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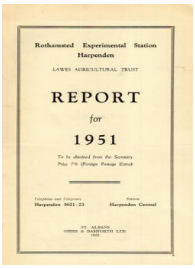

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BEET YELLOWS VIRUS AND OTHER YELLOWING VIRUS DISEASES OF SUGAR BEET

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.

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REVIEW OF WORK ON MANGANESE OXIDATION IN HIGHER PLANTS

By P. J. G. Mann

The work to be reviewed was undertaken in the Biochemistry Department at Rothamsted in 1948 with the object of investigating whether manganese undergoes a cycle of oxidation and reduction in higher plants. The work developed from the observation that with water extracts of certain plant roots in presence of pyrophosphate at pH 7 a pink colour was obtained within a few minutes of adding manganous sulphate and hydrogen peroxide; this suggested that manganipyrophosphate had been formed. Except in strongly acid solutions manganic ions are unstable and dismute to manganous ion and hydrated manganese dioxide :—

$$2\,Mn^{+++} + 2H_2O \longrightarrow Mn^{++} + MnO_2 + 4H^+ \quad \ldots \quad (1)$$

Some complexions of manganic manganese are more stable. Thus manganipyrophosphate is stable up to pH 9 but dismutes at more alkaline reactions. By making use of this dismutation Kenten and Mann (1949) isolated manganese dioxide from large-scale reaction mixtures of plant extract, manganous sulphate and hydrogen peroxide and thus proved that the colour reactions obtained were indeed due to the formation of manganipyrophosphate. The manganese dioxide isolated in such experiments was equivalent to only 25-40 per cent of the hydrogen peroxide added. A manometric study of the reaction showed that these low yields were due to the fact that manganipyrophosphate reacts stoicheiometrically with hydrogen peroxide under the experimental conditions. The oxidation therefore causes oxygen evolution and the reactions taking place in the pyrophosphate media may be represented by the equations :—

$$2MnO + H_2O_2 \longrightarrow Mn_2O_3 + H_2O \quad \ldots \quad \ldots \quad (2)$$

$$Mn_2O_3 + H_2O_2 \longrightarrow 2MnO + H_2O + O_2 \quad \ldots \quad (3)$$

The accumulation of manganipyrophosphate therefore depends on reaction (2) being more rapid than reaction (3). No accumulation of manganese oxidation product could be demonstrated in orthophosphate media. Here a stable manganic complex cannot be formed and oxidation would result in the formation of manganese dioxide either directly or through the dismutation of an unstable manganic complex. Manganese dioxide does not accumulate in the orthophosphate media because the rate of its reduction by hydrogen peroxide (4) is as rapid as its formation :—

$$MnO_2 + H_2O_2 \longrightarrow MnO + H_2O + O_2 \quad \ldots \quad (4)$$

Under these conditions the rate of decomposition of the hydrogen peroxide is a measure of the rate of manganese oxidation. In manometric experiments it was shown that the addition of as little as 1 μg Mn^{++}/ml extract increased the rate of decomposition of hydrogen peroxide by plant extracts. Indirect evidence was thus obtained that manganese oxidation by the system proceeded readily at physiological concentrations of Mn^{++}.

Analysis of the oxidizing system showed that it consisted of a thermolabile and a thermostable factor in addition to hydrogen peroxide. Evidence was obtained that the thermolabile factor is

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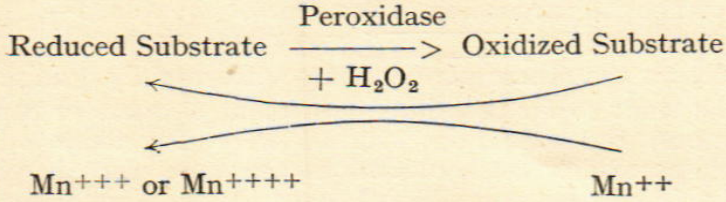
the enzyme peroxidase and the thermostable factor a peroxidase substrate. It was suggested, as a working hypothesis, that intermediate products of the oxidation of certain peroxidase substrates can bring about manganese oxidation according to the scheme below which involves the substrate in a cycle of oxidation and reduction.



