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the inoculum would become most abundant as tubers reach maturity, when they are most susceptible to infection (Allen, 1957).

The lack of macroscopic symptoms on tubers at lifting is a difficulty in the detection and control of skin-spot disease. Assessment of the attack on the root system of crops grown for seed, or improved methods of direct examination for eye infections on seed tubers, may reveal stocks likely to become severely diseased. They may then be rejected as seed, or treated chemically at lifting and stored under conditions that prevent the development of severe symptoms on the eyes and skin surface.

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## EXPLANATION OF PLATES 3 AND 4

## PLATE 3

- Fig. 1. Stem bases of Majestic, showing cortical lesions due to natural infection by *Oospora pustulans*. (a) Slight; (b) moderate; (c) severe.
- Fig. 2. Severely infected stem base, showing cracked cortex.
- Fig. 3. *O. pustulans* sporing on an infected root after incubation.
- Fig. 4. *O. pustulans* sporing on infected stem base, showing branching habit.
- Fig. 5. Tap-root lesions on tomato seedlings grown in soil inoculated with *O. pustulans*.

## PLATE 4

- Fig. 1. Transverse section of uninfected part of potato stem base, showing normal cortical parenchyma.
- Fig. 2. Transverse section through lesion 1 in. below on the same stem, showing septation within parenchyma cells to form cork layer below superficial infection.
- Fig. 3. Cork formation in the outer layers of the phloem, where the cortex has developed a deep longitudinal crack.
- Fig. 4. Transverse section of infected stem base, showing mycelium in cortical cells.

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## Cereal Virus Diseases in Britain

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CEREAL VIRUS DISEASES of cereals and grasses have been described or identified in Great Britain during the last four years: cereal yellow mosaic, European wheat striate mosaic, rye grass mosaic and barley false mosaic. They are transmitted respectively by aphids, delphacid leafhoppers, mites, and seed and pollen.

## Cereal Yellow Dwarf Virus

At present the most important economically is cereal yellow dwarf virus. This was first described by Oswald and Houston in 1953 [4]. Attention was drawn to it by the apparently sudden appearance in the "best barley-producing countries" of an injurious disease of barley, whose principal symptoms were dwarfing of the plants and yellowing of the tips of the leaves. As with many viruses of field crops the serious outbreak of disease led to the identification of the virus. Once Oswald isolated it, he realized that it had occurred sporadically in cereal crops for many years, but the symptoms had not been distinguishable from the many other causes of yellowing in cereals. The first cereal virus isolated in Great Britain came from a few dwarfed plants in the headland of Long Hoos barley experiment at Rothamsted. They were selected because they seemed to resemble Oswald's description. They showed yellowing of the flag leaf and delay in emergence of the ear within it, in contrast to conditions such as nitrogen deficiency, or root fungus diseases which usually cause yellowing from the lower leaves upwards. However, this symptom is not always diagnostic, for it may occur with other nutrient deficiencies, e.g., potassium deficiency. The writer has seen it in potash deficient barley plots. The symptoms of cereal yellow dwarf are more easily diagnosed in oats, because of the bright yellow or purplish discoloration, but there is still uncertainty, especially as the virus is not always transmitted in glasshouse tests from field plants apparently showing characteristic symptoms of infection.

## THE FIRST SERIOUS OUTBREAKS

For a time, the study of yellow dwarf seemed to be a subject of purely academic interest, because for several years the only plants found were isolated specimens. Then came the mild winter of 1956 followed in 1957 by the earliest and largest spring migration of aphids ever recorded. Symptoms appeared much more plentifully in the spring crop. With the help of the advisory services, specimens were obtained from many parts of Great Britain, and a considerable number contained the virus. At least two specimens, one from North Wales and one from Kent

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Maisack, 1955). Mann & Smithies (1955a) suggested that these compounds arise by spontaneous

\* Part 2: Mann & Smithies (1955b).

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yielded much more virulent virus (YDV), than that collected at Rothamsted, and it was obvious that the disease was a potential menace to any crop that became infected in a young stage of growth.

