(Dudley[3] states that only a minute amount of the active principle of insulin passes through a collodin sac), the ends of the primary roots were cut off in one series of seedlings, so that the solutions might enter the plants directly through the vascular bundles. Seedlings treated in this fashion made better growth in the stronger solutions of insulin and glucokinin than the seedlings with uncut roots. In this connection the growth of green seedlings from which the endosperm had been removed was followed. Seedlings with the endosperm removed, when grown in distilled water under the same conditions as treated plants, showed retarded growth and produced root systems strikingly similar to those of seedlings with the endosperm intact but grown in strong insulin or glucokinin solutions. These tests collectively suggest a comparison with the effects of insulin on normal and diabetic animals. Banting and collaborators[4] have shown that it is possible to lower the blood sugar in normal animals by doses of insulin to a point where the animal dies in apparently the same condition as an animal dying from hypoglycemia following the removal of the liver. The beneficial effects of small quantities of insulin given to diabetic animals have been observed by many experimenters. It seems possible, therefore, that the retarded growth of the seedlings in strong solutions of insulin or glucokinin was produced by an action of these substances which rendered the carbohydrates of the endosperm unavailable. Since plants with cut roots made better growth in strong solutions than plants with uncut roots the experiments suggest that the retardation of growth is produced by a fraction which is easily absorbed through the uninjured root cells, and that the increased growth of plants with cut roots was dependent upon the presence of a fraction not readily absorbed by uninjured roots.

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"VITAMIN A" DEFICIENCY IN POULTRY

THE occurrence of a destructive disease of poultry, resembling an infection in its manifestations, but thought and now known to be caused by incorrect feeding methods, was discussed in the report of the California Agricultural Experiment Station[1] for 1919–20. The opinion in regard to the etiology of the disease was based upon (1) negative results of bacteriological examinations and failure to transmit the disease to healthy fowls by inoculation, and (2) on success in controlling the disease in several flocks by increasing the amount of green food in the ration, decreasing the proportion of meat scrap and eliminating cocoanut meal.

The disease is characterized by (1) a discharge from the nostrils; (2) an ophthalmia producing a viscid secretion which glues the eyelids together, followed by the formation of a tightly adherent white film over the membrana nictitans and the accumulation of a mass of white caseous material in the conjunctival sacs; (3) the appearance of white pustule-like lesions one half to 2 mm in diameter on the mucosa of the mouth, pharynx and esophagus; and (4) in the later stages, weakness and emaciation. These symptoms have often caused the disease to be diagnosed as a form of avian diphtheria or roup by veterinarians and poultrymen. Besides the above lesions, the most prominent changes found on autopsy are pale, swollen kidneys marked by a network of very fine white lines which are urate-filled tubules.

The results of controlled feeding experiments conducted by the writer in 1920[2] suggested that the disease was caused by a deficiency of some vitamin in the ration and experiments just completed have shown this to be the case. In these experiments eleven pens of fifteen fowls each were fed a basal ration of mixed grains and meat scrap properly balanced for poultry but containing no yellow corn. No other food was given in pen I, the control. In pen II the basal ration was supplemented by a salts mixture consisting of calcium carbonate, calcium phosphate, sodium chloride, sodium sulphate and iron sulphate; in pen III by buttermilk; in pen IV by cod-liver oil; in pen V by dried yeast; in pen VI by orange juice; in pen VII by cod-liver oil and dried yeast; in pen VIII by cod-liver oil and orange juice; in pen IX by dried yeast and orange juice; in pen X by cod-liver oil, dried yeast and orange juice; and in pen XI by lawn clippings.

None of the fowls in the pens which have received cod-liver oil or lawn clippings have been affected, but the disease has occurred in all other pens, affecting 11 of the 15 fowls in pen II, which were fed buttermilk, and all the fowls in the other pens.

These studies throw light on the etiology of a serious poultry disease which the writer has previously designated as "a nutritional disease resembling roup." We feel that we are now justified in using the more definite designation "Vitamin A deficiency," for this disease, although the term "nutritional roup" might be more suitable for general use among poultrymen.

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[3] *Biochem. Jour.*, XVII, 376–390, 1923.

[4] *Amer. Jour. Physiol.*, LXII, 162–176, 1922.

[1] Report of the California Agricultural Experiment Station, 1919–20, p. 79.

[2] Report of the California Agricultural Experiment Station, 1920–21, p. 140.