CHAPTER 10

Plant Health and the Possibilities of Biological Control

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PLANT HEALTH

This review is based on some 80 papers (66 quoted) from Australia, Canada, Denmark, England, Finland, France, Germany, Japan, Sweden, USA and Wales. Those omitted either did not mention wild oats in their host-lists, or repeated information published elsewhere. Specialist assistance in compiling the list of references was willingly given by many present and some former workers at Rothamsted Experimental Station and at the MAFF Plant Pathology Laboratory, Hatching Green, Harpenden*, but the reviewers accept responsibility for the final choice and for the comments on them. Grateful acknowledgement is due to Dr. L. Kiewnick, of the Landwirtschaftskammer Rheinland, Bad Godesberg, for permission to draw extensively on his data.

There is no previous review of pests and diseases attacking wild oats, but Dadd (1957) and Thurston *et al* (1970) list some of the organisms concerned which were known at the time.

As with weeds, the nomenclature of pests and pathogens keeps changing, with the result that an organism called by its currently-valid name in this review may appear under a different one in the references quoted. Where the older Latin names are the better-known ones, they have been added in brackets. Names of diseases caused by the organisms seem a little more stable,

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but are not necessarily internationally uniform. Those used here are widely understood in England.

Although most wild oat plants appear healthy, they can be attacked by a number of pests and diseases. There does not seem to have been any systematic study of growing wild oat plants to see what pathogens affect them. All the records available have been obtained in the course of studies of the alternative hosts of economically-important pests and diseases of crops, especially of cereals. It follows that there is little hope of using the known pests and diseases to control wild oats. The main interest is in finding out how important wild oats are in the carry-over of pathogens from one susceptible crop to another. There is little qualitative evidence and no quantitative information for any pests or diseases on wild oats in the field. It does not follow that, because a certain species of crop pathogen is also found on wild oats it will necessarily transfer from one to the other, because some, particularly the fungi and nematodes, can have within the species physiologically distinct but morphologically indistinguishable host-specific races.

Because of the way the evidence has been obtained, it is possible that wild oats may, at least under certain circumstances, be susceptible to pathogens not yet reported to attack them and which are not harmful to crops. Conversely, reliable information on immunity is only available where deliberate attempts at inoculation or infestation have failed with wild oats and succeeded with other species by the same methods in the same experiment.

These investigations have led to the use of several wild *Avena* spp. as sources of genes conferring immunity to pests and diseases, notably cereal root eelworm and mildew, for use in breeding resistant varieties of cultivated oats (*Avena sativa*).

The relationship between seed- and soil-microflora and the viability and dormancy of *Avena fatua* seeds has been studied by Kiewnick (1963, 1964). Some of the fungi concerned are known pathogens of cereals.

INVERTEBRATES ATTACKING WILD OATS

NEMATODES

Weeds sometimes show damage by nematodes, but there is no evidence that nematodes affect the size of weed populations (Franklin 1970). Goodey *et al* (1965) listed the first records then known of nematodes on host plants including *A. fatua* and *A. sterilis.* At least four nematodes are associated with stems or roots of wild oat species.

Heterodera avenae (= *H. major*), the cereal root eelworm, is by far the most studied, with an almost worldwide distribution and seven main races or undescribed new species differing in their host range (Jones 1972). Some countries have several races and others only one. The search for resistance genes for incorporation in *A. sativa* has led to widespread testing of *A. sterilis,* especially of two resistant strains orginally discovered in Denmark (Andersen 1961). These cannot have been native and their origins and descriptions are

not given. Selections of *A. sterilis* differ in susceptibility to strains of *H. avenae* (Davies and Griffiths 1962, Fiddian and Kimber 1964, Kort *et al* 1964) but useful resistance has been shown to nematodes present in England and Wales (Davies and Griffiths 1962, Fiddian and Kimber 1964, Cotten 1963, 1967), Australia (Brown 1969, Brown and Meagher 1970), Poland (Wilski 1972), Netherlands (Kort *et al* 1964), and presumably India (Gill and Swarup 1971) though the sources of nematodes and *A. sterilis* used in laboratory tests are not stated in this paper. *A. sterilis* is a hexaploid and thus convenient for crossing with *A. sativa,* and one of Andersen's (1961) selections (I. 376) under its Aberystwyth number Cc 4658 has been used in breeding *A. sativa* resistant to all the races of *H. avenae* so far encountered in Britain (Cotten 1969). Resistance is controlled by two major genes, with resistance incompletely dominant. There do not seem to be any exhaustive tests of native *A. sterilis* against native *H. avenae* in countries where both occur, eg Brown (1969) in Australia tested only *A. sterilis* from the Netherlands, and from Denmark via Wales, against native eelworms.

Avena ludoviciana, according to Malzew (1930) a subspecies of *A. sterilis,* was not resistant to *H. avenae* in Wales (Davies and Griffiths 1962) nor in Australia where Brown and Meagher (1970) tested three types. *A. fatua* was not included in many of the host-range tests with *H. avenae,* but it has been found attacked by the cereal root eelworm in Wales (Thomas *et al* 1946) Denmark (Petersen 1961) and West Australia (Parkin and Goss 1968) and one sample tested in Victoria, Australia was very susceptible (Brown and Meagher 1970).

