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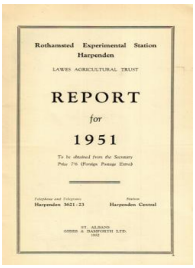

Beet Yellows Virus and Other Yellowing Virus Diseases of Sugar Beet

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BEET YELLOWS VIRUS AND OTHER YELLOWING VIRUS DISEASES OF SUGAR BEET

By M. A. Watson

Introduction

Healthy sugar beet remain green until harvest. If they become yellowed it is because of deficiency of mineral nutrients, or infection by fungus, virus, or other pathogens (Hale, Watson and Hull, 1946). The most important cause of yellowing is beet yellows virus (Watson, 1940). Every year it causes serious losses of sugar in Europe, and in some years, when in large areas every plant may become infected, the losses amount to a considerable proportion of the potential yield.

Until recently the disease was thought to occur only in Europe, but yellowing diseases of sugar beet have now been reported from Australia (Stubbs, 1949) and from the United States of America by Dr. Hull on his recent visit. Whether these diseases are identical with that caused by beet yellows virus in Europe is still undetermined, but one of the purposes of this article is to suggest that there is a range of viruses, not all of them closely related to one another, causing yellowing symptoms in beet.

Beet yellows virus in the field

The disease now known to be caused by beet yellows virus (S.B.Y.), was first described in Europe as " Jaunisse ". Quanjer, in 1934, suspected it to be a virus disease. This was confirmed by Van Schreven (1936), and Roland (1936), who showed that it was transmitted by the green peach aphid, *Myzus persicae* and the black bean aphid, *Aphis fabae*. It was first identified in England in 1938 in plants from a small field experiment at Rothamsted (Watson, 1940). In the same year the virus was obtained from the Rothamsted and Woburn farms, and from other parts of England. There is no doubt that at this time it was common throughout the sugar beet growing districts of England. It had been described by Petherbridge and Stirrup (1935) under the name of " crackly yellows " and attributed by them to physiological causes.

Aphis fabae is usually much more numerous than *Myzus persicae* in sugar beet root crops and it was at first thought that this aphid was mainly responsible for spreading beet yellows virus (Watson, 1942), but field observations started in 1940, soon showed *A. fabae* to be of little importance compared with *M. persicae* (Watson, and Hull, 1946 ; Watson, Hull, Blencowe and Hamlyn, 1951).

Field experiments made between 1940 and 1943 demonstrated that serious losses could be caused by the disease, for early infection reduced sugar yield by more than half (Watson, Watson and Hull, 1946). Also the nutritional status of crops had little effect on proportional loss of potential yield, and no commercial varieties of sugar beet in present use, nor single lines derived from these varieties, showed promise of providing breeding material for the production of

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tolerant or resistant strains (Hull and Watson, 1947). All these results showed that the disease was a potential threat to the sugar beet root crop, but it was not until after 1944 and 1945, when the first early and widespread outbreaks occurred, which were recognized as being caused by the virus, that serious attempts to find a means of controlling it were started.

Thousands of plants have been raised from seed set by infected beet plants, but no evidence has been obtained that S.B.Y. virus is transmitted through the seed. Attention was therefore concentrated on finding the sources from which the virus is introduced into the initially healthy root crop by the aphids. It was already known to be more prevalent in areas where beet and mangold seed crops are grown intensively than in other areas. These seed crops are raised in late summer as " stecklings ", and remain in the ground until they are planted out as seed plants, usually in the following early spring. They become infected in the steckling stage by aphids migrating from the root crops, and the virus remains in them through the winter, after the root crops have been harvested. Present control measures are mainly directed towards maintaining healthy seed crops. Stecklings are raised in isolation in areas where other chenopodiaceaous crops are not intensively grown, and transported to the seed-growing areas before planting out. These, and other methods that prevent the stecklings from becoming infected have been successful in producing healthy seed-crops, which give a heavier yield of seed than those from stecklings raised in conditions where they become infected (Hull, Rothamsted Reports, 1950, 1951). How far they will succeed in controlling the disease in the root crop as well, depends on how far the seed crops are the dominant sources of overwintering infection. Other sources, are clamped mangolds (Broadbent, Cornford, Hull and Tinsley, 1946), overwintering horticultural crops such as spinach and spinach beet, and *Beta maritima* in coastal areas, but their importance is uncertain. Recent examination of field data collected between 1943 and 1948 has shown that a high proportion of the variance in percentage infection between fields can be accounted for merely by variation in numbers of winged *M. persicae* visiting the crop (Watson and Healy, in preparation).