The concentrations of hydrogen peroxide used in the preceding experiments were necessarily high compared with those likely to be present *in vivo*. It was suggested that *in vivo* the manganese oxidation product would react preferentially with plant metabolites other than hydrogen peroxide thus involving the manganese in an oxidation-reduction cycle which could be responsible for its known effect on plant respiration.

In subsequent work Kenten and Mann (1950) established the fact that peroxidase in presence of certain of its substrates, catalyses the oxidation of Mn^{++}. The reaction mixtures used in this work were similar to those previously described with the exception that the plant extracts were replaced by partially purified peroxidase preparations together with peroxidase substrates such as phenol or p-cresol. Manganese dioxide was isolated from large-scale reaction mixtures. In manometric experiments systems active in manganese oxidation were produced by the use of a few μg of peroxidase preparation and phenolic substrate. The catalytic activity of the phenolic substrates in the oxidation was illustrated by the fact that in one experiment the addition of 1 μg p-cresol to the system led to an accumulation of manganipyrophosphate equivalent to 605 μg Mn_2O_3. Probably the most important results of the manometric work were those obtained in a study of the effect of variation in the hydrogen peroxide concentration. Under the conditions used, the accumulated manganipyrophosphate, calculated as a percentage of that theoretically possible, increased with decreasing concentration of hydrogen peroxide from 30 per cent at 0·0066 M. hydrogen peroxide to 80 per cent at 0·0017 M. which was the lowest concentration practicable with the technique used. This result supported the suggestion previously made that with sufficiently low hydrogen peroxide concentrations all the manganese oxidation product would be available for the oxidation of plant metabolites other than hydrogen peroxide.

The possibility that the oxidizing capabilities of peroxidase systems towards inorganic compounds are not confined to compounds of manganese was investigated by Kenten and Mann (1951). The results of this work showed that peroxidase in presence of suitable phenolic substrates catalyses the oxidation of ferrocyanide by hydrogen peroxide and possibly that of molybdates, vanadates and tungstates, though in the latter cases direct proof of the oxidation was not obtained. It was found that under certain conditions the rate of oxidation of Mn^{++} by peroxidase systems is markedly

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increased by catalytic concentrations of these compounds. Thus a clear effect of ammonium molybdate was obtained at a concentration of 10^{-6} M. (2·4 μg Mo/3ml). This was attributed to catalysis by peroxidase systems, of the oxidation of molybdates, tungstates and vanadates by hydrogen peroxide to the corresponding peroxy compounds according to the general equation :—

$$X{\cdot}OH + H_2O_2 \rightleftharpoons X{\cdot}O{\cdot}OH + H_2O \quad \ldots \quad (5)$$

It was shown that these peroxy compounds rapidly oxidise Mn^{++}.

In the work so far described with plant extracts or peroxidase preparations, the oxidations studied were dependent on added hydrogen peroxide. Such oxidations can take place in plants *in vivo* only if hydrogen peroxide is formed by the plant tissues. The work of Kenten and Mann (1952a) showed that Mn^{++} is oxidized by plant extracts in absence of added hydrogen peroxide and that such oxidation forms a useful test for the presence in the extracts of enzyme systems producing hydrogen peroxide. The blue coloration obtained when benzidine and hydrogen peroxide are added to most plant and animal tissues is known to be due to the catalysis by peroxidase, or by the peroxidatic action of haem or haematin derivatives, of the oxidation of benzidine by hydrogen peroxide. Therefore a blue coloration obtained with tissue and benzidine in the absence of added hydrogen peroxide suggests the formation of hydrogen peroxide by the tissue. Using this test for the production of hydrogen peroxide by plant extracts Kenten and Mann (1952a) obtained negative or only weakly positive results. But extracts of many plants gave positive benzidine tests after incubation with Mn^{++} and pyrophosphate at pH 7. Evidence was obtained that this was due to oxidation of the Mn^{++} and accumulation of manganipyrophosphate which readily oxidizes benzidine. The manganese oxidation was shown to be dependent on the formation of hydrogen peroxide by the plant extracts. Hydrogen peroxide cannot accumulate to any extent in the extracts owing to their strong catalatic activity.

