.[6] We have confirmed the observation of the former workers that the turnover of nucleic acid in non-growing liver is very slow. The higher turnover rates found by analyses of "total protein"[3,6] are not necessarily representative of the nucleic acid portion alone. The relatively higher turnover found by Marshak[2] in the nuclei of resting liver cells probably depends upon fractions of nuclear phosphorus other than nucleic acid phosphorus. Our methods do not distinguish cytoplasmic from nuclear nucleic acids, and some evidence has been obtained that the former have a higher rate of phosphorus turnover than the latter.[9] Thus the resting turnover of nuclear nucleic acids may be even lower than shown by our figures.

The present results lend chemical confirmation to the belief that the nucleus is the stable element in the cell, and point to the nucleic acid component as a compound ensuring this stability. Changes in the more labile compounds within the cell can readily occur through shifts in the steady state, in which rapid synthesis and degradation of these compounds are balanced. In the case of nucleic acids, such continuous turnover occurs very slowly, while synthesis takes place rapidly during growth. This distinguishing characteristic of nucleic acids may be of great importance for the mechanism of growth.

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[9] A. M. Brues, M. M. Tracy and W. E. Cohn, unpublished data.

ISOLATION OF A FILTERABLE VIRUS FROM CHICKENS AFFECTED WITH "BLUE COMB" DISEASE

FOR the past several years poultry pathologists throughout the Northeast have repeatedly encountered a disease entity in domestic chickens, and to a lesser extent turkeys, of unknown origin. It is referred to as blue comb, pullet disease, "X" disease and several other such terms referring to the symptoms exhibited or the conditions or time of encounter. Jungherr and Levine[1] have given a detailed description of the gross and microscopic lesions of the disease as well as their observations on its epidemiology and mortality rate. All attempts, with the possible exception of one by Bullis,[2] to transmit this condition from one bird to another of similar age have failed.

At the University of New Hampshire Agricultural Experiment Station in September, 1941, the writer had the opportunity of observing the course of the malady in two flocks in which the attack was extremely acute and severe. Many of the birds were found dead without having been observed sick; others died after an illness of only a few hours. From the blood stream of such acutely affected birds from both flocks we have been able to obtain a filterable agent that grows readily on the chorio-allantoic membrane of chick embryos. One strain has been carried through 56 transfers made at 72- or 96-hour intervals and the other strain through 39 such transfers. A third strain obtained from the eggs of an infected flock is now in its seventh transfer.

When the infected chorio-allantoic membrane, embryo or embryonic fluid are injected into susceptible chickens they, after 84 to 96 hours, become somewhat depressed and cyanotic. Death has not been produced by such an inoculation. If the inoculated birds are sacrificed at the end of 96 or 120 hours the following gross lesions may be observed; subcutaneous edema, generalized icterus, hemorrhages into the skeletal muscles, marked congestion and swelling of liver and kidneys, collection of urates in ureters, petechia-

[1] Erwin Jungherr and J. L. Levine, *Am. Jour. Veterinary Research*, 2: 4, 261–271, 1941.
[2] K. L. Bullis, personal communication, 1941.

tion of heart and serus coat of duodenum, sub-periosteal hemorrhage of flat bones, hemorrhage into the lungs and an acute catarrhal or hemorrhagic duodenitis. Various combinations of these lesions will be encountered. If the bird is not sacrificed the lesions will tend to disappear by the seventh day after inoculation. A microscopic examination of the liver of birds sacrificed at the end of 96 hours reveals foci of cloudy swelling. These swollen liver cells also reveal contracted pyknotic nuclei. The kidney changes are more pronounced but are also limited to foci. The epithelial cells lining the tubules show stages of degeneration varying from swelling with contracted pyknotic nuclei to actual destruction of the cells. The glomeruli are markedly swollen and contain few erythrocytes. There is round cell infiltration between the tubules in these foci.

A suspension of the feces and intestinal contents of experimentally infected birds is infective to other birds of the same age when given by way of the mouth. A bacterial free filtrate of the feces is infective when injected intraperitoneally. Bacterial free fecal filtrates are also capable of establishing the virus in incubating chick embryos. The virus is readily filterable through either or both the Seitz E-K 3 and the Chamberland-Pasteur L-3 filter.

Since this filterable virus alone does not produce death when injected into birds of a susceptible age we can not, at this time, say with any certainty that it is the sole etiological agent of the so-called "blue comb" disease.

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SCIENTIFIC APPARATUS AND LABORATORY METHODS

A SIMPLE ULTRACENTRIFUGE WITH PLASTIC ROTOR[1]

THE ultracentrifuge constructions presently in use are based either on the principle of the oil-turbine velocity centrifuge, equipped with mechanical bearings and a vertical steel rotor, or on the principle of the bearing-less "spinning top," which represents an air turbine floating on a cushion of compressed air. The analytical centrifuges of McBain and the concentration and optical centrifuges of Beams, Wyckoff and Pickels and Bauer are derived from the latter type. The most frequently employed rotor material in these constructions is duralumin.

During a recent visit to the Svedberg centrifuge laboratory of Professor J. W. Williams,[2] at the University of Wisconsin, it occurred to the writer that the use of materials of very low density for the construction of ultracentrifuge rotors might result in a considerable simplification of centrifuge design and obviate the necessity of employing expensive steel and aluminum alloys which are now difficult to procure on account of the National War effort. The first trials were made in Madison with 0.5 inches thick discs of polystyrene and of polyacrylic, transparent resins of 1.5 and 2 inches diameter, respectively. With the mechanical assistance of Messrs. E. Hanson and L. Henke these discs were transformed into simple air-turbines. Employing a 2-inch Lucite disc, tank nitrogen as propellant and a primitive optical set-up with a spectacle lens as objective, the sedimentation of aggregated earthworm hemoglobin within the cylindrical fluid cell was photographed with the kind help of Mr. Ch. Vilbrandt. Since the Lucite turbine showed no signs of irreversible deformation, even when spun for 10 minutes at approximately 40,000 r.p.m., the writer felt encouraged to continue these experiments with plastic rotors after his return to New Haven. A 2-inch Lucite turbine was accelerated to 57,000 r.p.m. with the aid of 80 pounds air-pressure per square inch, as measured with the Kahler-Hunt photoelectric speed-measuring circuit. After the mechanical features of the centrifuge had been improved in various respects, the construction of a 6-inch plastic turbine was undertaken with the mechanical aid of Mr. H. Nelson. Throughout the later phase of this work much benefit was derived from the expert advice of Professor F. W. Keator, of the Department of Mechanical Engineering, and several improvements, based on his suggestions, were incorporated in the design.

A schematic drawing of the centrifuge at its present stage of development is reproduced in Fig. 1.

The top speed, thus far attained with this model, has been 17,400 r.p.m. at 48 lbs/sq. inch air-pressure and an estimated free air flow of 40 to 60 cubic feet per minute, yielding a force of 20,200 times gravity at the center of the analytical fluid cell which is situated at a distance of 6 cm from the center of rotation. This speed is sufficient to cover practically the entire size range of plant and animal viruses as given by Stanley,[3] and, in general, to bring about molecular sedimentation, at appreciable rate, of protein particles from about 10^6 molecular weight upwards. As examples of such materials, the sedimentation of earthworm hemoglobin and of Stanley's crystalline

[1] This work was aided by a grant from The Jane Coffin Childs Memorial Fund for Medical Research.

[2] The writer wishes to thank Professor J. W. Williams for his hospitality and his encouraging interest in this work.

[3] W. M. Stanley, in Handb. d. Virusforschg., p. 538. Wien, 1938.