Transmission

Glasshouse studies with *M. persicae* and *A. fabae* as vectors (Watson, 1940, 1946), showed that beet yellows is a persistent virus. This means that the vectors do not become infective immediately they start to feed on infected plants, and they retain the ability to cause infection for hours or days after starting to feed on healthy plants. Persistent viruses are usually not transmitted by sap-inoculation, or are transmitted with difficulty, and this is true of beet yellows virus. For some years it could be transmitted only by aphids, but methods were later found by which it could be transmitted mechanically (p. 115).

M. persicae needs to feed for about six hours on infected plants before becoming fully infective, and for about six hours on healthy plants before they can cause all the infections of which they are capable. These times vary considerably, and some infections can

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be caused with much shorter feeding times. Longer feeding times than six hours do not greatly increase their efficiency, though there is often a slow rise up to about 20 hours.

With some persistent viruses there is an appreciable period, after the vector is removed from the infected plant and placed on the healthy one, during which it cannot cause infection. This phenomenon is not exhibited by beet yellows virus, for some insects can transmit after only 15 minutes on the infected and 15 minutes on the healthy plants, and either period can be reduced to seven minutes if the other is more prolonged (Watson, 1940).

M. persicae may remain infective while feeding on healthy plants for at least three days after leaving the infected plants. The ability to infect is also retained through prolonged periods of fasting, though infectivity is lost rather rapidly during the first few hours. These properties contribute greatly to the widespread distribution of beet yellows virus in the field, for a single infective *M. persicae* can infect several plants, even after a prolonged migration flight.

Symptoms of beet yellows virus

Under glass, the first symptoms of infection in seedling beet appear within 7 to 10 days. The tissues immediately above the veins on the distal portions of the developing leaves usually become yellowed; the yellowed cells are at first raised above the leaf surface, but very soon they collapse and become necrotic. This "etch" symptom, so-called because of the fretted appearance of the tissues above the veins, forms a net-like pattern which spreads towards the base of the leaf. At a slightly later stage the etched leaves become generally yellow; after two or three weeks, the etch symptoms change to a generalized necrosis, and do not re-appear on leaves which develop subsequently. From this time onwards the developing leaves are green and healthy looking, but when almost fully expanded their tips become yellow and the yellowing spreads downwards over the whole leaf, tending to avoid the areas immediately around the veins. In the field affected leaves are bright golden colour, sometimes with scarlet spots or freckles; they become thickened and brittle, giving rise to the term "crackly yellows" by which the disease was first known in England.

This is a description of symptoms caused by the beet yellows virus which was isolated at Rothamsted in 1938. This isolate has been maintained in the glasshouse up to the present time, and has shown no appreciable modification of symptoms, or behaviour in relation to aphids. However, it was realized, even in 1938, that not all isolates from the field gave exactly the same symptoms. The general type of yellowing, and development was the same, but apparently most isolates at the time did not give the etch symptom (Watson, 1940). The older leaves merely became yellowed and the younger leaves were symptomless. In 1939 infected leaves were obtained from Professor Quanjer at Wageningen, to compare the English and continental yellowing viruses. The disease isolated from these leaves was of the mild type, free from the etch symptom. Vector relationships of the mild viruses resembled those of the severe virus, but the mild viruses were more difficult to transmit, and the symptoms were more difficult to observe, so they were

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discarded. It was assumed that they were " mild strains " of the type virus.

Serology

An antiserum, made by injecting rabbits with sap from infected plants, was prepared against beet yellows virus in 1942 (Kleckowski and Watson, 1944), and some physical properties of the virus were determined. The activity of the antigen was destroyed by heating for 10 minutes at 50°C, and by keeping for two or three days at room temperature. It was unaffected by pH changes between 5 and 9, and could be reversibly precipitated by addition of ammonium sulphate to the clarified sap.