There was not long to wait, for many winter crops, particularly oats in the south of England became severely infected during the winter of 1957-58, with very serious effects on yields.

Investigation of this disease has been curiously like that of beet yellows virus, which also was first isolated from a few yellowed plants on Rothamsted farm, where it appeared to be doing little harm to the crop. Observations made in the southern and eastern counties in 1940-42 gave no indication that beet yellows would ever become sufficiently widespread to cause serious damage [11]. At this time most of the virus isolated from the field was avirulent, but virus causing more virulent symptoms was isolated and replaced the mild virus in experimental work. The potential menace to the crop was then recognized, but it was not until 1943 that a serious outbreak actually occurred in the seed-crop growing areas of Lincolnshire. In 1944 and 1945 outbreaks increased in size and severity, and from that time only the virulent necrotic types of beet yellows virus could be isolated from the field; mild types were sought, but not found. In 1950 measures were taken to break the cycle of overwintering virus and spring infection of the root crop, by controlling the spread of virus in the seed crops. After this there were several years of reduced incidence of beet yellows, and in 1957 avirulent viruses again appeared in the field. Fifty-two out of 140 specimens collected in East Anglia contained them [6]. The same sequence may be occurring with cereal yellow dwarf viruses.

#### EFFECTS OF YELLOW DWARF ON WHEAT, OATS AND BARLEY YIELDS

Since 1955 small-scale field experiments have been made in which plots were infested with virus-carrying *Rhopalosiphum padi* [13] under muslin cages, and the yields compared with those of untreated plots or those fed on by virus-free aphids. The results for the first three years are summarized in Table 1.

Losses were very variable and sometimes negligible. This was partly a result of using the avirulent virus (YDA), and partly because it was not possible to infect each crop at the same stage of growth in each year. The results suggested that most of the crops and varieties tested could lose up to 30 per cent of their potential yield if infected at the right time, but we were not able to distinguish the critical times. The 1958 experiment compared both YDA and the virulent virus isolate, YDV, on infected and healthy crops of wheat, oats and barley (see Fig. 1 on p. 96).

Koga II wheat and Proctor barley infected with YDV at the earliest date lost two-thirds of their potential yield; Blenda oats lost four-fifths and probably would have produced no grain at all except that a few plants escaped infection. A few plants also remained healthy for a time

Table 1  
Losses of Yield caused by Avirulent Cereal Yellow Dwarf Virus (YDA)  
in Varieties of Wheat, Oats and Barley, tested 1955-1957.

	25-30 April per cent	3-4 May per cent	12-14 May per cent	
BARLEY				
Plumage Archer . . . . .	37	19	—	± 5.5
Maythorp . . . . .	24	—	20	± 11.2
Rika . . . . .	20	—	2	
Provost . . . . .	42	—	21	
Proctor . . . . .	26	—	8	
Herta . . . . .	0	-6	-7	± 3.9
Proctor . . . . .	16	-2	15	
WHEAT				
Atle . . . . .	3	—	33	± 11.2
Bersec . . . . .	14	—	34	
Koga II . . . . .	20	—	20	
Rescue . . . . .	11	—	16	
Atle . . . . .	-6	0	-5	± 4.9
Koga II . . . . .	-4	6	-9	
OATS				
Milford . . . . .	23	—	35	± 11.2
Onward . . . . .	5	—	12	
Blenda . . . . .	32	—	33	
Stormont Iris . . . . .	-4	—	19	
Milford . . . . .	17	17	16	± 7.1
Blenda . . . . .	37	27	22	

in the wheat and barley plots. YDA virus caused smaller losses, roughly comparable with those caused by YDV. There are several divergencies from expected trends in the data that may be of interest, but the full results and statistical analysis are not yet available.

