New Zealand's seed health

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Abstract

Seed pathology, which has at least a 120year history in New Zealand, began with experiments for the control of cereal smuts. A brief history of seed pathology in New Zealand for the eras pre-1930, 1930-1960, 1960-1980, 1980-2000 is presented. New Zealand seed pathology has concentrated on diseases of the Poaceae and large-seeded legumes. Little is known of the occurrence or significance of seed-borne pathogens of other species, particularly vegetables. The New Zealand seed trade has for many years relied on fungicide seed treatment to control seed-borne pathogens of cereals, peas and brassicas, but currently there are problems with Fusarium spp. in cereals. New Zealand, as a biosecurity conscious nation, has strict measures in place to prevent the unwanted introduction of new plant pests. These requirements are briefly discussed. However seed health testing is carried out routinely only for seed lots requiring evidence of freedom from seed-borne pathogens for export phytosanitary requirements. In many cases knowledge of the health status of seed lots sown in New Zealand would enable better informed crop management decisions. Keywords: seed-borne diseases. seed pathology, seed health testing, seed treatment

Introduction

Detecting and controlling seed-borne pathogens has been a part of the New Zealand agricultural scene for well over 100 years. A seed-borne cereal pathogen was the subject of one of New Zealand's first recorded agricultural experiments, when, at Canterbury Agricultural College (now Lincoln University), Ivey (1881) demonstrated that 'pickling' wheat seed in copper sulphate could control covered smut (*Tilletia caries/T. foetida*).

New Zealand is an island nation, and the plant species used in its land-based industries are, for the most part, introduced. So therefore, are its seed-borne pathogens. In a survey in the mid-1980s, Hampton and Liew (1985) reported that for 14 temperate field crops and 13 temperate vegetable crops, around half the seed-borne pathogens recorded internationally had also been recorded in New Zealand. New Zealand's isolated situation and strict biosecurity requirements have helped to restrict the more recent introduction of new seed-borne pathogens. However there have been some slip-ups; for example Ascochyta lentis in 1985 and cucumber mosaic virus in 1994 (Ramsey et al. 1997).

Until the last decade of the 20th Century, the New Zealand seed industry was dominated by agricultural species: cereals for domestic use and multiplication/re-export, forage brassicas, and herbage species and peas (primarily for export). Seed pathology rather naturally has concentrated on diseases of the *Poaceae* and large-seeded legumes. The last 10 years has seen the rapid expansion of vegetable seed production, primarily for export to Asia (McKay 2010). Seed-borne diseases in vegetable crops in New Zealand have received relatively little attention.

A history of seed pathology in New Zealand Pre-1930

Pre-1930

Seed pathology research in the early part of the 20th Century was dominated by methods for control of the cereal smuts (*Tilletia* and

Ustilago spp.). Kirk (1906) and Neill (1926) reported success with liquid steeps or dry dusting with copper products and mercuric chloride for covered smut control (*Tilletia* spp.), but being protectants, these treatments had little effect on loose smut (*Ustilago* spp.).

1930-1960

By 1937 covered smut was 'of little importance throughout the wheat growing districts' (Blair 1937). Neill (1933) adopted a hot water soak method for control of loose smut, and this method became part of the Department of Agriculture's cereal seed certification scheme, in that all nucleus and breeders seed was hot-water treated. The subsequent 'flow-on' effect meant that seed released for commercial multiplication was generally loose smut free. In addition, seed crops found to contain more than 1% loose smut at field inspection were rejected from certification.

This era also saw one of the biggest research efforts into any seed pathology problem in New Zealand; blind seed disease of ryegrasses caused by the fungus Gloeotinia temulenta. Neill and Hyde (1939) had problem of low established that the germination in New Zealand produced ryegrasses was due to a seed-borne pathogen, and in 1941 a 'Blind Seed Disease Committee' was established to plan research and co-ordinate work. The committee continued to meet until the late 1950s. Control strategies investigated included breeding for disease resistance, fungicide seed treatment, and fertiliser management (see Hampton & Scott 1980), but none were successful as between 1948 and 1960, 70% of ryegrass seed samples were positive for blind seed disease, with an average infection level of 12% (Hampton & Scott 1980).

1960 - 1980

Concerns about the environmental/health hazards of organo-mercurial products in the late 1960s lead to their complete withdrawal as seed treatments in 1973, and for cereals the replacement was captan (Sheridan 1976). While outbreaks of covered smut (Close 1970) and loose smut (Sheridan 1977) were subsequently reported in commercial crops, poor seed treatment application techniques (Hampton 1976) were considered the most likely reason. However captan provided little control of other cereal seed-borne pathogens, and Drechslera spp. began to cause problems in cereals (Arnst & Fenwick 1973). Net blotch, caused by D. teres became an epiphytotic in the 1975-1976 and 1976-1977 seasons (Arnst et al. 1978). Matthews and Hampton (1977) reported that for barley seed lots from the 1975 and 1976 harvests, 76% carried D. teres and 16% carried *D. sorokiniana*. Hampton and Matthews (1978) isolated the pathogens D. avenae. D. tritici-repentis and D. sorokiniana from wheat seed, and Hampton (1980a) reported that 35% of wheat seed lots from the 1978 harvest and 57% from the 1979 harvest carried Fusarium, Drechslera and Septoria nodorum. Fusarium spp. accounted for over 85% of all recordings in both seasons (F. avenaceum > F. culmorum > M. nivale > F. graminearum > F. poae). By the 1977-1978 season, systemic-based products such as carboxin + thiram and carbendazim + mancozeb began to replace captan as a cereal seed treatment (Hampton 1980b).