Sap taken from plants naturally infected in the field gave specific precipitates to this antiserum, and it was later found that sap from plants naturally infected in European countries also gave positive precipitin tests with it, and our virus with antisera prepared in Holland and Sweden.

The antiserum was of value for field diagnosis, but the results were not always clear-cut. Sometimes sap from old plants, late in the season, failed to precipitate with the antiserum in the usual way because they contained substances which inhibited specific precipitation except with very high concentrations of the antiserum. Aphid transmission tests from these plants usually showed that the virus was present.

Sometimes both tests failed, and it was assumed, with reservations, that the yellowing of the leaves was not caused by virus. But later results show that some of these leaves could have contained mild yellowing diseases which do not give positive precipitin tests with beet yellows antiserum, and which give symptoms that are difficult to identify, because most of these tests were done in the autumn, when light conditions are not very good. On the other hand the existence of apparent " mild strains " was known, and attempts had been made to re-isolate them so as to compare their effect on yield with that of beet yellows virus, but had been unsuccessful.

It is possible that the mild viruses were really absent from the English sugar beet crops at this time for the tremendous spread of viruses which occurred in them in 1944 and 1945 could have caused the mild viruses to be " swamped " by the more virulent beet yellows virus. Thus the composition of the yellowing diseases in the English sugar beet crops may have changed. This is also suggested by the fact that saps from field infected plants now give much greater precipitin titres than they did in earlier years.

The effect of concentration of inoculum on symptoms of beet yellows virus

Isolates which appeared to be mild forms of beet yellows virus were obtained from the field, and on several occasions were propagated in the glasshouse, to provide material for field experiments in the following year, but always, during the winter when sub-inoculations could not be made continuously, they reverted to the ordinary beet yellows type, showing the characteristic etch symptom.

Unstable " mild strains ", were also isolated from the type virus. The plants in any particular batch of inoculations show considerable

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variation in symptoms, and it is possible, by repeated selection from the most mildly infected plants to produce " strains " from which the etch symptom is almost eliminated. However, these " strains " also could only be maintained so long as the transfers were made continuously. During the winter the distinctions between the mild isolates and the type virus disappeared.

It was thought that these so-called " strain " differences might be purely quantitative, that a plant with weak symptoms contained little virus, and that transmissions from it would give weakly infected plants, only so long as they were made sufficiently frequently to prevent the virus from building up to a " normal " level, i.e. that which provoked " normal " beet yellows virus symptoms. This was supported by the fact that saps from the mildly infected plants gave low precipitation titres.

A way of testing the effect of very small doses of virus compared with larger ones, was to vary the number of aphids used for transmission. With most other viruses this does not affect the final symptoms ; small or large doses of inoculum, whether applied mechanically or by means of aphids, give rise to identical symptoms, though the development time may vary. With beet yellows virus varying the number of aphids did cause variation in symptoms. When 1, 5 and 10 aphids were used to transmit the virus to groups of 25 plants in 4 replications (total of 100 plants per treatment), the total number of plants which became infected were : 34, 75, and 94 respectively. These figures fit well with the hypothesis that the infections are local and independent (Watson, 1936), the chances of a plant becoming infected being no greater than the chance that a single aphid in any group will give rise to infection. However, the numbers of plants showing severe symptoms with definite etch, were 4, 31, 51, for the 1, 5 and 10 aphid groups. The increase in severe symptoms with aphid number was thus greater than would be expected if the mild and severe symptoms were caused by infection with strains of different virulence, but seemed to depend rather on the quantity of virus initially introduced into the plants.