#### GLASSHOUSE EXPERIMENTS WITH YELLOW DWARF VIRUS

Like beet yellows virus, the yellow dwarf viruses are persistent. The vectors probably remain infective for at least a week or ten days after a single adequate feed on infected plants. The most successful vector of both yellow dwarf viruses is *Rhopalosiphum padi*, which can infect 100 per cent of the plants fed on by single individual aphids carrying YDA, and 80 per cent if they are carrying YDV. *Sitobium fragariae*, *Trialeurodes avenae* and *Metopolophium dirhodum* transmit both with varying degrees of success. We have no evidence that either virus is specifically transmitted by different aphid species, as has been described for comparable isolates by Toko and Bruehl [10] in America.

Sixty-two species of Gramineae belonging to 14 genera have been tested for susceptibility to yellow dwarf in Great Britain and America. In the whole there is agreement between the two estimates of host range. All the cereals including maize and rice are susceptible, so are many species of Agrostidae, Hordeae, Phalaridae and Festucaea. The only species we have found to be almost certainly immune are *Agropyron*

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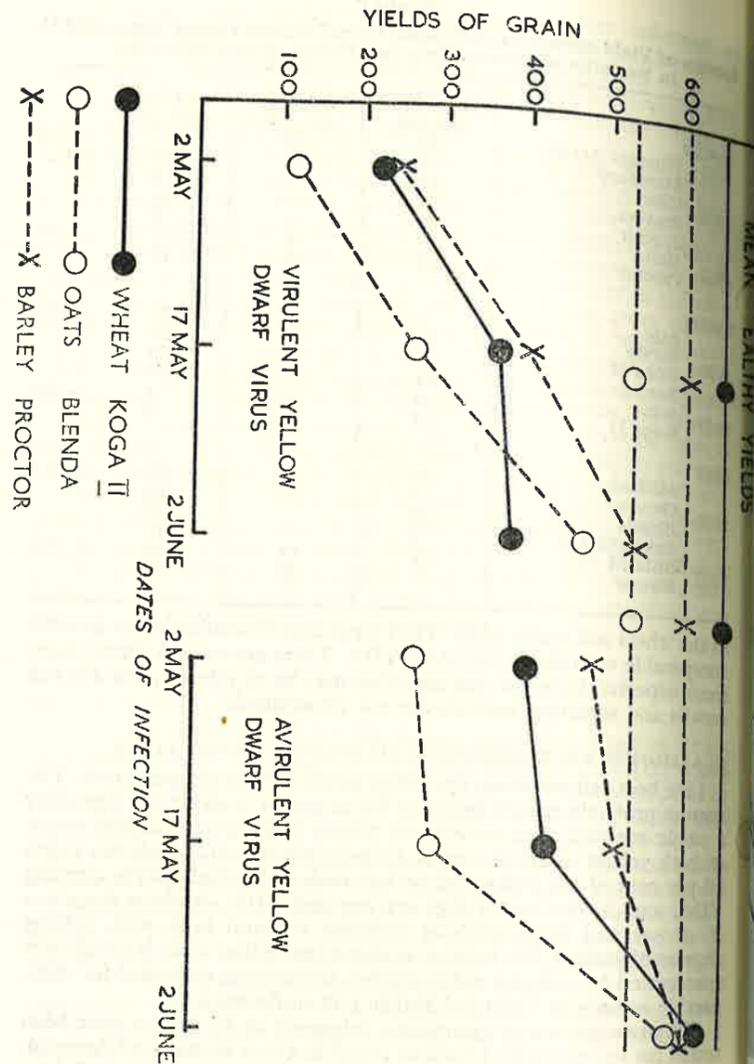
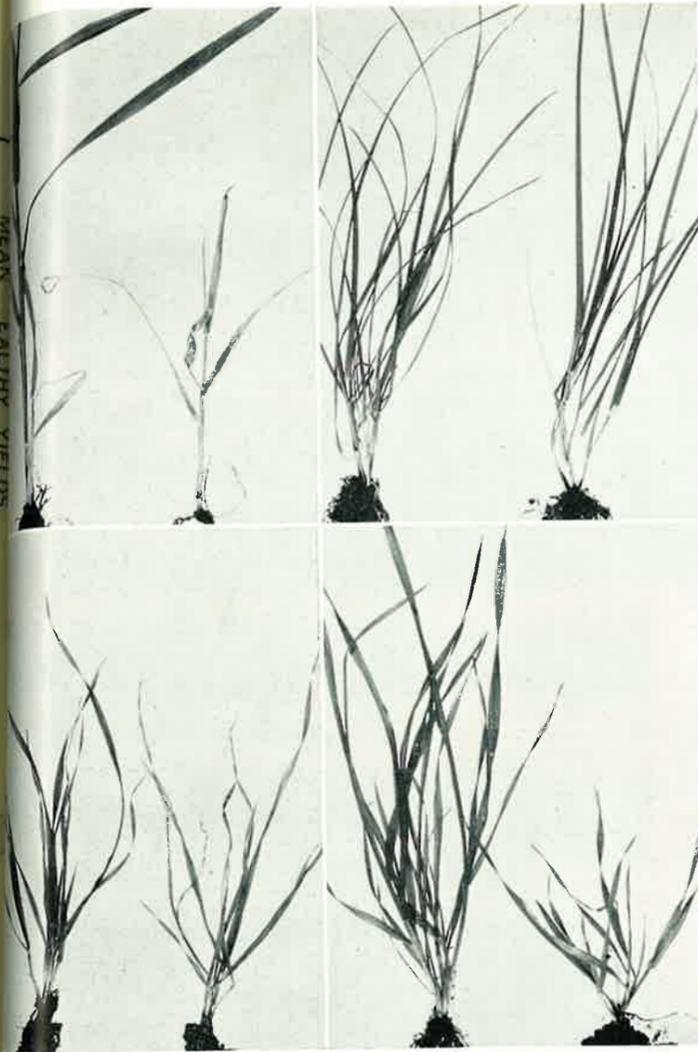


