THE PROBLEM OF FUNGUS DISEASE IN CEREALS

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Summary

Substantial failures of sown seed to emerge still occur. Fungicidal disinfection to control covered smut, using the alternatives to out-moded organo-mercury, will be required concurrently to aid seedling emergence from soil. Control of mildew and rust through the use of resistant cultivars has been unpromising because of the changing spectrum of biotypes of the pathogens. Some chemical treatments as foliar sprays warrant continued investigation. Stem and root diseases associated with *Ophiobolus*, *Fusarium* and *Cercospora* fungi continue despite the recommendations of agronomic control. There are better prospects now for chemical control, with the success of benomyl against *Cercospora* lodging being a striking example.

INTRODUCTION

AVERAGE YIELDS of harvested cereals in New Zealand have in 70 years advanced almost 70% (wheat and oats) and 80% (barley) (*Farm Production*, Department of Statistics, 1969). These average yields of 51 bu (wheat), 72 bu (oats), and 62 bu (barley) appear to have resulted from the use of better varieties imported or locally bred, utilized within improved systems of husbandry. As many farmers can attain yields considerably in excess of the national averages, it is questioned whether disease is of major consequence in limiting yields. Yields are certainly recorded well below the averages. The limiting factors operating, in addition to drought effects that occur in some districts, include persistent and increasingly aggressive diseases.

SEED MORTALITY, PLANT ESTABLISHMENT AND SMUT

Field emergence of cereals is often much less than expected of elite, high germinating seed. Figures of Price (1971), Shipton and Tweedie (1967) in Australia, Alessi and Power (1971) in U.S.A. give support to an earlier report (Blair, 1938) that, under field conditions, emergence loss (in wheat) is commonly as high as 30 to 40% of sown seed. Adverse soil conditions and inferior seed are sometimes responsible (Chambers, 1961-3) but the interaction of pathogenic and decay-inducing soil fungi, favoured by soil temperatures and moisture levels conducive to their development (Blair and Copp, 1953), seems to be significant. The effect tends to be more severe in wheat sown out of grass cultivation. Grasses are known to support a composite of fungi on their roots which contribute to pre-emergence injury to germinating cereals. This is alleviated in some measure by fungicidal seed disinfection initiated in this country by Neill (1925) to eliminate seed contamination of wheat by bunt

(covered smut) (*Tilletia caries* and *T. foetida*) and subsequently used also against the barley and oat forms of this disease. It is accepted now that normal, healthy cereal seeds when treated with fungicidal seed dressings produce seedlings of greater vigour than those from untreated seed. The effect is obvious in field inspection and in dry or green weight data. Colhoun (1969), in commenting on this, said the disinfection appears to protect mesocotyls of seedlings from invasion by soil-borne fungi normally regarded as saprophytes and the effect can be expected to persist for several months after autumn sowing of cereals.

Organo-mercury materials which have been favoured seed dressings for 45 years are being phased out now because of potential environmental pollution. Sanderson (1964) showed that satisfactory control of covered smut and benefit to germination could be obtained with then available alternative chemicals—as did Jones (1971) in Australia with more recent formulations. The thiocarbamates (maneb and mancozeb) were the most promising.

Poor emergence and crop failure from excessive dosage of organo-mercury on seed of greater than 15.5% moisture content has occurred frequently (Smith and Blair, 1959). This fault has been examined and tested by law, with farmers now conceded the right to expect adequate compensation for loss, not only of seed that has failed to emerge, but also for estimated loss of production. This should be borne in mind in marketing replacement products.

A measure of caution may also emerge in the thought that reports will likely occur on the capacity of specific plant pathogens to develop resistance to some chemicals, in the nature of what Noble *et al.* (1966) described in regard to organo-mercury.

Through the benefit of general seed disinfection, *Tilletia* smut in wheat can be regarded as under practical control. Subinfective spore levels can be found on seed (Blair, 1956) and within the past three years sporadic but severe covered smut disease has been observed in Canterbury, directly attributable to the use of non-disinfected “home-grown” seed.

Absence of loose smut (*Ustilago tritici*) continues to be a criterion in the certification of quality in wheat and barley seed. Crops are still being rejected from certification by the Department of Agriculture and the Seed Testing Station reported that, in the past four years, rejections on account of loose smut were:

Barley 5%, (Carlsberg 2%, Zephyr 3%).

Wheat 36% (Raven 27%, Gamenya 9%).

Oats 1% (Mapua 1%).

Hot water treatment (Neill, 1927), applied to nucleus seed, has been regarded as a durable form of successful control but the chemical alternative now appears to have supplanted it. Mulholland (1970) and McEwen (1970) have given local confirmation of the overseas success of a fungicidal slurry of benomyl for wheat and of carboxin for barley which, as systemics, eradicate endophytic or subsurface smut fungi as effectively and economically (approximately \$2/bushel) as more laboriously applied HWT.