Other yellowing diseases of sugar beet

In 1946 an aphid transmissible yellowing disease was isolated from a single " breeder's pure line " of sugar beet (Family 41), bred by B. Crombie of the Eire Sugar Corporation. This virus was remarkable in being readily transmissible through the seed of Family 41, and it was this property which led to its discovery, for had it not appeared in a large proportion of the progeny from a single " mother beet ", it would probably have escaped notice. The Family 41 disease was investigated in Eire by Clinch and Loughnane (1948), who found that two yellowing diseases of sugar beet were common in Eire. One was a mild yellowing disease which did not give the etch symptoms, and the other was a severe yellowing disease which seemed to be the same as the beet yellows virus in England, but differed from it in that the etch symptom persisted throughout the life of the plant, nor merely for the first two or three weeks after inoculation. The symptoms and behaviour of the mild yellowing strain of beet yellows virus seemed to be indistinguishable from those of the yellowing disease of Family 41, so Clinch and Loughnane

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concluded that they were the same, and that both were strains of beet yellows virus. Their explanation of the seed transmission in Family 41 was that this strain of sugar beet had developed a genetical abnormality which permitted the passage of the virus into the seed.

They made the interesting observation that neither 41 yellows nor the " mild strain " of beet yellows virus could protect a plant against subsequent inoculation with the " severe strain ". A positive cross immunity test is usually accepted as indicative of strain relationships between plant viruses, but Clinch and Loughnane doubted its validity as a test for relationship between aphid transmitted viruses of the beet yellows type, which were thought to be confined to the phloem. At this time beet yellows virus was not known to be sap-transmissible.

Work done at Rothamsted on the yellowing disease of Family 41, and the mild virus (Irish Mild Yellows, I.M.S.), isolated from ordinary sugar beet crops in Eire, has confirmed that neither willl protect against S.B.Y. virus. It was also found that saps from 41 Yellows and I.M.S. infected plants, whether taken from the glasshouse or grown out of doors, did not precipitate specifically with beet yellows antiserum. Failure to precipitate would be caused if the mild yellowing viruses did not contain the antigen against which beet yellows antiserum is formed, or if the antigen were in very low concentration relative to the amount in beet yellows virus. If it is merely a question of concentration, special techniques might be used to increase the concentration of virus in the extracted sap and induce it to precipitate specifically with the antiserum, but so far this has not been possible, and the evidence, at present, is that the viruses are not serologically related. I.M.S. virus appears to be quite stable and has been maintained in the glasshouse for several years.

Although symptoms of 41 yellows are very similar to those of Irish mild yellows when the plants are grown out of doors, in glasshouse conditions they do not seem to be identical. The disease caused by I.M.S. virus resembles the mild yellowing diseases isolated from fields in England in 1938, and also the unstable mild strains isolated from beet yellows virus by selection. The main difference between them and beet yellows virus is that there is no etch symptom. With 41 yellows the symptoms of the virus when transmitted to healthy seedlings are much weaker and more ephemeral. Sometimes only one leaf becomes yellowed or shows yellowed patches, and recovery may appear to be complete within a few days. If the plants are planted out of doors the yellowing symptoms return, and the seed always contains a high proportion (sometimes over 40 per cent), of infected progeny. The symptoms in the progeny are very variable. If they appear soon after germination they may cause stunting, distortion or death of the plant. If they develop when the plant is a few weeks old, they may look very like Irish mild yellows. The 41 yellows virus is also more difficult to transmit by aphids than Irish mild yellows. Using 10 aphids per plant only about 10 per cent of plants showed visible symptoms, compared with about 80 per cent for I.M.S. virus. With very large numbers of aphids or constant movement between infector and test plants

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(Clinch and Loughnane, 1948), this kind of difference largely disappears.

The virus of 41 yellows was found to be transmissible through the seed of other varieties than Family 41. These were Kleinwanzleben E variety, and some breeders' pure lines derived from Hilleshog variety. Therefore seed transmission is not confined to Family 41, and the suggestion that a genetical mutation in the plant is the cause of seed transmission is untenable. Work is still in progress to find whether I.M.S. virus is seed transmissible, but it seems unlikely, because the disease is common in Eire, and probably many seed stocks would have become infected if it were normally seed transmitted.

On present evidence the three viruses seem to be distinguishable from each other by serological heterogeneity, by the property of being seed transmitted, and by the symptoms produced in certain conditions, and it seems unlikely that they are the same virus, or even very closely related strains.

The existence of stable mild yellowing diseases suggested an explanation for the behaviour of the disease discovered in Australia. This also resembled beet yellows virus in the field, and it was transmissible by aphids to spinach, but apparently could not be reintroduced into sugar beet under glass. It also failed to precipitate specifically with beet yellows antiserum. These characters suggest that it might be another mild yellowing disease such as Irish mild yellows.