Populations of *Heterodera avenae* may increase far more rapidly on wild oats than on susceptible wheat or barley varieties. In microplot studies of population dynamics, six successive crops of the resistant oat cv. Avon produced no significant increase in the nematode population. In the same period, populations increased 14 or 15-fold under barley cv. Prior or wheat cv. Olympic, but *A. fatua* required only two successive seasons to achieve the population level reached by Olympic after six successive sowings (Meagher and Brown 1972). The role of *A. sterilis* in transmitting cereal root eelworms from one susceptible crop to another, or in permitting them to multiply, has not been studied.

Ditylenchus dipsaci, the stem and bulb eelworm, one of the most devastating plant parasitic nematodes, especially in temperate regions, has been found on *Avena fatua* and *A. sterilis* in USA (Steiner and Buhrer 1932) and in Germany (Salentiny 1959). In Britain *A. fatua* is recognised as an important alternative host of *D. dipsaci,* enabling the eelworm to survive from one susceptible crop to the next. Wild oat control is therefore important (MAFF 1966) for control of *D. dipsaci.*

Meloidogyne incognita, the root-knot nematode, of major economic importance throughout the tropics and warmer regions of the world, was able to infest *A. fatua* in a greenhouse test of susceptibility of common weeds of Indiana (Gaskin 1958), although wild oats were not among the most severely-affected species. *Pratylenchus minyus* has been found on wild oats (presumably *A. fatua*) in Ontario (Baker 1955).

APHIDS

Wild *Avena* spp. occur in some lists of aphid food-plants, eg Patch (1938) refers to four aphid species found on *A. fatua,* one of them also on *A. sterilis* and another also on *A. barbata* (without specifying the weed *A. barbata* Brot., or *A. barbata* Pott). However, other *Avena* spp. would probably be attacked by any aphids commonly found on cultivated oats and there are about ten of these in Britain (Empson 1965).

Aphids (often brownish) clustering around the bases of spikelets on ripening ears, probably the English grain-aphid *Macrosiphum (Sitobion) avenae* (F.), are believed to do little damage when they occur on cultivated oats; their direct effect on wild oats has not been investigated, but they can transmit virus (see under Viruses, p. 224).

Greenish aphids on the leaves are probably either the bird-cherry aphid *Rhopalosiphum padi* (L.) or the rose-grass aphid *Metopolophium dirhodum* (Wlk.). *R. padi* overwinters on grasses in southern England where its winter host, *Prunus padus,* does not grow wild, and could thus easily transmit viruses. *M. dirhodum* transfers from roses to Gramineae in late April, and, although it would not bring in cereal viruses, it is suspected of transmitting them between plants within a crop.

In Finland, *R. padi* multiplied at least as freely on *A. fatua* as on *A. sativa,* at least twice as well as on any of the 34 other grasses, 16 Cyperaceae and 10 Juncaceae tested (Markkula and Roukka 1972). The results were the same for all strains of aphids. *M. avenae* was tested similarly, but results are not presented for *A. fatua.* In this paper, the captions for Figures 2 and 3 were inadvertently reversed: Figure 3 showing *A. fatua* is for *R. padi.* The text is correct.

FRIT FLY

Oscinella frit damages cultivated oats at three stages. First the shoots of young plants are attacked by larvae, and the centre leaf dies. Next, ears may be twisted and distorted, with white, withered spikelets. Finally the grain may be reduced to a fine black powder, with an insect puparium instead of an oat embryo. The timing for grain-damage is crucial, as the oats are susceptible only for a short time after the ears have emerged from the leaf-sheath. After flowering, oats are resistent to frit-fly (Empson 1965). Wild oats are affected in the same ways, but research workers most often notice the attacks on the grain, causing up to 30% of non-viable seeds; sometimes the small black flies hatch out in the bags containing affected seed-samples.

Cunliffe (1929) found that *A. fatua* sown in Sweden on 25 April had 22% of primary shoots and 30% of total shoots attacked by frit-fly, but if sowing was delayed until 23 May when the first fly was seen in the field, 44% of primary and 37% of total shoots were attacked. This placed *A. fatua* at both sowings in the same category as the most susceptible of the 32 cultivars tested at the same times.

OTHER INSECTS

Some insects which might be expected to attack wild *Avena* spp. but do not seem to have been recorded on them by entomologists or weedworkers are thrips *Limothrips cerealium* and *Stenothrips graminum*, oat stem midge *Mayetiola avenae* and the stem-borers *Opomyza florum* and *Geomyza* spp. The slug-like grub of the cereal leaf beetle *Lema melanopa* is occasionally seen clinging to wild oat leaves.

