One point is not yet clear; that is the action of alkali. Treatment with 0.1 N alkali at 100° C. destroys the activity in a few minutes. No phosphate is liberated; but there occurs a stabilization of the glucosidic linkage so that the glucose now undergoes hydrolysis at the same rate as the first phosphate group. Presumably alkali brings about a rearrangement of the molecule.

The mechanism by which uridine diphosphate accelerates the conversion of galactose into glucose will require further investigation. The fact that glucose phosphate can be recognized as a part of the coenzyme suggests that this portion may exchange with the substrate during the reaction cycle.

An interesting fact is the similarity of uridine diphosphate with the compound which Park and Johnson⁶ have found to accumulate in *Staphylococcus aureus* grown in the presence of penicillin. It is not possible from their data to conclude that it is the same compound; but the resemblance is striking.

A full account of this investigation will be published elsewhere.

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Aphosphorosis in Ireland

FOLLOWING Colonel Doherty's observation¹ that 'bog crook', a chronic disease of cattle which has been indigenous in certain districts of Ireland for many generations, was clinically similar to stywesiekte, Sheehy², in a series of controlled feeding experiments, showed that the feeding of phosphatic supplements was effective in the cure of this condition and concluded that it was aphosphorosis.

Several hundred determinations of blood inorganic phosphorus and of serum phosphatase have been carried out in this Laboratory on samples both from clinical cases of 'bog crook' and from apparently normal animals grazing on the same farms. Analyses of the herbage from the appropriate pastures have also been undertaken.

The results of this work show that a sub-clinical or incipient form as well as a clinical form of aphosphorosis exists, and that both forms are widespread in districts where there is a low phosphorus content in the pasture. In both the clinical and sub-clinical forms of this condition, there is a low level of inorganic phosphorus in the blood; only in the clinical form is there a significant rise in serum phosphatase. On phosphorus-deficient pastures both forms often co-exist, but on such pasture the sub-clinical form is always present and in many cases may be detected only by blood analysis. Sub-clinical aphosphorosis is often associated with unthriftiness, low milk-yield in dairy cows and anestrus or oestrus with repeated failures to conceive after service. Of the clinical cases, first-calf heifers usually exhibit the severest symptoms. Mature cows and calves are not so seriously affected.

Experiments show that a change of cattle from phosphorus-deficient to phosphorus-sufficient pasture is attended by a more spectacular rise in blood inorganic phosphorus than takes place if the animals are maintained on phosphorus-deficient pasture and fed a phosphatic supplement.

It has been found that where the blood inorganic phosphorus values of members of a grazing herd are of the order of 1.0–2.0 mgm. per cent the pasture herbage contains less than 0.35 per cent P₂O₅ dry matter. Provided that cattle are not receiving supplementary feeding in addition to pasture grass, the simplest method of establishing phosphorus-deficiency in pasture is by blood analyses of the cattle grazing thereon.

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Copper-Deficiency Disease of Pear Trees

COPPER has long been considered as one of the essential trace elements connected with satisfactory growth and development of most higher plants¹, and copper-deficiency in various top fruits has been established by workers in the United States, Australia, New Zealand and South Africa. The affected trees show varying amounts of leaf chlorosis, followed by the death of the terminal bud and the dieback of the vigorously growing shoots.

In September 1948, during the course of my duties as an advisory officer, my attention was directed to a very severe case of dieback of terminal shoots in vigorously growing young nursery pears. The trouble was immediately suspected to be copper deficiency, as the symptoms so closely resembled the description of the disease by workers in other countries²⁻⁴.

During the winter season all the dead wood was pruned to induce new growth the following season. In May 1949, when the trees had made some shoot growth, and before any dieback appeared, twelve trees were sprayed with 0.1 per cent copper sulphate solution. The treated trees, by the end of August, made more than 3 ft. of healthy new wood and showed no symptoms of the disease, while the untreated trees made very slow growth and developed severe dieback in early August. The copper content of the leaves from the untreated trees was less than half that of the treated trees.

I believe this to be the first case of copper deficiency of fruit trees and its control by copper sulphate spray recorded in the British Isles. Other troubles peculiar to this nursery are suspected of being connected with the same deficiency, and work is in hand on these problems.

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