In attempts to identify the enzyme systems producing hydrogen peroxide it was shown that pea seedling extracts (*Pisum sativum* L.) oxidize both mono- and di-amines according to the general equation :

$$R{\cdot}CH_2{\cdot}NH_2 + O_2 + H_2O \longrightarrow R{\cdot}CHO + H_2O_2 + NH_3 \quad \ldots \quad (6)$$

Some evidence was also obtained of the presence of an aldehyde oxidase catalysing the oxidation of phenylacetaldehyde with hydrogen peroxide formation. It seems of particular interest that tryptamine is among the amines attacked by the amine oxidase in view of the evidence that tryptamine can function as a precursor of indoleacetic acid in the plant. The action of the amine oxidase on tryptamine presumably results in the formation of indoleacetaldehyde. It was suggested that indoleacetaldehyde is a substrate of the aldehyde oxidase present in the extracts and that indoleacetic acid could be formed from tryptamine by the action of the amine oxidase followed by that of the aldehyde oxidase.

The oxidation of added Mn^{++} by plant extracts in absence of added hydrogen peroxide was attributed by Kenten and Mann (1952a) to the presence in the extracts of enzyme systems producing hydrogen peroxide. In subsequent work Kenten and Mann (1952b) working with partially purified enzyme preparations demonstrated

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the oxidation of Mn^{++} by enzyme systems producing hydrogen peroxide coupled with peroxidase. The enzyme systems used to provide hydrogen peroxide were the D-amino-acid oxidase and xanthine oxidase from animal sources and the plant amine oxidase systems already described. The reactions catalysed by these enzymes may be represented by the general equation :—

$$RH_2 + O_2 \longrightarrow R + H_2O_2 \quad \ldots \quad \ldots \quad (7)$$

If the hydrogen peroxide formed is decomposed by catalase according to the equation :—

$$H_2O_2 \longrightarrow \tfrac{1}{2}O_2 + H_2O \quad \ldots \quad \ldots \quad (8)$$

the apparent oxygen uptake is halved and the net reaction is :—

$$RH_2 + \tfrac{1}{2}O_2 \longrightarrow R + H_2O \quad \ldots \quad \ldots \quad (9)$$

It is well known that the hydrogen peroxide produced by these enzyme systems may be used for secondary or coupled oxidations catalysed by peroxidase. Where catalase is present such coupled oxidations cause increases in the apparent oxygen uptake which may reach 100 per cent if all the hydrogen peroxide is used in the coupled oxidation rather than decomposed by catalase. In manometric experiments with these enzyme systems in both pyrophosphate and orthophosphate buffers it was shown that, in presence of catalase, the addition of peroxidase and manganous sulphate together with catalytic amounts of p-cresol caused a doubling of the oxygen uptake of the primary reaction suggesting that all the hydrogen peroxide was used for manganese oxidation. This was confirmed by the finding that the manganese oxidation products which accumulated were equivalent to the hydrogen peroxide produced in the primary reactions. Manganese oxidation was also observed in systems composed of the plant enzyme α-hydroxyacid oxidase and its substrates, lactic or glycollic acids, with peroxidase. Proof was thus obtained that hydrogen peroxide is formed in oxidations catalysed by α-hydroxyacid oxidase. This has since been confirmed by more conventional methods (Kenten and Mann, 1952c).