DISEASES

FUNGI

Three categories of fungi are associated with wild oats. Two of these are definitely pathogens. First and most obvious are those attacking the above ground parts of the growing plant, eg *Erysiphe graminis* causing mildew, *Hymenula cerealis* (=*Cephalosporium gramineum*) causing leaf-stripe, *Puccinia* spp. causing rusts and *Ustilago* spp. causing smuts. Second, there are those causing foot-rots, eg *Gaeumannomyces graminis* (formerly *Ophiobolus graminis*) causing take-all and *Cercosporella herpotrichoides* causing eyespot. The fungi of the third group are associated with seeds in the soil and may be seed-borne or soil-borne. Some are well-known pathogens but others are weak parasites or saprophytes.

Erysiphe graminis is sometimes seen causing mildewed leaves of *A. fatua* and *A. ludoviciana* in the field (Reed 1920). It is also a major hazard of pot-grown wild oats in glasshouses and growth rooms, but can be controlled by mixing ethirimol in the soil when the pots are filled (at about 20 mg a.i. per kg air-dry soil) or by spraying the growing plants with benomyl. Strains of *Erysiphe graminis* show considerable host specificity. Those attacking *Avena* spp. are not usually the same as those found on wheat or barley (Marchal 1902) or grasses (Frauenstein 1970). Selections of *A. ludoviciana* resistant to strains of *Erysiphe graminis* commonly attacking *Avena* spp. have been found (eg Thurston 1957) and the genes for resistance can be incorporated reasonably easily into cultivated oats as both are hexaploids (Lawes and Hayes 1965, Degras 1966), but virulent strains of oat mildew able to overcome this resistance have been found (Hayes and Jones 1966).

Hymenula cerealis (=*Cephalosporium gramineum*) was found causing leaf-stripe on wheat and *A. fatua* in Japan (Nisikado *et al* 1934), although the lesions were less distinct on the wild oat than on wheat. Howell and Burgess (1969) found that *A. fatua* grown in infected soil in England showed leaf-stripe symptoms similar to those on cultivated oats. Infected culms were dwarfed and the inflorescences died prematurely. Naturally-infected *A. fatua* have been associated with leaf stripe in cereals grown under minimum cultivation (Howell 1969) (see also fungi associated with seeds, p. 217-22).

Puccinia spp. (rusts) are seldom reported on wild *Avena* spp. although they cause serious diseases of cultivated oats. Wild oats, especially the hexaploid species which cross easily with *A. sativa*, have been tested for resistance to

Canadian races of crown rust (*Puccinia coronata* f. sp. *avenae*) and stem rust (*Puccinia graminis*). Dinoor and Wahl (1963) tested 202 collections of wild oats (*A. sterilis* L. and *A. barbata* Brot.) from Israel. Nine *A. sterilis* were resistant to crown rust in the field but none resisted stem rust. Eight *A. barbata* resisted crown rust and eight stem rust in the field, but crown and stem rust resistance factors were independent. One selection of *A. barbata* (D.203) showed outstanding resistance. Fleischmann (1970) carried this work one stage further, examining 12 lines of oats containing resistance from *A. sterilis,* collected in Portugal, Tunisia and Israel. The lines contained two genes, and three wild oat collections gave effective resistance to nearly every culture of crown rust. The rust cultures from Western Canada were more virulent than those from the east to oats containing the *A. sterilis* resistance. Reed (1920) in Missouri inoculated *A. fatua* with uredospores of *Puccinia coronata* from *A. sativa* and got 100% infection, but only 70-100% of *A. sterilis* and 32-100% of *A. ludoviciana* plants developed rust, ie there were differences in susceptibility but no immunity in his material.

Ustilago avenae, causing loose smut of oats, attacks *Avena fatua, A. ludoviciana* and *A. sterilis* ssp. *macrocarpa* (Vavilov, quoted in Malzew 1930). Reed (1920) says it has been recorded on *A. fatua* in USA (California only) and in Denmark, on unspecified wild oats in Australia (where McAlpine 1910, transferred it experimentally from wild oats to *A. sativa* and *vice versa* using spores) and on *A. sterilis* in Germany. This German record may, however, refer to cultivars of *A. sterilis* (lacking the abscission-scar at the base of the spikelet), as the weedy forms are very rare there.

Ustilago levis causing covered smut does not seem to have been found occurring naturally on any wild oats, but in inoculation tests over several years Reed (1920) got 17-61% of plants of *A. fatua* infected, 0-56% *A. sterilis* (mainly cultivars) and 0-21% of *A. ludoviciana.* In some seasons the proportion of plants of all wild oats which took the infection was far below average.

Gaeumannomyces graminis (formerly *Ophiobolus graminis*) causing take-all of cereals usually attacks wheat or barley, but there is a less common variety *avenae* which attacks cultivated oats. A constituent of oat sap inhibits isolates from wheat (Turner 1956). Thus the fact that Canadian isolates from wheat and *Agropyron* did not attack *A. fatua* (Padwick and Henry 1933) does not rule out the possibility that wild oats might be susceptible to var. *avenae.* Indeed, Nilsson (1969) quotes two instances of *A. fatua* being attacked and one where, although usually considered immune, slight attack could occur. Similarly, *A. sterilis* was attacked in two investigations and appeared slightly affected in a third.