PLANT ILL-THRIFT AND FOLIAR DISEASES

Powdery mildew (*Erysiphe graminis*), and leaf and stem rust (*Puccinia* sp.) are ubiquitous on wheat and barley, though their severity is variable within crop and district. These pathogens have marked yield-reducing effects when invaded lower leaves and stems shrivel at an early growth stage with associated loss of physiologically active tissue.

Based upon the identification of 4 physiologic races, or strains, of *Erysiphe graminis* in New Zealand (Smith and Blair, 1950), plant breeding work at the Crop Research Division, DSIR, resulted (Copp and Wright, 1961) in the release and farm utilization of wheat varieties claimed to have an adequate level of genetically based resistance to mildew disease. An early breakdown or failure to retain this resistance was ascribed by Copp (1964) to the occurrence then in the wheat districts of newly established physiologic races not encountered during the breeding programme. Paradoxically, the older varieties, Dreadnought and Fife-Tuscan, outmoded and redundant on other agronomic criteria, did show a high level of resistance to mildew. It seems certain that many biotypes or races of this pathogen are present in this country, possibly as numerous as the 44 reported in Sweden and 39 in Denmark (Wiberg, 1970). If breeding is to be continued, to fix in our cultivars significant levels of genetic resistance, it appears that, in accord with the view of Smith and Smith (1970), the basis of wheat breeding might advantageously be altered away from simply inherited monogenic or specific resistance towards examination of multigenic characters that provide field, generalized or adult plant resistance.

Meanwhile in the face of this kind of difficulty in breeding work, there are now better prospects in chemical control. At Lincoln College, B. Arnst has found promise in the mildew controlling effect of the systemic triarimol used as a wettable powder, 4% a.i. foliar spray, seed dressing or soil additive. Unfortunately the material is to be withdrawn because of demonstrable mammalian toxicity. As a seed dressing, benomyl was shown by Evans *et al.* (1969) to control mildew for a little time after emergence, but as a spray at Feekes 7-10 growth stage, good control of the disease in barley was achieved with 15 to 20% yield increase.

An aspect of aetiology of the mildew fungus awaiting clarification is the extent to which cross-infection of wheat, for instance, occurs from other sources within the host range of *Erysiphe graminis*. This was listed by Dingley (1969) as blue wheat grass (*Agropyron scabrum*), oats (*Avena sativa*), barley (*Hordeum vulgare*), cocksfoot (*Dactylis glomerata*) as well as wheat (*Triticum aestivum* (*vulgare*)).

Rust disease like mildew seems to be ever with us. On field evidence it is highly detrimental but no control programme is currently in action in New Zealand. *Puccinia graminis* "form species" causing stem rust of wheat, barley, oats are not cross-infective. Distinct species cause leaf rust on the cereals, namely *Puccinia recondita* (wheat), *P. hordei* (barley), *P. coronata* (oats). This discussion is confined to the problem in wheat but it illustrates the whole. Dingley (1969) recorded for New Zealand, *P. graminis* f. *tritici* on 25 grass host plants (6 native) and McNabb (1962) for *P. recondita* listed 9 (2 native grasses). It is believed some infection of wheat arises from carrier grasses though this point does not appear to have been critically tested. Uredospores certainly survive on residual (self-established) wheat plants and in the absence from this country of an aecial spore stage on *Berberis* sp., the persistency of the red rust (uredo) stage is the concern here.

As air distribution of rust spores is now generally understood to occur (Ingold, 1971), it is manifest that air dispersal from the abundant rust inoculum in eastern Australia, where the wheat season is 2 to 3 months in advance of New Zealand's, is also part of the aetiology of this disease. Allen (1961), quoting the work of I. A. Watson, Sydney University, indicated at that time, 10 years ago, that strains of *P. graminis* dominant in the South Island had occurred earlier in N.S.W., while strains then

identified in the North Island wheat districts were common to Queensland. Now, a decade later, the situation is generally similar but with some differences, for Watson's survey from rust specimens sent from New Zealand (Rep. 1970-71, Dep. Agric. Bot., Sydney Univ.) shows:

P. graminis strains 21-2.5, 21-2.3.7, were common to N.Z., Tasmania, Victoria, N.S.W.

Strain 21-2.3.4.5.7 occurred in the North Island and Queensland.

Strain 194-2.3.7.8, the rust prevalent in Northern N.S.W., was also in the North Island of N.Z.

Strain 34-7, the most commonly occurring strain in that season in the South Island, was not recorded at all in any Australian state.

Uredospores certainly appear to be both air-borne from Australia and to originate in any one season from within New Zealand.