FIG. 1. Yields of wheat, oats and barley infected at different ages with avirulent and virulent strains of cereal yellow dwarf virus. Mean yields of uninfected plots are given as horizontal lines at the appropriate levels.



Marion A. Watson

SYMPTOMS OF VIRULENT CEREAL YELLOW DWARF ON OAT, RYEGRASS AND BROMUS SPP.

In each photograph, a healthy plant is shown on the left; an infected one on the right, the latter having been experimentally infected in the glasshouse.  
 Top left: *Avena sativa* var. Blenda. Top right: *Lolium perenne* var. S.23.  
 Bottom left: *Bromus sterilis*. Bottom right: *Bromus arvensis*.

PLATE I

(Maisack, 1955). Mann & Smithies (1955a) suggested that these compounds arise by spontaneous

\* Part 2: Mann & Smithies (1955b).

diaminobutane and 1:5-diaminopentane as enzyme substrates the amine aldehydes postulated as possible intermediates in the reactions would be  $\gamma$ -aminobutyraldehyde and  $\delta$ -aminovaleraldehyde.

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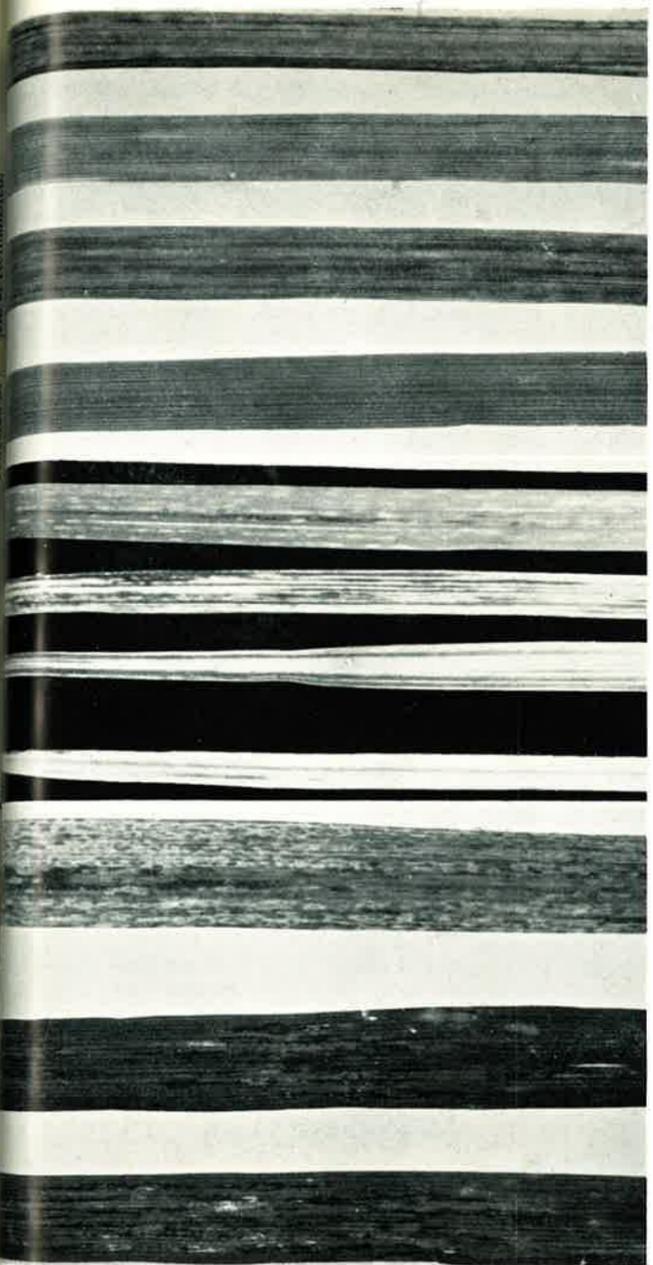
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RYEGRASS MOSAIC IN BLEND OAT  
Healthy plant on left; infected one on right.



STRIATE MOSAIC INFECTED WHEAT  
collected in field at Rothamsted.



Photos: Marion A. Watson

SYMPTOMS IN INDIVIDUAL LEAVES OF THE PRINCIPAL HOST PLANTS  
Wheat stripe mosaic in wheat.  
Barley stripe mosaic in barley.

PLATE III

Ryegrass mosaic in ryegrass.

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(Pers.), *Arrhenatherum elatius* (L.), and *Agrostis tenuis* (Sibth.).  
 with YDV some of these species seem to be symptomless carriers;  
 show no symptoms other than a slight reduction in size. Many  