Fusarium graminearum, another fungus causing foot-rots of cereals, has been found capable of attacking *A. fatua* in Canada (Padwick and Henry 1933) and the fungus was re-isolated from the lesions (see also *Fusarium* spp., p. 217-22).

Helminthosporium sativum also appeared to cause foot rot in *A. fatua* but the fungus could not be re-isolated from the lesions (Padwick and Henry 1933) (see also p. 217-22).

Cercosporella herpotrichoides, causing eyespot of wheat, has been shown to attack experimentally-inoculated *A. fatua*, but infection varied from 10.5 to 73.7% of the treated plants, in experiments on different dates, compared with 48.3 to 88.0% of wheat. In an experiment using different conidial concentrations as inoculum, *A. fatua* was less susceptible than wheat, only 10% of plants becoming infected at 2.10^6 compared with 84% of wheat at the same concentration (Hartz 1969).

Fungi associated with seeds of Avena fatua were studied by Kiewnick (1963, 1964) and Rademacher and Kiewnick (1964). Previously, Kommedahl *et al* (1958) had found *Alternaria* sp. and *Helminthosporium sativum* associated with seeds, but produced no evidence of pathogenicity to them. The first study (Kiewnick 1963) was of the numbers, species and properties of micro-organisms occurring on seeds of *A. fatua* ssp. *fatua* var. *pilosissima* collected in two contrasting years from eight places in Germany. He found more micro-organisms present in the wet year, with the greatest effect on bacteria, giving a maximum of over 37 million bacteria/g of seed and over 43 million micro-organisms altogether. The maximum number of fungi was only $9\frac{1}{2}$ million but the plating-out method used for counts favours bacteria and yeasts where every cell can start a colony; fungal mycelia are less easily fragmented. Yeasts and actinomycetes were usually less than 500,000/g but the maximum for yeast was nearly 3 million/g. The number of micro-organisms varied with the crop, most in wild oats out of spring barley and least from cultivated oats; although in oats the fungi formed a higher proportion. He identified 52 species of fungi on German wild oat seeds, 45 of them Fungi Imperfecti. English seeds yielded only 12 species, including three *Fusarium* spp. Swedish seeds had only five fungi, *Stemphylium, Cladosporium, Humicola* and two *Fusarium* spp. but not *Alternaria* or *Cephalosporium.* Czech seeds resembled the German in fungus flora with much *Chaetomium* and *Cladosporium*, followed by *Stemphylium, Alternaria, Humicola* and *Stachybotris*, with *Fusarium* spp. frequent.

He tested 36 fungi for pathogenicity to wild oat seeds, because many were known only as saprophytes. *Stemphylium consortiale* was the most damaging, causing 32% dead seeds, followed by *Helminthosporium gramineum* and *Hymenula cerealis* (=*Cephalosporium gramineum*) (29% each), *Fusarium culmorum* (26%) and *F. solani* (24%). These five fungi all cause diseases of cereals although *Hymenula cerealis* is usually considered to infect only barley. The next in order of pathogenicity, *Phoma hibernica* (23% dead seeds), was almost as damaging as the known pathogens.

The fungus used for the inoculation was recovered from dead seeds, but mycelium was not found in partly-damaged seeds with discoloured embryos. *Fusarium culmorum* was the most aggressive to roots; affected plants produced deformed ears and sterile seeds.

The relationship between air humidity, seed moisture-content, viability, and numbers of fungi and bacteria was investigated at laboratory temperatures (Kiewnick 1963). The uncontrolled relative humidity (RH) fluctuated between 60 and 80% and controlled relative humidities between 75 and 100% were compared with this. Kiewnick's data, reproduced in Table 10.1 in slightly

Table 10.1 *Effect of storage under different relative humidities on germination, water content and number of micro-organisms on* Avena fatua *seeds (after Kiewnick 1963, Fig 4).*

Air humidity %	% germination after 3 months	Number (thousands) Fungi	Bacteria	% germination after 6 months	Number (thousands) Fungi	Bacteria	% water content of seeds after 3 months
Uncontrolled	86	800	22,560	76	350	22,040	8.8
70	86	25	26,400	77	20	31,600	11.6
75	78	55	12,200	79	75	24,400	12.0
80	75	200	21,000	77	150	30,400	12.2
85	78	1360	17,800	76	440	35,600	14.6
90	75	2800	17,450	73	6,550	22,650	16.8
95	74	5500	19,750	70	14,000	39,500	26.0
97	67	5500	42,000	50	16,700	72,000	26.2
100	66	6950	59,700	49	34,750	119,400	27.0

modified form, show that viability was maintained better at lower RH values, whereas populations of fungi and bacteria generally increased with increasing humidity. The exception to this was the set in uncontrolled humidity where numbers of fungi and bacteria exceeded those for constant 80%, the highest humidity reached in fluctuations. The sharp drop in viability after six months at 97 and 100% RH is parallel to the steep rise in numbers of bacteria, up to six times as many in 100% RH as where it was uncontrolled. The increase in fungi, also greatest at 100% RH, had begun at 95%, which had little effect on viability at six months. Similarly, the moisture-content of the seeds rose sharply between 90 and 95% RH. Evidently, the bacterial counts were more closely associated with decreased viability than either numbers of fungi or moisture-content of seeds, but this might be either the cause or the effect of death of the seeds. The bacteria were not identified, but the fungi at lower humidities were the xerophytic *Penicillium* and *Aspergillus*, and at higher humidities *Cladosporium*, *Verticillium*, *Hymenula (Cephalosporium)* and *Fusarium* spp. The fungi were 100 times more numerous at 100% RH than in the uncontrolled conditions, ie they showed a greater increase than the bacteria. Kiewnick suggested that toxins produced by the fungi may have killed the seeds.