Sap transmission of beet yellows virus

In 1941 Kassanis showed that beet yellows virus could be transmitted by sap inoculation to sugar beet plants. The necessary conditions were that the test plants should be kept for at least one or two days in the dark, that the inoculum should be obtained from severely affected plants showing good etch symptoms, and that the inoculation should be made with an abrasive. In these conditions rubbing a mature healthy leaf with infected sap caused the appearance of numerous dark coloured necrotic lesions. About 25 per cent of the plants became systemically infected, showing both etch and yellowing symptoms, and sap from them precipitated specifically with beet yellows antiserum. When similar inoculations were made with beet yellows virus into the leaves of Irish mild yellows infected plants, lesions appeared just as quickly as in healthy plants, and appeared to be even more numerous. This seemed to show conclusively that the failure to show immunological relationships between these two viruses was not because they were confined to the phloem.

The necrotic strain of beet yellows virus

The statement made by Clinch and Loughnane that etch symptoms persisted in their beet yellows infected plants also led to further investigation of the symptoms of this virus. It might appear to be a rather unimportant difference, but we had paid so much attention to following the course of the etch symptom, and failed so often either to eliminate or materially to increase it in any stable isolate, that it seemed to be of considerable interest. We had

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already observed that some plants in the field retained their etch symptoms throughout the growing season (Hale *et al*, 1946), and we had attributed this, vaguely, to some genetical attribute of individual plants. Our beet yellows virus was not collected from such a plant, but from one showing typical "crackly-yellows" from which any early etch symptoms had presumably disappeared. Therefore new isolates were made from the field, from old plants in which etch symptoms had persisted. These isolates gave rise to infected plants in which the etch symptom persisted throughout life, even through the winter months when the yellowing symptom had completely disappeared. Saps from these plants precipitated with antisera made against S.B.Y. infected sap, and gave higher titres than saps from S.B.Y. infected plants.

Cross inoculation tests were made between this virus and the S.B.Y. virus by the expedient of waiting until the etch symptoms had almost disappeared from the S.B.Y. plants, and then inoculating these plants, and healthy plants of the same age, with the new isolate (S.B.Y.N.). The results were quite clear-cut, for the S.B.Y. plants failed to develop any further etch symptoms, but the healthy plants became infected with the new virus, and produced etch symptoms plentifully on their developing leaves, later exhibiting typical S.B.Y. symptoms of the persistent etch type. This experiment showed that one sugar beet virus can protect against another. With other viruses this is regarded as evidence of relationship. The fact that protection can be established means that failure to exhibit it also suggests lack of relationship. Therefore I.M.S. virus is more distantly related to S.B.Y. virus than is S.B.Y.N., as its failure to give a positive precipitin test with S.B.Y. antiserum also indicates.

S.B.Y.N. has proved stable, and is useful for experimental work because of its ability to cause easily recognizable symptoms in the glasshouse in winter.

Yellow-net virus

In 1949 Sylvester, in California, described another aphid transmitted virus of sugar beet, which was persistent in the vectors *M. persicae* and *A. fabae*, and was apparently not sap-transmissible. In its general properties it thus resembled beet yellows virus, but its symptoms, superficially, did not. The disease caused a striking yellow vein-banding symptom, which never became necrotic and which affected all the veins of the leaves, sometimes over the whole plant. The net-like pattern of the anastomosing yellowed veins suggested the name of Yellow-net virus.

In 1950 Dr. Hull isolated a virus whose symptoms and properties appear to be identical with yellow-net, from a plant found in a field in Lincolnshire. This virus is less readily transmitted by *M. persicae* than is beet yellows virus. The aphids require longer feeding times on infected and healthy plants to develop optimum infectivity, and probably they remain infective for a longer time, though this has not yet been measured exactly.

The symptoms are rather erratic in their time of appearance; sometimes they are obvious within 10-14 days of inoculation and sometimes they appear at intervals of three or four weeks. There

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is little difference in symptoms whether the plants are grown in or out of the glasshouse, and many plants retain the symptom fully, throughout the winter.