During the summer of this year, the writer saw one field instance of wheat stem rust that was extremely severe. Close (1967) had previously reported sporadic severe outbreaks. It is contended that, in contrast to 25 years ago, this potentially serious disease of stem rust is increasing in incidence and severity. If this is proven, the development would be an intriguing one considered in relation to whether or not the breeding of cultivar releases, while agronomically effective, may have a linked rust susceptibility. The explanation, however, may be the same as that advanced above to account for mildew, namely, the arrival of new biotypes or strains of the pathogen.

Breeding for rust resistance has functioned actively in some countries, notably Canada, for at least 50 years. Whether it is an admission of failure in breeding, or more the availability now of better materials, it is nonetheless clear that chemical forms of prevention are being increasingly examined. Hagborg (1970), throughout his career a scientist in the midst of the Winnipeg wheat breeders, has reported success with systemic and other chemicals—carboxin, oxycarboxin, Nickel-Zineb applied as foliar sprays. So also Van Meyer *et al.* (1970) whose work, however, indicated that some of the materials seem to be more effective against *P. recondita* than *P. graminis*. If foliar spraying of cereals can become fully economic, the consequential elimination of leaf blotch (*Septoria tritici*) will, on the evidence of Wenham (1959), also significantly contribute to better wheat crops.

PLANT ILL-THRIFT AND ROOT DISEASE

The composite of root and lower stem infecting fungi, *Ophiobolus graminis* (take-all), *Cercospora herpotrichoides* (eyespot lodging) and *Fusarium* spp. (seedling blight and footrot), occur annually in barley and wheat. There is no comprehension whatever regarding their overall economic importance in New Zealand. These organisms seem to be implicated in poor growth leading to constricted ears and shrivelled grain—although Smith and Wright's (1964) revelation that much of this kind of loss was caused by barley yellow dwarf virus is convincing. *Ophiobolus graminis*, causing stunted plants and bleached empty ears, builds up in severity under successive cereal growing. But it also appears in cereal crops sown straight out of ploughed grass where the cultivation of the latter has been through too brief a period to ensure desiccation of grass root residues on which this fungus is maintained. Paradoxically, take-all disease is now known suddenly to decline after a period of prevalence. Reasons are obscure but seem to be linked with microbiological activity, including

antagonistic and competitive effects among soil organisms and also the influence of nutrient levels, notably nitrogen. The *Fusarium* fungi known to be cereal pathogens in New Zealand are dominantly *F. avenaceum*, *F. graminearum*, *F. culmorum*, *F. roseum*, *F. nivale* (the latter rare in New Zealand). They produce progressive invasion of wheat and barley, from pre-emergence loss and seedling damping off to brown footrot of maturing plants, and ultimately blight and constriction of the seed. The latter develops when the lower stems are invaded or when infective spores from that origin are air-borne to the inflorescence. Control of *Ophiobolus* and *Fusarium* has been identified with crop rotation (Slope and Etheridge, 1971). Disinfection of soil has been regarded as impracticable but it may be significant that there is now renewed interest in the possibility of utilizing chemical treatment to eradicate pathogenic soil organisms. Salt (1970) and Ebbels (1971) have described work at Rothamsted and elsewhere, covering 50 years through which it has been variously shown that fumigation of field soil with materials like formalin, 1,2-dichloropropane/1,3-dichloropropene, dazomet, could significantly increase cereal yields through decreasing the incidence of soil diseases. The advances of chemical controls being so impressive, instanced by the arrival first of systemic insecticides and more recently systemic fungicides, it seems reasonable to expect a soil fumigant will be soon developed that can be economically applied by conventional equipment to extensive open field areas. At least in regard to *Cercospora* eyespot, a Southland wheat problem, there was a strikingly successful development when Witchalls and Close (1971) showed how this persistent disease, that had defied agronomic patterns of control, could be greatly eliminated through the systemic action of benomyl. Their treatment of one foliar application of 0.28 kg/ha in 91 l/ha water to spring-sown Southland wheat at Feekes 7-8 growth stage resulted in lodged tillers being reduced from 38.6% to 2.2%, with a yield increase of 1344 kg/ha in favour of the spray.

The use of chlormequat as reviewed by Humphries (1968) is also promising through its effect on shortening and stiffening the growth of wheat stems, straw resistance to eyespot lodging being thus induced. This is confirmed as an economic practice in Southland by Witchalls and Hawke (1970).

From this review it may be concluded that there is a persistence and in some instances an increase in importance of fungus-induced wheat disease in New Zealand. One can be restive in the thought that, in 40 years of interest by research and advisory officers, rather little impression has been made on control, until right now. We appear to be in the beginning of a new era when the chemical industry and its practitioners are achieving positive and economic successes with further opportunities awaiting their initiative.

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