In the second paper Kiewnick (1964) dealt with the influence of the microflora of the seeds themselves and of the soil in which they were sown, on the longevity and dormancy of *A. fatua* seeds in soil. Seeds, either unsterilised or surface-sterilised, were sown in pots of unsterilised or steam-sterilised field soil, giving four combinations of treatment. From these, the relative importance of seed-borne and soil-borne micro-organisms for survival and dormancy of the buried seeds could be deduced (Table 10.2).

In a footnote, Kiewnick states that in the three-year totals, 78 differs very significantly from 34 and 60 but not from 64. Replication is not mentioned.

Kiewnick's attempts to explain what is happening are complicated and involve reference to Garrett's (1956) discussion of partial sterilisation of soil. In Table 10.2, the two effects of microflora, on (1) viability and (2) dormancy,

Table 10.2 *Germination of* A.fatua *as % of seeds sown (after Kiewnick 1964, p. 33).*

	Both seeds and soil sterilised	Seeds only sterilised	Soil only sterilised	Neither seeds nor soil sterilised
1st year	49	42	8	6
2nd year	26	20	52	28
3rd year	3	2	0	0
Total for 3 years	78	64	60	34
Total as % of 'both sterilised'	100	82	77	44

are considered together, one partly obscuring the other. It is simpler to consider first the effect on viability, and to postpone discussion of dormancy until later.

Only 78% of the seeds sown gave seedlings, even with both seeds and soil sterilised, so this is the viability of the seed-sample used. Therefore, ignoring those seeds which were incapable of germinating, the total germination in each treatment can be expressed as per cent viable seeds, as in the bottom line of the table. Thus soil micro-organisms have decreased germination to 82%, ie have killed 18% of the viable seeds. Similarly the soil microflora killed 23%. Both acting together killed 44%, a very similar result to 18 + 23 = 41% for either acting alone. This suggests that seed-borne and soil-borne fungi are acting on different seeds, although we might have expected weak seeds to be attacked by both. Kiewnick does not make this deduction. He identified the seed-borne fungi (Kiewnick 1963) but not the seed-borne bacteria or any of the soil micro-organisms so we cannot yet discover whether different organisms were acting on different seeds, nor which soil-borne ones were active against seeds of *A. fatua.*

Kiewnick concludes that destruction of seeds by micro-organisms is a useful part of wild oat control and should be encouraged, but points out that as the microflora is determined by the environment, some alterations to it, eg ousting the antagonists, altering the pH, would be necessary. Obviously this could best be done if the imporatant organisms and their requirements were known.

Turning now to the effect of the microflora on dormancy of *A. fatua* seeds buried in soil, it is convenient to convert Kiewnick's data to germination percentage of the viable seeds surviving in each treatment, as in Table 10.3. Eliminating those seeds which never germinated, brings the figures in the last column (neither sterilised) in line with those for only soil sterilised, in contrast to Kiewnick's table. Evidently sterilising the seeds greatly stimulated germination in the first year, and also prolonged the life of 3-4% of these seeds into the third year, whereas sterilising the soil had little effect on seed-dormancy and there was no interaction between seed and soil sterilisation.

Two explanations are possible for the dormancy-breaking effect of seed-sterilisation. Either some or all of the seed-borne microflora can induce

Table 10.3 *Percentage of the total viable seeds of* A. fatua *in each treatment, germinating in each year (after Kiewnick 1964).*

	Both seeds and soil sterilised	Only seeds sterilised	Only soil sterilised	Neither seeds nor soil sterilised
1st year	63	66	13	18
2nd year	33	31	87	82
3rd year	4	3	0	0

dormancy, or they antagonise all or part of the soil micro-flora which can break dormancy. Further investigation of this point with its practical implications for wild oat control seems desirable.

In an experiment with soil moisture content 20, 50 or 80% of field capacity, and seeds inoculated with the five pathogenic fungi *Fusarium culmorum* and *F. solani*, *Stemphylium consortiale*, *Hymenula cerealis* (=*Cephalosporium gramineum*) and *Helminthosporium gramineum*, the fungi were most aggressive at 50% (Kiewnick 1963). At 20% the soil was too dry for germination, and seeds remained dormant. At 80% more germinated, but very few survived as dormant seeds. This could explain the results of Kott (1955) who found that seeds survived only $1\frac{1}{2}$-2 years in soil in the Moscow region and the Urals with precipitation over 600 mm/yr, but in the forest region (less than 500 mm/yr) they lasted two to three years and in South Russia (250-400 mm/yr) they survived for three to eight years in soil.