In the preceding experiments, designed to demonstrate quantitative accumulation of manganese oxidation products, Mn^{++} was added in amounts more than equivalent to the hydrogen peroxide formed by the primary enzyme system. It was shown that Mn^{++} when present in low concentrations was also oxidized by adding oxalate to the system which reduced the manganese oxidation product as fast as it was formed and thus allowed the Mn^{++} to undergo a cycle of oxidation and reduction. The oxidation of these small amounts of Mn^{++} with accumulation of the oxidation product produced little increase in the oxygen uptake. Where oxalate was present in addition to Mn^{++}, however, large increases in oxygen uptake were observed due to the manganese oxidation-reduction cycle.

In this attempt to reconstruct with purified enzyme preparations the type of systems which causes manganese oxidation in plant extracts the significance of the results lies not merely in the fact that manganese oxidation was observed but also that the oxidation product accumulated quantitatively. In previous results with added hydrogen peroxide the accumulation of manganipyrophosphate was not quantitative and no accumulation could be demonstrated in orthophosphate owing to reduction of the manganese

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dioxide by hydrogen peroxide (equations 3 and 4). Provided the hydrogen peroxide concentration is sufficiently low this reduction is negligible and all the oxidation product would be available in the plant for the oxidation of metabolites other than hydrogen peroxide as suggested by Kenten and Mann (1949). Under such conditions the Mn^{++} would undergo, in the plant, a cycle of oxidation and reduction as demonstrated *in vitro* in the experiments with oxalate as reductant.

The results obtained may throw light not only on the physiological action of manganese but also on that of peroxidase. Peroxidase is widely distributed in higher plants but its functions are unknown. It has not been shown to catalyse the oxidation of any compound recognized as an intermediate in the main pathways of metabolism. Since peroxidase catalyses the oxidation of Mn^{++} the range of compounds which peroxidase systems can oxidize will be extended by addition of Mn^{++} to include those compounds capable of oxidation by the manganese oxidation product. Thus, in work as yet unpublished, it has been shown that in presence but not in absence of Mn^{++} peroxidase systems oxidize not only oxalate but also oxaloacetate, ketomalonate and dihydroxytartrate.

Manganese oxidation has not yet been demonstrated in the plant *in vivo* under normal conditions of manganese supply. In preliminary experiments with plants grown in water culture with high manganese concentrations evidence has been obtained of the accumulation of manganese oxidation products in the tissues. This can generally be shown in the pea plant (*Pisum sativum* L.) with concentrations of Mn^{++} of the order of 50 p.p.m. in the culture solution. It has already been shown that the oxidation takes place *in vitro* with physiological concentrations of Mn^{++}. At such concentrations of Mn^{++} *in vivo* it is possible that the oxidation product is reduced by plant metabolites as fast as it is formed and that only under conditions of manganese toxicity is the rate of oxidation faster than the rate of reduction. This suggests the possibility that the symptoms of manganese toxicity may be due, at least in part, to the deposition of higher oxides of manganese in the plant tissues.

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HOW FAR DO INSECTS TRAVEL?

By C. B. WILLIAMS

A knowledge of the distances that either injurious or beneficial insects can move is of major importance to economic entomologists all over the world in planning either direct control, or measures for the prevention of outbreaks of insect pests or of insect-borne diseases.

Apart from accidental transport in cars, trains, ships and aeroplanes, which is a major problem in itself, movements of insects over longer or shorter distances can take place actively—by the action of the insects themselves, or passively—when they are carried away by the wind. The active movements may be just the normal flight of the insects wandering round their habitat, or may be definite flights over a long distance in a fixed direction. For the latter type of movement we use the term " migration ".

As the study of insect migration and insect drift has been one of the major investigations of the Department of Entomology at Rothamsted for many years, it may be of interest to summarize some of the results obtained, and to show how they have altered the outlook of the applied entomologists.

There is no doubt that 50 years ago, or less, there was little knowledge and much misunderstanding, particularly among the general public, about how long insects lived in their adult winged stage and how far they could move during that period.