In 1965 an epidemic of bacterial blight (*Pseudomonas syringae* pv. *pisi*) was recorded in field pea crops in Canterbury, the inoculum source being subsequently traced back to infected pea seed lots imported from Australia (Close 1966). This had serious implications for pea seed exporters, and beginning in 1969, both field inspection (rejection as a seed crop if the disease was detected) and seed health testing (not able to be exported if the pathogen was detected) were instituted as control measures (Watson & Dye 1971).

The pre-harvest testing of ryegrass seeds for the presence of blind seed disease had peaked in the early 1950s (over 3,000 samples were tested in 1952), but then fell rapidly during the 1960s to the extent that pre-harvest testing was discontinued (Scott 1974). During 1976-1978, 27% of seed lots carried the pathogen, with a mean infection level per seed lot of 4% (Hampton & Scott 1980).

Gaunt *et al.* (1978) reported the presence of *Ascochyta fabae*, the causal agent of leaf, stem and pod spot broad and field beans (*Vicia faba*) in Canterbury and estimated yield losses of 15% in many crops. Hampton (1980c) reported that from 10% to 30% of seed lots tested carried low (0.1-3.0% infected seeds) levels of the pathogen. Effective seed treatment (Gaunt & Liew 1981) has subsequently provided good control of the disease.

1980-2000

After a review of 30 years of testing data, Hampton and Scott (1980) concluded that blind seed disease was decreasing in importance, and proposed the hypothesis that because the spring application of urea can significantly reduce blind seed disease (Hampton & Scott 1981; Hampton 1987), the increasing use of nitrogenous fertilisers for herbage seed production was a major contributor to the decline in importance of the pathogen. However, while in most seasons from 1980-1990 only low levels of blind seed disease were recorded, there were major problems in the following three seasons (Hampton 1994). Although fungicides applied at flowering may reduce the incidence of the disease (Rolston et al. 2006) there are still no effective control measures available in New Zealand (Skipp & Hampton 1996).

Severe outbreaks of Ascochyta blight of peas (*Mycosphaerella pinodes* and *Phoma pinodella*) occurred in 1993 and 1994 (Ramsey *et al.* 1997) and again in the late 1990s, the occurrence and incidence being weather dependent. Mean seed-borne infection was 3.5% in 1993, 8.5% in 1994,

but only 0.6% in 1995 and ranged from 0.2% to 5.2% in the period 1996-2000 (Grbavac unpublished data). Symptoms of seed stain, tennis ball line and coat split attributed to pea seed-borne mosaic virus (PSbMV) caused serious quality problems in pea seed lots during the mid-1980s (Ramsey et al. 1997), with seed infection levels of up to 40% being recorded in field peas (Fletcher et al. 1989). Halo blight (Pseudomonas syringae pv. phaseolicola) in beans Phaseolus spp. was also a concern in the 1980s, with the percentage of seed lots infected ranging from 0% in 1981 to 20% in 1985 (Johnson 1985). An epidemic of bacterial blight of peas occurred again in 1983, with P. syringae pv. pisi being detected in 33% of 480 garden pea seed lots and 44% of 56 field pea seed lots tested (Johnson 1983).

The current situation

For some 25 years, systemic-based seed treatments have been used on virtually all cereal and pea seed lots sown in New Zealand, and more recently on all brassica seed lots (as a constituent of the seed coating routinely applied). The New Zealand seed industry believes that this 'insurance' approach is cost effective, and is strongly supportive of its continuation.

Routine seed health testing has never been a requirement of the New Zealand seed industry. Until recently, requests for seed health tests (such as for the Ascochyta complex, PSbMV and bacterial blight of peas) have been only for seed lots destined for export (where import requirements need a declaration of freedom from a pathogen) or elite seed lots for further multiplication. However, there is growing concern over Fusarium spp. in cereal seed lots (see next paragraph) and since 2001 one major New Zealand company has had all its cereal seed lots tested for the presence of *Fusarium* spp. Cromey et al. (2001) surveyed seed samples from 40 wheat and barley crops harvested in 2000 and reported that all carried Fusarium

spp., the incidence ranging from 3 to 52% of seeds infected, with a mean of 14%. Most common were F. avenaceum > M. nivale > F. graminearu > F. culmorum > F. poae. This survey is noteworthy for two reasons: firstly, the results are very similar to those reported by Hampton (1980), illustrating the fact that the systemic seed treatments in use for the last 20 years do not effectively control Fusarium spp. (indeed, the incidence of Fusarium spp. within a seed lot is often greater after seed treatment than in untreated seed, presumably because competing fungi are removed by the seed treatment (Hampton unpublished data)); and secondly, it is the only published survey of seed lots for the presence of seed-borne pathogens conducted in New Zealand in the last 20 years.