Soil type, particularly as it affected moisture-retention and aeration, also influenced seed survival and speed of germination. Slower germination was detrimental to seed-survival, presumably because the micro-organisms had more time to attack the seeds.

Soil temperature, although less important than moisture, also affected inoculation experiments with fungi. Rising temperatures increased aggressiveness in *F. solani* and decreased it in *F. culmorum*. *Stemphylium, Hymenula (Cephalosporium)* and *Helminthosporium* were most infective at 25°C but the *Fusarium* spp. were very active at 15°C.

Rademacher and Kiewnick (1964) investigated the germination of sterilised and unsterilised seeds of *A. fatua* sown in pots of sterilised or unsterilised soil with no added fertiliser, unsterilised dung, or one of four combinations of inorganic fertilisers (N + P + K, 2N + P + K, N + 2P + K, 2N + 2P + K). Although there were five replicates, no standard errors are given, nor even the range of results in each treatment. The percentage germination was low and the differences between treatments were small, eg the mean germination in

Table 10.4 *Germination as percentage of seeds sown (after Rademacher and Kiewnick 1964, Table 3).*

	No fertiliser		N + P + K	
	Both seed and soil sterilised	Neither sterilised	Both seed and soil sterilised	Neither sterilised
Total germinated in 3 years	15.4	4.2	53.2*	32.8
Dormant seeds	0.8	2.6	0.6	0.8
Dead seeds	83.8	93.2	46.2	66.4

* The highest individual % germination in any treatment, although the mean percentage was greatest in 2N + P + K.

the four inorganic treatments ranged from 28.5% with neither seed nor soil sterilised, to 34.3% with both sterilised. Seed and soil microflora are said to retard germination, the effect being most evident in low soil-fertility, but such differences are unlikely to be agriculturally important. The extremes are shown in Table 10.4.

Thus, sterilising both seed and soil increased germination by 11% without fertiliser and by 20% with N + P + K, ie increasing it nearly four times without fertiliser but not even doubling it with N + P + K. The 11% decrease caused by micro-organisms is a step in the right direction, but only a small one.

Unsterilised dung gave the least germination: 13.8% with neither seed not soil sterilised and 12.2% with only soil sterilised, compared with 4.4% with only seed sterilised and 6.8% with both sterilised. The dominant factor is thus seed-sterilisation, but it acts in the opposite way from treatments with inorganic fertilisers or none; but, again, the difference is small. Possibly seed-borne micro-organisms offered some protection against seed-rotting organisms present in dung. The other possibility, that sterilisation itself killed some seeds, is eliminated by comparison with other fertiliser treatments (or none) where seed-sterilisation increased germination.

A table given by Rademacher and Kiewnick (1964) compares the total germination in each year, in the five fertiliser-treatments, for the four sterilisation treatments. However, the variation in viability from 9 to 39% obscures the effects of dormancy. Table 10.5 shows germination converted to percentage of viable seeds, and also total seedlings, ie viability.

Germination percentage was greatest in the second year, as at Rothamsted (Thurston 1961). Treatments never differed from the means by more than 11%. First year figures for the treatments with the lowest viability were based on only a few seeds. The treatments with lowest viability (no fertilisers, or dung) also showed the most prolonged dormancy.

The fungi found on dead seedlings were chiefly *Fusarium culmorum* and *F. avenaceum*, with *Stemphylium consortiale* and *Epicoccum nigrum* next in abundance, and *Alternaria* sp. and *Aspergillus* sp. less frequent.

Table 10.5 *Germination of* Avena fatua *seeds under various fertiliser regimes (after Rademacher and Kiewnick 1964 p. 376).*

Fertilisers	Germination as % of viable seeds			Total seedlings in 3 years per 2000 seeds sown
	1st yr.	2nd yr.	3rd yr.	
None	5	63	32	234
Dung	1	72	27	186
NPK	17	59	24	759
2N P K	16	59	25	781
N 2P K	17	64	19	750
2N 2P K	15	67	19	694
Mean	12	64	24	

Rademacher and Kiewnick concluded that applications of mineral fertilisers, especially those containing nitrogen, give faster and more complete germination of wild oat seeds. This would tend to increase the infestation in the absence of control-measures, but further experiments might show that faster decontamination of infested fields could be achieved by a judicious combination of high-N applications, with crop rotations and cultivations to destroy the seedlings. They do not comment on the use of dung to encourage the rotting of seeds, possibly because their results indicated prolonged dormancy of surviving seeds, whereas Thurston (1963b) obtained quicker germination in dung than in soil. Dung treatment in Thurston's experiment decreased total germination over two years to 83% of that in unmanured soil and in Rademacher and Kiewnick's to 80% over three years.

For other effects of *Helminthosporium*, *Fusarium* and *Hymenula* see p. 215-6.

Other fungi are occasionally found on wild oats but seem to be of minor importance to them, eg Sprague (1950) includes nine species not mentioned above, some of them causing diseases of cereals and other plants, others of weak pathogenicity.