To clear up the former point we may say that an adult life of less than a week is probably unusual in flying insects ; that most live two or three weeks ; that quite a number live several months ; and that a small number of species (including bees and ants) are known to live several years. In a life of even only a few weeks there are possibilities of movement much greater than that of the hypothetical May-fly which hatches in the evening and is dead before daybreak, and we now know that even in 24 hours the distances covered could be measured in miles rather than in yards.

In lectures on Agriculture at the University of Cambridge in 1910, one of the major reasons given for the rotation of crops was its value in preventing outbreaks of insect pests. There are undoubtedly some pests, particularly those in the soil, which have little power of movement ; and there are others, such as wireworms, with a larval stage lasting several years, in which a sudden break in continuity of food supply might possibly be injurious to the insect. But to imagine that the average insect pest would be seriously incommoded by having to move from one side of the hedge to another in order to find its next year's food supply is quite unjustified.

There are many good reasons for a rotation of crops in farming practice, but the value of the process as a method of avoiding insect pests has been greatly exaggerated.

It is perhaps interesting to note at this stage that Broadbalk field has, in the first 100 years of wheat growing, not yet suffered from any major disaster. So that the absence of rotation does not appear to be—by itself—a necessarily dangerous proceeding in the development of insect attack.

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In a light trap that was worked by the Entomology Department on one of the fields at Rothamsted for four consecutive years 1933-1936, 240 different species of moths were captured. The total number of known species in the families of insects concerned is about 750 for the whole of the British Isles, so that in four years almost one-third of all the British species of moths had visited one spot in one field. Some of these may have bred within a few yards of the trap—some others, on the contrary (such as the Silver-Y Moth, *Plusia gamma*), may have migrated to the field from hundreds of miles away—but the majority had undoubtedly wandered accidentally to within the very limited sphere of danger round the traps from their natural habitats within perhaps a few miles of the trap.

Had the trap been a plant newly introduced to the area, then we must realize that in about four years perhaps one-third of the British moths would have come near enough to see whether or not it was suitable for food; and there is little doubt that most other groups of winged insects are capable of similar wanderings.

When we come to study the problem of movement in insects known to migrate, the distances covered rise from yards and miles, to tens or hundreds of miles, and occasionally even to a thousand or more.

Nearly every year our British population of Large Cabbage-White Butterflies (*Pieris brassicae* L.) is reinforced by considerable immigration from the Continent across the southern North Sea and the eastern half of the English Channel. Millions of butterflies cross without difficulty the 30 to 300 miles that separate us from France, Belgium, the Netherlands and Denmark. We have good reason to believe that before setting out to cross the North Sea, the butterflies had already in many cases flown from north Germany, from southern Scandinavia, or from islands in the Baltic (Williams, 1939).

An entomologist who studied the biology of this butterfly in England some years ago expressed an opinion that its death rate from parasites was so high that it was doubtful if it could long survive in Britain if it were not for the regular reinforcements which arrive from the Continent.

The Silver-Y Moth (*Plusia gamma* L.)—an irregular pest of peas, beans and many other field crops in England and Central Europe—does not survive the winter in any stage in these latitudes, but flies in from the Mediterranean area each spring in varying numbers. A big migration usually means a big outbreak and widespread damage—but the real source of the trouble is 400 miles or more from the area of damage if they have come from the northern shores of the Mediterranean, and more than twice that distance if their origin has been in North Africa.

In Egypt I have had experience with a case of an opposite kind. The Greasy Cutworm (*Agrotis ipsilon* L.) is a serious pest of grain in the autumn and winter months and occasionally attacks young cotton in the early spring. Money was at one time spent on a "control" measure which consisted of trapping, by means of a poison bait, large numbers of the adult moths particularly about April and May. It was then discovered that the insect was a

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migrant and only a winter visitor to Egypt. Every spring all the moths left the country and, almost certainly, flew to Europe—hundreds of miles away. A failure to understand this aspect of their behaviour resulted in much wasted effort and expenditure.

