Effects of Calcium Deficiency on Potato Sets in Acid Soils

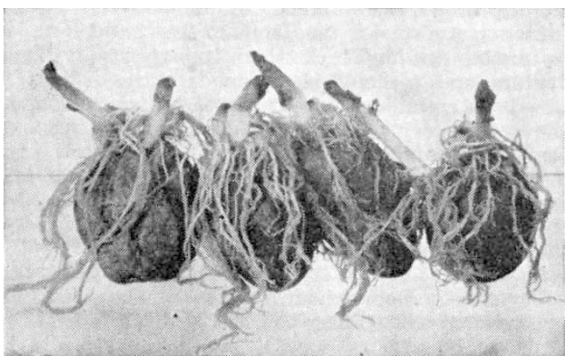
During the War, when many poor upland areas and commons in Britain were ploughed up for crop production, potatoes were commonly planted as a first crop in view of their tolerance to acid soil conditions. Many failures, however, were experienced where the soils were very strongly acid, with *p*H values of the order of 4·0, and it was shown that liming was necessary to correct the unfavourable conditions. Plants which survived on these acid soils usually showed pronounced symptoms of calcium deficiency in the haulms, and it was clear that the supply of this element was an important factor in determining success or failure of the crops.

A second point which was noted where the shoots failed to emerge, and which has so far remained unexplained, was that the planted tubers remained firm and apparently failed to make any shoot growth. This phenomenon appeared unusual, since tubers will sprout and soften normally in the absence of any soil, and seemed to suggest an inhibiting effect on shoot-growth, such as might result from toxic substances in the soil, as, for example, excess of hydrogen ions, manganese, aluminium or ammonium.

In the course of sand-culture experiments carried out at Long Ashton, in which the effects of possible soil-acidity factors have been examined on a number of crop plants[1,2], observations have been made on potatoes which indicate that the effect is a direct result of calcium deficiency and is not due to the toxic action of elements present in excess.

Sprouted potato sets, variety Majestic, were grown in sand, with and without calcium, both pots and sand being specially treated to ensure very acute conditions of calcium deficiency where the element was omitted from the nutrient solutions. Differential nitrogen treatments, including nitrate, ammonia and urea, were given, and some cultures also received concentrations of manganese and aluminium sufficiently high to produce severe toxic effects. Two series of parallel nutrient solutions, with *p*H values of 3·5 and 5·5, were used.

In the cultures from which calcium was omitted, the plants failed completely and the shoots did not emerge above the level of the sand. On examining the tubers of these plants, it was found that the sprouts had broken down immediately behind the growing points, after which they died off. The roots, in contrast, appeared fairly normal and the tubers remained as firm as when they were planted (see accompanying illustration).



Sprouted sets, showing death of growing points resulting from calcium-deficiency treatment in sand cultures

The affected tubers were afterwards thoroughly washed; then they again sprouted normally in boxes without sand or soil.

In all cultures to which calcium was given the shoots emerged normally, and it was not until a later stage of growth that signs of the other unfavourable treatments were developed.

It thus seems that the hard-tuber condition which occurs in failure-plants on acid soils results from the dying back of the young shoots prior to emergence above the soil, and that this is caused by deficiency of calcium and is not due to the effects of toxic concentrations of elements such as manganese and aluminium.

T. Wallace
E. J. Hewitt

Long Ashton Research Station,
University of Bristol. Sept. 13.

[1] Wallace, T., Hewitt, E. J., and Nicholas, D. J. D., *Nature*, **156**, 778 (1945).
[2] Hewitt, E. J., Ann. Rept. Long Ashton Res. Stat. 51 (1945): 50 (1946).

Transmission of a Disease Resembling Virus Yellows through the 'Seed' of Sugar Beet

In the course of breeding experiments, Mr. B. Crombie of the Irish Sugar Co. observed in 1946 that three separate field crops of a new family of sugar beet showed in each case approximately 25 per cent of 'yellowed' plants two to three weeks after singling. He stated that the disease resembled typical virus yellows but did not appear to spread to neighbouring beet crops. The parent plants of the family (No. 41) had been selected from good commercial beet crops in a relatively virus-free district, but no information was available as to their freedom or otherwise from yellows. The cross had been made in 1945 in a pollen-proof cage.

On September 7, 1946, the Sugar Co. transmitted to this laboratory field-plants of family 41 showing yellows, together with a small quantity of the residual 'seed' of this family. (The term 'seed' is used in the popular sense and refers to the fruit clusters.) A portion of the latter was sown in 1946, and the remainder in March 1947, in an insect-proof glasshouse, the seedlings being transplanted to 7-in. pots when of appropriate size. No abnormality was noted until the plants were about 10 in. in height, with ten to twelve foliage leaves, when the tips of the two basal leaves of certain plants became tough, then changed from the normal colour to light green and, gradually, through yellow to orange. As a general rule, the yellowing afterwards spread downwards through the leaves, only the areas around the veins remaining green. Similar symptoms meanwhile developed in the next leaves above, the advance of the disease being thus acropetal. No vein-clearing or other abnormality was detected in the young developing foliage. In some of the affected leaves the yellowing remained confined to the tips with, occasionally, an island of affected tissue midway on the lamina; in others it spread irregularly down one side. Frequently a yellow area was clearly demarcated from a green one by a main vein, this being more noticeable as the plants became older. Eventually the chlorotic areas withered and turned brown and the margins of the leaves curled upwards. Of the 143 plants which were raised from seed, 47·5 per cent showed the foregoing symptoms, which, in general, resemble those of virus yellows as described by Watson[1].