YEASTS

Yeasts are not usually regarded as pathogens, but have been found associated with wild oat seeds. Kommedahl *et al* (1958) obtained 20,000 yeast colonies per germinated grain (1,500,000/g) on potato dextrose agar and ten times as many from grains which were slow-germinating or dead. On malt-salt agar, germinated grains gave only 2500 yeast colonies per grain (187,500/g) and slow-germinating or dead seeds 50 times as many, although even this was less than 1% of the number found using the other culture-medium. They did not know whether the association of yeasts with slow-germinating and dead seeds was cause or effect.

Kiewnick (1963) found up to 2,800,000 yeasts/g of seeds of *A. fatua* collected in a wet season and 990,000 in a dry one. They were far more abundant in both years on seeds from Stuttgart than from seven other places in Germany. In the samples, the percentage germination and the number of yeasts per seed varied independently.

BACTERIA

No records of bacterial diseases of wild oats have been found while preparing this review. However, unidentified bacteria found on seeds have been associated with their viability and germination (Naylor and Christie 1957, Kommedahl *et al* 1958, Kiewnick 1963).

Bacteria far outnumbered fungi, and usually all other micro-organisms together, on all except one of the samples of *A. fatua* seed investigated by Kiewnick (1963). The greatest number he found was 37,380,000/g of seeds. Numbers increased two- or threefold in RHs of 97 and 100%, at which levels viability of seeds declined within six months. Kommedahl *et al* (1958) also

isolated on potato dextrose agar far more bacteria from non-viable and slow germinating than from germinated seeds, but the numbers were too great to count.

Naylor and Christie (1957) suggested that the hull of *A. fatua* may contain a bacterial inhibitor. While it lasted, this would discourage the rotting of the seed in the soil. There is scope here for further investigation of the interaction of bacteria and inhibitors on buried seeds, and as the hull disintegrates long before the dormant seed it envelops, inhibitors in the seed itself should also be considered (see also p. 83-4).

VIRUSES

Wild oat species can be infected by at least five cereal or grass viruses and two wide-spectrum viruses usually associated with cultivated dicotyledons.

Barley yellow dwarf (formerly cereal yellow dwarf) is the only virus commonly affecting wild oats in England. It causes reddening of the leaves and whitish sterile spikelests in the ears, and is most prevalent in seasons when the aphids which transmit it are abundant. Virus-carrying *Metopolophium dirhodum* probably arrives in the crop too late to affect seed-set in wild oats, but *Rhopalosiphum padi* and *Macrosiphum (Sitobion) avenae* are probably important vectors (Plumb 1971). Oswald and Houston (1953) showed that it has a wide host-range in the Gramineae. In inoculation and recovery experiments both *A. fatua* and *A. barbata* become infected, showing characteristic leaf-reddening. Infected plants of *A. barbata* were moderately stunted and *A. fatua* severely stunted.

Oat necrotic mottle was first recognised in cultivated oats in Canada in 1965 (Gill 1967). All the species and varieties of *Avena* tested were susceptible, developing systemic chlorotic lines of various lengths on young leaves. As the leaves matured, the chlorotic lines enlarged and merged, forming a chlorotic mottle, passing through orange and brown spots to necrosis as the leaves aged. Symptoms appeared more quickly at 25°C or 30°C than at 20°C. The virus was not transmitted by the five species of aphids and two of leafhoppers tested, nor by seeds or in soil. Using hand-inoculation, 84% of *A. barbata* plants, 84% of *A. sterilis* and 36% of *A. fatua* became infected.

Wheat streak virus (wheat streak mosaic virus) infected a few plants of *A. fatua* with mosaic symptoms in a host-range test using manual inoculation, but *A. fatua* was not susceptible to mites, so natural transport by them seems unlikely (Slykhuis 1955). Wheat spot mosaic, often associated with it in natural infestations, did not cause mosaic symptoms in *A. fatua* (Slykhuis 1956).

A mosaic virus found in *Lolium multiflorum* West of the Cascade Mountains in USA, and believed to be identical with a virus found in *Dactylis,* was transmitted with difficulty to various Gramineae including *A. fatua* (Bruehl *et al* 1957).

Anthoxanthum mosaic virus, considered distinct from all other grass viruses, was transmitted by sap inoculation, but not by several insect species,

seed or soil, to 18 species of Gramineae, including *A. fatua.* Symptoms in wild oats were not described, but infected cultivated oats were severely stunted and yielded 40% less than healthy plants (Catherall 1970).

An isolate of aster yellows virus from naturally-infected barley was capable of infecting wild oats (presumably *A. fatua*). Affected plants developed yellow blotches and became severely stunted, forming no panicles. Wild oats were less susceptible than cultivated cereals to this virus, as judged by percentage plants infected in tests (Westdal and Richardson 1969).

A virus (or possibly a Rickettsia) affecting numerous dicotyledons and causing Pierce's disease of grapes and lucerne dwarf or alfalfa dwarf was found naturally infecting 12 out of 59 *A. fatua* plants. In experiments three plants of *A. fatua* out of five inoculated gave virus recoverable by nymphs of the leaf-hopper vector, but showed no symptoms (Freitag 1951).