In transmission tests some plants failed to produce yellow-net symptoms, but did produce symptoms which were not distinguishable, either in or out of the glasshouse, from those of I.M.S. virus. This virus, which we call the yellow-net mild strain, is easily isolated from the yellow-net complex, for it seems to be more readily transmitted by the aphids, and many transmission tests which fail to transfer yellow-net are highly successful with the mild strain. Subcultures can be made repeatedly from these plants without the yellow-net virus re-appearing. The yellow-net mild strain appears to be quite uniform and stable. So far, yellow-net has not been isolated free from the mild strain. Whether there is an obligate association between the two viruses, or whether the failure is due to the greater ease with which the mild strain is transmitted, requires more work to establish, but the character and properties of the two viruses are sufficiently like those of beet yellows virus for them to be included in the same group. In fact there seem to be some points of resemblance between the yellow-net virus and the etch component of S.B.Y. virus. Both affect the tissues above the veins causing chlorosis, neither are greatly affected by glasshouse conditions compared with outdoor conditions, and both persist throughout the winter. They are both associated with mild yellowing viruses which can exist independently, but neither has been isolated free from these yellowing viruses so far.

Neither the yellow-net complex nor the yellow-net mild strain virus has given positive precipitin tests with beet yellows antiserum.

The effects of light and carbohydrate supply on beet yellowing viruses

One aspect of the work with the beet yellowing viruses which has continually caused anomalous and contradictory results, was the apparent dependence of the appearance of the yellowing symptoms on conditions of growth. The factor believed to be mainly concerned is light intensity.

Beet yellows virus has been associated with abnormalities in carbohydrate metabolism and translocation ever since it was first described by Quanjer, who observed that the yellowed leaves contained unusually large quantities of starch. He attributed this accumulation to degeneration of the phloem which was thought to interfere with translocation, but Klinkenberg, 1948, showed that phloem symptoms are not necessarily associated with symptoms of beet yellows virus, and when they do occur, it is after most of the carbohydrate has accumulated.

Watson and Watson, 1951, showed that, though starch, sucrose and hexose all accumulated in the yellowed leaves of plants infected with beet yellows virus, translocation was not obstructed but was the same for healthy and diseased leaves. They showed also that carbohydrate is not increased in the young, green leaves of infected plants, but only in the older ones. This is shown by the following abstract from the data :—

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Total carbohydrate as per cent dry matter content

	Old leaves		Young leaves	
	Healthy	Infected	Healthy	Infected
Per cent carbohydrate ...	8·4	18·7	12·5	12·0

These results suggested that the yellowing depended in some way on the accumulation of carbohydrate caused by the virus, and that where this did not naturally occur, as in the young leaves, or was prevented by lack of light as in shade or winter conditions, yellowing also did not occur.

Experiments were made to compare the effects of light and shade on symptom production, and also to see whether artificially increasing the carbohydrate content of the leaves would intensify the yellowing symptoms. This was done by spraying the leaves of infected and healthy plants daily with a 10 per cent solution of sucrose, in ordinary glasshouse conditions in the spring, and treating other plants at the same time in muslin cages placed in the glasshouse, which reduced the light intensity to about half.

The concentration of sucrose and starch in leaves of healthy and infected plants was increased by sugar spraying. Yellowing symptoms were increased on infected plants, but not produced on healthy ones. The increase of yellowing symptoms was more conspicuous in plants which were not shaded than in those which were. The shaded plants produced scarcely any yellowing, and the improvement produced by spraying was small and somewhat irregular. The etch symptoms were less affected both by sugar spraying and by shading than were the yellowing symptoms.

Serological tests on saps from the differently treated plants were rather unsatisfactory, because the antiserum then available was poor, and the virus titres were very low, at best only 1/32. The virus was detected in old (yellowed) and young leaves of all infected plants. Titres were higher in the unshaded plants, and in these plants, were higher in the yellowed than in the young leaves. The best titres were obtained for the yellowed leaves which received sugar, but whether this indicated a real increase caused by sugar is uncertain. In shade conditions the young leaves gave a slightly higher titre than the old (unyellowed) leaves, and again sugar caused a rather doubtful increase.