 dwarfed and show typical yellowing, or red discoloration of the  
 leaves. Plate I shows two species of *Bromus*, perennial ryegrass, and  
 oat all infected at the same time with YDV. The infected ryegrass  
 is slightly "poorer"; the oat and *Bromus arvensis* are dwarfed, and  
*B. sterilis* shows very marked discoloration of the leaves, which is  
 a purplish red in the original.

Not many attempts have been made to recover the virus from  
 naturally infected grasses, but often their appearance in nature is  
 strikingly like that of plants infected experimentally. The red leaves of  
*B. sterilis* are so common that they seem a permanent characteristic of  
 the species, but in the glasshouse, healthy *B. sterilis* remains green and  
 only the infected ones turn red. We do not know the extent to which  
 wild and cultivated grasses act as reservoirs for the virus or what part  
 they play in transferring it to the cereal crops.

#### CROSS-INOCULATION EXPERIMENTS

No anti-serum has yet been obtained for YD viruses so we could not  
 use this method for determining their relationship with each other.  
 However, the symptoms of YDA and YDV are sufficiently different for  
 cross-inoculation experiments to be carried out, and, as with the yellow-  
 viruses of sugar beet, [12], these experiments gave evidence that the  
 two are not strains of the same virus but different viruses with similar  
 properties. Plants showing symptoms of YDA, when inoculated with  
 YDV, became just as dwarfed as healthy plants of the same age inocu-  
 lated at the same time, and virus recovered from these plants produced  
 typical YDV symptoms when re-inoculated into healthy seedlings.