In North America one of the major pests of cotton during the nineteenth century was the "Cotton Leaf Worm" (*Alabama argillacea* Hbn.). It appeared each summer in the cotton belt of North America, and several generations in rapid succession often did enormous damage. During the winter the insect could not be found in any stage of development in any part of the cotton belt, nor could any alternative foodplant to cotton be discovered. Just a hundred years ago it was first suggested that the moths were coming in each spring as immigrants from South or Central America. This is now known to be the correct interpretation, although the exact area of origin is still uncertain, but for at least 50 years the suggestion was thought to be a wild improbability owing to the great distance of movement required.

The Painted Lady butterfly (*Pyrameis cardui* L.), which is occasionally a serious pest of artichokes in France, but is mildly beneficial in the U.S.A. where it feeds chiefly on thistles, breeds during our winter in the arid areas on the edges of the Great North African Desert. From there it moves north each spring, first to the north coast of Africa, then across the Mediterranean and northwards across Europe. It reaches the latitude of the British Isles almost every year about June, and not infrequently wanders on still further to Iceland in the west and to beyond the Arctic Circle in Finland. The total distance covered may be nearly 2,000 miles.

The same butterfly in North America appears to have its winter quarters in Western Mexico (where there are arid conditions somewhat similar to North Africa) and to spread out each spring towards the north and north-east. In some years thousands of millions of insects are concerned in the flights, and they may spread over the greater part of the United States and southern Canada, even as far as the mouth of the St. Lawrence River and to Newfoundland. Altogether they may move nearly 3,000 miles from their starting point in Mexico.

There is no evidence of winter survival of Painted Lady butterflies anywhere in northern Europe or in the United States.

There is no need to stress the great distance that can be traversed by swarms of locusts. Some of the few individuals that have occasionally been captured in Britain have been shown (by statistical measurements) to have almost certainly come from populations in south-eastern Europe—a distance of well over a thousand miles. Locusts and Painted Lady butterflies have on several occasions been captured on board ships more than a thousand miles from land.

An unusual adaptation to migration came to my notice in East Africa, where a burrowing wasp (*Sphex aegyptiacus*), which is predaceous on locusts, was found to have developed a habit of migrating along with the swarms of its host—so that both pest and predator were moving over many miles of country deliberately and simultaneously.

Many species of dragonflies are capable also of flights to be measured in hundreds of miles. I have seen them myself more than

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a hundred miles from water in the Egyptian desert, and—like the locusts and butterflies mentioned above—they have been recorded on ships at sea hundreds of miles from the nearest land, and hence from the nearest fresh water which is necessary to them for breeding.

Ladybirds in California are known to move from the coastal plains to the hills about 50 miles away every autumn for the purposes of hibernation, and to return back to the coast in the spring; and a somewhat similar habit is found in the American Bean Beetle (*Epilachna*), a relative of the ladybird which however lives on a vegetarian diet.

Turning now to the question of passive drifting of insects, this chiefly affects the smaller species with poor powers of flight which are easily carried away from the ground by winds and by convection currents. The insects mostly concerned are Aphidae (greenflies), small Diptera, small Hymenoptera, small beetles, with occasional Lacewings and other groups. They include many pests and transmitters of disease.

Small insects are often carried short distances of a hundred yards to a mile or so by winds quite near to the ground, but once they get away from the ground into the upper air they may be carried tens or even hundreds of miles before they come back to earth.

As early as 1913 entomologists in America had shown that the just hatched caterpillars of the Gipsy Moth (*Porthesia dispar*), which are covered with long hairs, are so buoyant in the air that they could be carried at least a mile by air currents near the ground. Two years later Collins (1915) showed that they could be carried up to at least 13 miles.

Experimental work with traps attached to aeroplanes was carried out between 1926 and 1931 in Louisiana, U.S.A., and the results (Glick, 1939) showed a total of nearly 25,000 insects caught at heights from 200-16,000 feet in a trap 1 foot square, in about 900 hours of flying during day hours. This is an average of about 30 insects per hour.