POSSIBILITIES OF BIOLOGICAL CONTROL

The fact that wild oats are so abundant and widespread proves that natural losses by predation of seeds and attacks by grazing animals, pests and diseases on the growing plants are insufficient to combat the natural increase of the weeds in habitats that suit them, ie in arable agriculture, especially cereals and beans. However, without these biological controls infestations might be even worse than they are.

Considerable losses of plants and seeds can be demonstrated and some of these natural losses can be exploited by cultural means. With the weed so closely related to the cereal crops, however, the introduction of biological control agents would inevitably involve some risk of crop damage.

PREDATION AND DEATH OF SEEDS

It has been shown that considerable loss of viable seed can occur from the surface of uncultivated soil (Whybrew 1964, Wilson and Cussans 1972, 1975), during the first 4-5 months after the seed is shed. By contrast, seed loss was very low when cultivated into the soil during this period.

The cause of this seed loss was at first ascribed largely to predation by birds and rodents (Whybrew 1964). Rodents do consume wild oat seeds but they do not appear to play an important role in the control of natural populations of these weeds. Populations have been commonly assumed to be low away from field margins, hedges, etc. However, Rhys-Green (personal communication) has recorded populations of the field mouse, *Apodemus sylvaticus,* of 15/ha in the centres of large arable fields. The daily consumption of an adult is of the order of 3.00 g of seed daily (Rhys-Green, personal communication) which is equivalent to about 200 seeds per day, if wild oat seeds were the sole diet. Some seeds may be buried by the animals and thus protected from other adverse agencies.

Birds can make a far more significant contribution to seed loss. Game birds and wood pigeons may be very numerous, and such birds, if they used wild

oat seeds as their sole diet, would be capable of removing large numbers perhaps 3000-7000 seeds per bird per day (calculated from MAFF 1970). In practice, wild-oats would form only part of a varied diet dominated by other seeds, notably shed cereals, and green herbage.

Although Whybrew (1964) noted greater survival of *A. fatua* in caged areas, Wilson (1972) reported that loss and death of seed of *A. fatua* was not affected to a great extent when plots were caged to exclude birds and rodents. In another experiment (Wilson and Cussans 1972) the difference between caged and uncaged plots decreased with an increased period of exposure during the autumn.

It was concluded that death of *A. fatua* seed on exposed soil surfaces is due to a composite of factors but that the most important single factor is probably a physiological response to climatic variations which can exist even when all predators are excluded and microbial activity greatly diminished.

These phenomena can be exploited culturally but it is far from clear how they could be accentuated by introduced biological agents.

CONTROL BY INSECTS

Wild oats are affected by many pests, of which the most striking example is late attack by frit-fly *Oscinella frit* which may cause up to 40% of non-viable seed. This insect provides an excellent form of integrated control in that the late generations of eggs are laid on panicles which have escaped or survived herbicide treatment.

This late attack appears to be specific to *Avena* species but in the spring *Oscinella frit* can also be very damaging to most other cereals, notably maize. There would appear therefore to be little scope for encouraging this or other native insect pests deliberately. More host-specific alien pests, if they exist, have not yet been recorded.

CONTROL BY PATHOGENS

The same generalisation applies: the known pathogens are too dangerous to crops to be encouraged deliberately. Wilson (1969) discusses the use of plant pathogens for weed control. Pathogens for this purpose must be imported as weeds are usually immune to local species. Moreover, pathogens seldom eliminate their host, and once successfully introduced are difficult to eradicate. Local conditions where a pathogen originates are not necessarily optimum for its development, and it may become more devastating or widespread in the place to which it is introduced.

Wapshere (1974) puts forward detailed tests for safety, to be applied to organisms being considered for introduction for biological control. They should be tested first against plants closely related to and morphologically similar to the weed, to make sure that they will not also be attacked. Then a selection of cultivated plants, especially those closely related to the weed, and any whose pests and diseases are little known, those evolved apart from or little exposed to the agent of control, those attacked by closely related

species and those already recorded as hosts, should be exposed to the proposed pathogen.

These tests might still fail to detect risks with polyphagous organisms attacking plants irregularly distributed between many families, with those having two alternate highly specific hosts (eg some rusts) and those attacking two or three phylogenetically widely separated plant groups.

So we must find an organism which will destroy wild oats and which is not already known to damage the crops in which they normally occur. The chances of success are not great, but a start could be made by examining the wild oats themselves to see what is affecting them—as distinct from looking for known cereal pathogens on them. It might also help if an attempt were made to grow wild oats in regions where they do not already occur, followed by a collection of any organisms attacking them. Such pathogens would need to be tested exhaustively according to Wapshere's (1974) programme, and introduction on a field scale contemplated only if the test showed them *probably* safe to use.

As indicated above, Kiewnick (1963, 1964) and Rademacher and Kiewnick (1964) investigated fungi attacking seeds (p. 217 *et seq*). The five which did most damage are all known pathogens of cultivated cereals, but the sixth, *Phoma hibernica,* which killed 23% of the seeds, was almost as damaging as the known pathogens and might be worth further study.