Both yellow dwarf and sugar beet yellows viruses cause accumulation  
 of carbohydrate in the discoloured portions of infected leaves. Cereal  
 leaves do not usually form starch, but starch and reducing sugars are  
 found plentifully in YD infected wheat and oats. Sometimes this sugar  
 concentration is strong enough to be detected by taste, and it seems  
 possible that some laboratory test for the presence of virus might con-  
 ventionally be based on it. This would be extremely useful, for the disease  
 is not easy to diagnose by eye observation, and even aphid transmission  
 tests in the glasshouse are not always successful. Whether it is that  
 other conditions simulate virus, or that there are strains that are not  
 transmissible by our test insects, or that plants infected for a long time  
 in the field contain little virus, it is impossible to say.

#### Wheat Striate Mosaic

Wheat striate mosaic is transmitted by a fulgorid leaf-hopper,  
*Delphacodes pellucida* (Fabricius). Like yellow dwarf it is persistent and  
 non-sap-transmissible. The symptoms, shown in Plate III, centre, start

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with fine discontinuous streaks along the veins of affected leaves, rather like "tacking stitches" of fine thread. These broaden and coalesce until almost the whole leaf is yellowed, and dies. If infected within the first two months of growth all the leaves become yellowed and most plants are killed by the virus [9].

The photograph in Plate II, right, is of plants taken from the Rothamsted farm on 22nd May, 1958, when about 0.02 per cent of the plants were infected. A month later scarcely any infected plants could be found. Apparently, none had become infected at any later stage than those we had already seen, and those plants had died, or become smothered by their healthy neighbours. So there appeared to be less infection than at the previous time, and no evidence of spread from plants that were then showing symptoms. This suggested that the vectors had entered the crop carrying the virus but had not bred or multiplied on the wheat plants. The instance was not an isolated one. When the virus has been found in wheat, whether early or late infected, there has been no evidence of spread from the plants first observed. The virus has infected all the varieties of wheat, oats, barley, rye and ryegrass we have tested, and could undoubtedly cause serious losses to crops if it spread rapidly in the field. In 1957, Dr. Mary Glynne brought specimens from Spain that were found to be infected with WSM virus. Patches of similar plants were common in the crops that she observed, so spread of the virus does not seem so restricted there as here. It already occurs frequently in this country, although it infects few plants in any crop, and similar diseases have been recorded from other parts of Europe and from Russia [8].

#### WHY DOES THE VIRUS OFTEN INFECT ONLY A FEW PLANTS?

To find out how the virus so frequently enters the wheat crops and infects a few plants without spreading within the crop, we must consider its properties, and in particular, its relations with its insect vector. Like the aphid-transmitted viruses already mentioned, WSM virus is persistent and non-sap-transmissible. It resembles other leaf-hopper transmitted viruses in undergoing a long "incubation period" within the body of the vector between the time when the virus is acquired during feeding on infected plants, and the time when the insect becomes able to transmit. This interval can vary from about 7 to 30 days, and seems to be largely independent of the length of time that the vector has fed on infected plants, although it cannot be measured at all if they feed for too long.

*D. pellucida* exists in a number of races that vary in their ability to acquire and transmit virus; i.e., in the proportion of individuals belonging to a particular group or race that are able to infect in comparable conditions. We have three races of *D. pellucida* which regularly transmit to 90-100 per cent, 40-50 per cent and 10-20 per cent respectively of the plants tested, and we refer to them as highly efficient, efficient and inefficient races [14]. We have no information about the proportions

these different races that exist in field populations, but there is direct evidence that the least efficient may be in the majority. American workers have long maintained that the incubation period of multiplication of a virus within its insect host. There is little doubt that this is true of wheat striate mosaic in *D. pellucida*, as the efficient races of hoppers transmit the virus to a high proportion of their progeny. The virus enters the eggs of infective mothers, and if the mothers belong to a highly efficient vector race, the nymphs can transmit almost as soon as they are hatched. The virus is not transmitted by an infective male to the progeny of a virus-free female, but is inherited through the female parent only. Vector-efficiency can, however, be inherited through either parent. Egg transmission of the virus is proportional to vector efficiency. It is common among females of highly efficient races, and a large proportion of their nymphs, up to 100 per cent, may be infective. Egg transmission is very rare among inefficient races, even if the mothers are infective; indeed only one instance of egg transmission in a very inefficient race has been recorded.