In about 100 hours flying at night time up to 5,000 feet, about 4,000 insects were caught.

The insects that were identified included about 700 different species belonging to 198 families and 18 different orders. There were also over 1,000 spiders and a small number of mites.

Included among the insects were over 50 wingless Thysanura and Collembola (one of the latter at 11,000 feet) and many wingless immature stages of Heteroptera, Homoptera, Orthoptera, Coleoptera, Lepidoptera and Diptera—also numbers of wingless ants.

In Britain Hardy, Milne and Freeman carried out upper air trapping by nets hung first from kites and later from tall radio masts (Hardy, Milne and Freeman). Hardy also had nets attached to the masts of ships crossing the North Sea, which were only opened when the ships were more than 50 miles from land. Their results agreed completely with the then unpublished results from the United States, and showed the unexpectedly large numbers of drifting insects that may be present in the upper air. Hardy calculated that on many fine summer days in the area over the North Sea more than 50 miles from land, there were millions of insects drifting above the sea, the majority of which were Aphidae.

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There is no doubt that with an easterly wind most of these would arrive alive in Britain.

In 1946 an investigation was started at Rothamsted under the immediate direction of Dr. C. G. Johnson and he was fortunate in being able to establish an excellent co-operation with the Research and Development Establishment of the Ministry of Supply, at their station for Barrage Balloons at Cardington, about 15 miles from Rothamsted. For the first time in this work it became possible to get continuous records, both by day and by night, simultaneously at several definite heights and almost independent of weather conditions.

In the first two years nets were attached to the cables of the balloons at different levels up to 2,000, and occasionally up to 4,000 feet, and the insects were thus filtered out of the air as the wind blew through the nets. Once again all previous results were confirmed with a great increase in reliability of interpretation. On a fine summer day in a net about 3 feet in diameter, at a height of 2,000 feet it was not unusual to get 10 or even 20 living aphids in a single hour. When one considers the microscopic proportion of the air that is being filtered by the net (which is practically invisible at 2,000 feet) it is possible to realize the enormous total population of insects that are being drifted across any mile front of land in any warm summer day.

More recently Johnson has put the work on a still sounder numerical basis by using suction traps which draw a fixed amount of air through the net, almost independent of the wind velocity. This technique enables quantitative results to be obtained even in dead calm weather—as for example may occur at night. A further refinement also enables him to separate the insects caught in each successive hour, so that the times of capture and the relative density for each hour of the day and night can be determined.

The majority of the insects captured in the traps are Aphidae and of these already over 60 species have been identified, of which about 20 species have been found at 1,500 feet or above. In addition to these, species of the following groups have also been observed :—Heteroptera, Homoptera, Diptera, Hymenoptera, Thysanoptera, Neuroptera, Coleoptera, Lepidoptera and Psocoptera.

Johnson's recent results indicate that although the density of insects (i.e., number in a given volume of air) is greater near the ground, the space above is so vast that on an average about 70 per cent of all the insects in the air are above 100 feet from the ground.

Turning away from experimental evidence to field observation we may quote the case recorded by Elton in 1924. He and his colleagues on an expedition to Spitzbergen found living Aphidae on the snow, up to several per square yard in places. The Aphidae were later identified as a species feeding on conifers and the nearest possible food supply was over 800 miles away in the mainland of Europe. At the same time that the Aphidae were found there were also seen a small number of hoverflies (Syrphidae) which are known to be predators on Aphids.

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Even in Britain there are many records of the air being full of Aphidae drifting on the wind and also cases when they have been washed up along the tide line of our shores in millions. These swarms are not infrequently accompanied by hover flies and lady-birds, both of which feed on Aphidae.