These results suggest that, though beet yellows virus is the cause of the yellowing symptom, it cannot produce that symptom when the carbohydrate content of the leaves is low. Thus, on this hypothesis, yellowing does not occur in the young leaves, because in them the carbohydrate of infected plants is not in excess of the normal carbohydrate content of the leaf, as judged by the condition of the healthy ones. Where there is great excess of carbohydrate the yellowing symptoms are severe ; in shade conditions when carbon assimilation is insufficient to cause excessive accumulation of starch and sugars, symptoms are slight or absent.

If this applies to all the viruses which cause typical yellowing symptoms in sugar beet, as their general behaviour suggests, it explains why plants grown in comparatively shady conditions in glasshouses produce poor symptoms, whereas the same plants placed out of doors have good visible symptoms. Unfortunately

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growing the plants out of doors is not a possible solution to the problem of symptom failure in experiments, because natural infection with other yellowing diseases cannot be controlled. However it is usually possible to ensure that critical experiments are done in as good light conditions as possible, and with most of the yellowing viruses, it is possible to detect symptoms in glasshouse conditions, if it is ensured that the plants grow well enough for the yellowing to be distinguished from nutrient deficiency, wilting, and so on.

The best results are obtained, in this country, between April and the end of July, but even then symptom production may be poor if the houses are too frequently shaded by heavy blinds. Good symptoms are not produced in most glasshouses if the plants are grown in cages to protect them from aphid attack.

The existence of these mild yellowing virus diseases of sugar beet, together with the extreme effect of shading on the symptoms, the ephemeral nature of the etch symptom in some S.B.Y. strains, and the fact that symptoms can be affected by varying the dose of inoculum during transmission, make it difficult to relate the results of experiments made under varying conditions. More work will be needed before the relationships between some of the recently identified yellowing diseases of sugar beet and those previously described are fully understood.

1. Clinch (P. E. M.) and Loughnane (J. B.). 1948. Proc. Roy. Dublin. Soc., **24**, 307.
2. Broadbent (L.), Cornford (C. E.), Hull (R.) and Tinsley (T. W.). 1949. Ann. appl. Biol., **36**, 513.
3. Kassanis (B.). 1949. Ann. appl. Biol., **36**, 270.
4. Kleczkowski (A.) and Watson (M. A.). 1944. Ann. appl. Biol., **31**, 116..
5. Hale (J. B.), Watson (M. A.) and Hull (R.). 1946. Ann. appl. Biol., **33**, 13.
6. Hull (R.) and Watson (M. A.). 1947. J. agric. Sci., **37**, 301.
7. Petherbridge (F. R.) and Stirrup (H. H.). 1935. Bull. Min. Agric. London, **93**.
8. Quanjer (H. M.). 1934. Tijdschr. PlZeikt, **40**, 40.
9. Roland (G.). 1936. Tijdschr. PlZeikt, **42**, 54.
10. Stubbs (L.). 1949. Plant Diseases Conference (Duplicated Report), Australian Agricultural Council, Melbourne, Australia.
11. Sylvester (E. S.). 1949. Phytopathology, **38**, 429.
12. Van Schreven (D. A.). 1936. Meded. Inst. Suikerbiet., Bergen-o.-Z., **6**, 1.
13. Watson (M. A.). 1936. Philos. Trans., B., **226**, 457.
14. ——————— 1940. Proc. roy. Soc. B., **128**, 535.
15. ——————— 1942. Ann. appl. Biol., **29**, 358.
16. ——————— 1946. Proc. roy. Soc. B., **133**, 200.
17. Watson (M. A.) and Hull (R.). 1946. Min. Agric. Advisory Leaflet, No. 323.
18. Watson (M. A.), Watson (D. J.) and Hull (R.). 1946. J. agric. Sci., **36**, 151.
19. Watson (M. A.) and Watson (D. J.). 1951. Ann. appl. Biol., **38**, 276.
20. Watson (M. A.), Hull (R.), Blencowe (J. B.) and Hamlyn (Brenda M.). 1951. Ann. appl. Biol., **38**, 743.

https://doi.org/10.23637/ERADOC-1-73