#### EFFECTS OF CLIMATE

Both the multiplication of the virus in the vector and its inheritance through the egg are important to the dissemination and survival of the virus, and therefore of great economic importance. Hoppers are on the whole heat-loving insects. They flourish in warm dry climates, hence the spread of curly top virus of sugar beet in California, but in England they do not usually become very numerous on any particular crop except in occasional spells of warm summer weather. It is extremely doubtful whether *D. pellucida* could survive even a mild winter in the winter stage, but it probably does in the form of eggs laid in the leaf axils of hedgerow and meadow grasses. It is doubtful whether they could start to breed early enough in spring for wheat to become infected by the end of May, if the virus had to depend on being picked up by the next generation of hoppers feeding on meadow grasses. It is even uncertain how many of these grasses are susceptible to the virus; we are only sure of *Lolium* spp. However, a number of these eggs give rise to nymphs that are already infective, and whether or not they feed on infected grasses until they are adult, they will carry the virus with them when they migrate into the wheat crop. If they multiplied on the wheat the virus would spread, but it does not do so. Probably they feed only in sheltered places. When they move on to the young wheat they have little shelter, so they usually do not survive, although they might do so in a particularly fine and warm May and June. Presumably they do not survive in Spain, which is why the virus appears in large patches there only on isolated plants here.

#### Ryegrass Mosaic

The writer is indebted to Mr. T. Mulligan for the substance of the following notes on ryegrass mosaic which was isolated by Dr. Slykhuis

(Malsack, 1955). Mann & Smithies (1955a) suggested that these compounds arise by spontaneous

\* Part 2: Mann & Smithies (1955b).

diaminobutane and 1:5-diaminopentane as enzyme substrates the amine aldehydes postulated as possible intermediates in the reactions would be  $\gamma$ -aminobutyraldehyde and  $\delta$ -aminovaleraldehyde.

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from cultivated grasses in Hertfordshire in 1956. It was found to be sap-transmissible, but not transmissible by aphids or by leaf-hoppers. As the virus in some ways resembled wheat streak mosaic [7], a collection of mites was made, several of which were found to belong to the Bryophyid group. These successfully transmitted the virus to a small proportion of ryegrass seedlings, and eventually the vector was identified, by a process of elimination, as *Abacarus hystrix* (Keifer). This is a small elongated translucent mite, about 0.2 mm long, that lives in the grooves on the surfaces of grass leaves. Its eggs hatch into active nymphs resembling the parents. The life history probably takes about three weeks. Several species of these mites appear identical, even under the microscope, and distinctions between them depend on minute characteristics, so it is not easy to obtain a pure culture, or to replace them if it fails.

The mites have not much mobility, and the virus is probably spread by wind dispersal of infective vectors [7], though infested leaf fragments could also be carried by machinery or grazing animals.

Some of the properties of the virus have been determined by sap inoculation. It is fairly stable at room temperature and serologically active. It has a wide host range among the Gramineae, but no dicotyledonous hosts. The host range is difficult to determine precisely, because in several genera of grasses some species are susceptible and others are not. In some species varieties respond similarly, and a species described as immune, may later be found to have susceptible varieties.

#### SUSCEPTIBLE PLANTS

Of the cereals, oats (Plate II, left), and rice are susceptible but not wheat, barley or maize. The virus was isolated from some naturally infected oat plants sent by Mr. E. Lester from Berkshire. They had been collected in the field because of their severe necrotic symptoms. It is common in old meadows or grass leys, particularly of Italian or perennial ryegrass (Plate III, left), which are the most favourable hosts for the virus. *Festuca pratensis*, *Cynosurus cristatus* (L.), *Poa annua* and *Dactylis glomerata* (L.) are important susceptible grasses.