It is not easy in many of these records to distinguish between migration and drift—and indeed from the purely economic point of view it does not matter so much—but quite recently apparently true migration has been established in the hover flies, by the observations of Lack and others, of Syrphidae passing in very large numbers through passes in the Pyrenees from France to Spain in the autumn in two successive years.

For many years we have had the co-operation of the Masters of many of the Lightships off our coasts, and they have sent in many records and specimens of insects which have come aboard. Between 1933 and 1939 (Gibbs, 1942) ten Lightships, situated from 1 to 30 miles off our south-east and east coasts, sent in about 390 records, and included among these were 120 different species of Lepidoptera, of which about 20 were previously known or suspected to be true migrants As the majority of the species which could be identified belonged to the so-called " Macrolepidoptera "—the figures indicate that at least 10 per cent of our native moths are capable of flying or drifting over the water a minimum of 1 mile and frequently at least 30 miles. Fifty of the 120 species identified were seen on the Outer Dowsing light vessel, which is 30 miles east of Spurn Head in Yorkshire.

Palmèn (1948) records that he examined large numbers of beetles washed ashore in windrows on the coast of Finland. In nine such aggregations over 1,000 species of beetles were represented, some in countless thousands of individuals, and the vast majority were alive. These beetles had probably flown or been carried out to sea by winds, and then brought down to the surface of the sea perhaps by rain, and finally carried back to the shore by surface winds and waves. It is, of course, also possible that they might have been carried out to sea by some flooded river. In either case millions of individuals of hundreds of species have been distributed over quite large distances by wind or water currents and were still alive at the end.

Some years ago I visited the south-western corner of a large lake in Minnesota, U.S.A., just after a strong north-easterly gale. The water line and the shore line was alive with insects, several hundred per square yard, and among these were hundreds of Colorado beetles. These had undoubtedly been blown from the opposite shore which was about 30 miles away. There is little doubt that many of the occasional outbreaks of this insect in Britain are due to adults being blown over from the Continent.

Records and facts of this nature could be quoted almost without end, but enough has been said to show that most of our ideas on the distances that insects can travel—either on their own wings or on the wings of the wind—have altered very considerably in the

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last few decades, and may have to be altered still more as our knowledge of the extent of the movements increases.

We have to face the fact that every year millions of millions of insects are distributed by natural causes—migration or drift—over distances to be measured in miles, and not infrequently in hundreds of miles. Among these are some of the major pests of the world—and perhaps to compensate, a small sprinkling of beneficial insects and parasites.

There must follow from this a re-orientation of many old ideas linked up with the prevention and control of outbreaks of insects and of insect-borne diseases.

Firstly, in the case of rotation crops, already mentioned above, it is doubtful if moving a crop from one area to another within any distance less than a few miles, is of any value in the control of the majority of insect pests.

Secondly, the possibility of really exterminating an insect pest becomes untenable, unless it can be done over the whole area of distribution. If an injurious aphid—for example *Aphis fabae*—could be got rid of momentarily in England—say by eliminating its winter food supply—there would be still a constant source of re-infection each summer from the Continent.

Thirdly, the problem of keeping an area free from virus infection takes on new difficulties when we realize that millions of living Aphidae are regularly distributed over distances reaching hundreds of miles; and not to recognize this fact is only to live in a fool's paradise.

Fourthly, some of the activities of quarantine and port inspection of plant imports must be viewed in a new light when we realize that millions of insects per square mile are drifting across the man-made national frontiers, and even over the heads of the inspectors. Inspection is often of great value in preventing the introduction of entirely new pests by artificial means from distant countries, but can do little to reduce the total movement between adjacent areas.

Finally, the knowledge forces us to recognize the international responsibility and the need for co-operation in the control of migrant pests, and of those liable to extensive drift. In the locusts this has already been done to some extent, and with benefit to all: also to a smaller extent with the Colorado beetle situation in western Europe. But more co-operation is needed and Governments must recognize that they have a responsibility to see that insect pests do not leave their country as well as that they do not arrive in it from abroad.

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