Ryegrass plants inoculated as seedlings in pots in the glasshouse lost 30-40 per cent of their yield compared with healthy plants, and plants infected in the glasshouse and grown outside sometimes form rather poor tussocks, but vary in their appearance. So there is little information about possible damage that the virus might cause to hay crops or grass leys.

Probably the reason for varietal differences in response to infection is that there has already been selection for resistance, susceptible plants in breeding material having been eliminated because of their poor appearance when infected.

#### Barley Stripe Mosaic Virus

Barley stripe mosaic virus [3], has only once been isolated from a crop

in Great Britain. This was Gloire du Velay barley grown from seed imported from France. Its presence was suspected in a variety "Olli" imported from Canada, but could not be confirmed.

The symptoms (Plate III, right), vary on different varieties of barley from a mild chlorotic mottle to a severe necrotic streak that resembles the "stripe" caused by *Helminthosporium sativum*; for this reason it was at first called barley false stripe. The characteristic symptom in very susceptible hosts is a chlorotic streak bordered by a narrow region of bronze necrosis, which eventually spreads and kills the

plants. Kassanis and Slykhuis [1], from whom most of this information was obtained, tested twenty varieties of barley. The least tolerant were the barley selections from the Plant Breeding Institute at Cambridge, H.B. 117/23/2, and H.B. 169/5/22/6. These became 100 per cent infected, with severe necrotic symptoms. The most tolerant were Rika, Archer, Plumage Archer and Maythorp, which gave mild symptoms in only about 10 per cent of the plants inoculated. Of other varieties commonly grown in England, Proctor was moderately tolerant and Provost moderately susceptible. Some wheat varieties and Italian ryegrass were also found to be susceptible.

BSM virus is the only one of the four cereal viruses here described that has dicotyledonous hosts. Kassanis and Slykhuis found that sugar beet and *Chenopodium amaranticolor* developed chlorotic lesions on the lower leaves, but did not become systemically infected; summer cich (Spinaceae oleracea) developed systemic symptoms of a bright yellow mottle in the form of rings, or "oak-leaf" patterns.

The virus was found to be rather stable in extracted sap, infective dilution of around 1/2000, and serologically active. Rod-shaped particles were observed in electron micrographs, and were much more numerous than the infectivity and serological dilution end points would suggest. BSM virus is transmitted through the seed of infected barley plants, and with susceptible varieties a high proportion of the progeny is infected. It also possesses the unusual property of being transmitted through the pollen, and virus particles have been seen in electron micrographs of preparations made from pollen grains collected from infected plants [2].

The virus does not seem to be a problem at present for cereal growers, but it is something to be watched for, particularly in cereals grown from imported seed. According to American and Canadian workers the disease can cause very serious losses in barley and other cereals. It is potentially dangerous in breeding stocks in this country, but plant breeders are now on their guard against it, and will be taking all possible precautions.

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### Plant Enzyme Reactions Leading to the Formation of Heterocyclic Compounds

#### 3. PLANT AMINE OXIDASE AND THE FORMATION OF PYRROLIDINE AND PIPERIDINE ALKALOIDS\*

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The products of the oxidation of 1:4-diaminobutane and 1:5-diaminopentane, catalysed by plant amine oxidase in the presence of catalase, accumulate in the reaction mixtures as compounds of  $\Delta^1$ -pyrroline and of 2:3:4:5-tetrahydropyridine respectively (Mann & Smithies, 1955a; Hasse & Maisack, 1955). Mann & Smithies (1955a) suggested that these compounds arise by spontaneous

cyclization of the aldimines, which are probably the primary products of the enzyme reactions, or of the amine aldehydes formed from the aldimines by spontaneous reaction with water. Thus with 1:4-diaminobutane as substrate, the suggested reactions were those given in Scheme 1. With 1:4-diaminobutane and 1:5-diaminopentane as enzyme substrates the amine aldehydes postulated as possible intermediates in the reactions would be  $\gamma$ -aminobutyraldehyde and  $\delta$ -aminovaleraldehyde.

\* Part 2: Mann & Smithies (1955b).