Biosecurity

While New Zealand has no seed laws (seed of any quality can be traded), it does have quarantine legislation relating to the importation of seed. In accordance with Section 22 of the Biosecurity Act (1993), the New Zealand Ministry of Agriculture and issues import Forestry (MAF) health standards detail (IHS) which the phytosanitary requirements that must be carried out, either in the country of origin or of export, during transit, or in quarantine, before biosecurity clearance can be given for a seed lot to enter the country. MAF must ensure that any requirements:

1. are technically justified

2. do not impose unjustified technical barriers to trade

3. provide an appropriate level of biosecurity protection (i.e., prevent the entry of unwanted organisms into New Zealand).

In its IHS, MAF provides a list of 'pests' (not previously recorded in New Zealand) for which specific actions must be taken. For fungal pathogens of wheat for example the actions are:

1. seed treatment (with any one of five MAF prescribed fungicide combinations) and

2 a declaration that the seed lot has been sourced from a 'Pest Free Area' free from *Tilletia contraversa* and *Tilletia indica* or

3. a declaration that the seed lot has been sourced from a 'Pest Free Place of Production' free from these two *Tilletia* spp. (see FAO International Standards for Phytosanitary Measures, Publications No 4 (Pest free area) and 10 (Pest free place of production) or

4. that a representative sample of 600 seeds drawn from the seed lot has been tested for the two *Tilletia* spp. according to International Seed Testing Association rules. (Note: for regulated bacteria and viral pathogens of wheat, options 2 or 3 above apply).

Are these requirements technically justified? There is no doubt the answer is yes for T. contraversa and T. indica. But what about Typhula incanata. Alternaria triticina. Drechslera tetramera. Fusarium chlamydosporum, Rathavibacter tritici and wheat streak mosaic virus which have not been reported from New Zealand? Are they serious threat to New Zealand's а biosecurity? After all, they have had over 100 years to become established. Or are they present but have never been officially recorded as so? This can occur, as recently demonstrated with Ustilgo maydis, the fungus which causes common smut of maize. Froud et al. (2006) reported the 'newto-New Zealand' detection of this disease in Gisborne in January 2006. However, the pathogen was present in maize fields near Gisborne in the mid-1970s (Hampton, unpublished). Technical justification is often a difficult area, particularly when data are scarce or non-existent, but it is probably sensible to err on the side of caution until such time as reliable information becomes available.

Can the appropriate level of biosecurity protection be provided? Again this is an interesting question, because with the exception of the *Tilletia* spp., the IHS for wheat requires only that seed be fungicide treated to prevent the entry of other regulated pest fungal species listed. While this seems sensible, the potential problem is that fungicides will not control bacterial or viral pathogens and there is little evidence that the seed treatment options listed in the IHS actually control the fungal pathogens in question. It is also of interest, and perhaps concern, to note that with the exception of the two *Tilletia* spp., the IHS for all cereals and large-seeded legumes for seed for sowing imports do not include a requirement for seed health testing (and even for the *Tilletia* spp. it is optional).

New Zealand, as a seed exporting nation, has encountered problems with what it sees as the unjustified use of phytosanitary regulations. Two examples are:

1. a seed-borne pathogen was detected on a weed seed in an imported New Zealand grown grass seed lot. Shipments of further grass seed lots were suspended, because the pathogen was declared to not occur in the importing country. Subsequent and costly investigation by New Zealand officials proved that the pathogen had first been recorded in the importing country in the 1920s, and while not common, was certainly present (Hampton 1998).

2. after over 100 years of exporting grass seed lots to another country, from 2000 New Zealand had to assure freedom from a number of seed-borne pathogens which have been recorded in New Zealand, but are of no economic significance. Government to government negotiations have resulted in a revision of the importing countries regulated pest list to the satisfaction of both parties (Hampton 2002; R. Bakker pers. comm. 2004).

The first example was, in the New Zealand seed industry's view, a non-tariff barrier to international trade, while the second example appeared to result from a lack of access to scientific information on the pathogens in question (R. Bakker pers. comm. 2004). The world's phytosanitary system should protect against the spread of economically important pests without causing unnecessary barriers to the international movement of seeds (McGee 1997).

New Zealand has had a long involvement with seed pathology. It is somewhat ironic therefore that in 2008 so little is know about the current state of New Zealand's seed health. For New Zealand's seed exporters, seed health testing is available through the AsureQuality laboratory at Lincoln University which is accredited to both MAF the International Seed and Testing Association. This laboratory can provide reliable data for seed lots routinely tested for export phytosanitary requirements, but this mostly involves only one species (pea) and three pathogens (Asochyta complex, PSbMV and bacterial blight). There are, for example, no data for the health of vegetable seeds produced in New Zealand, yet there have been recent production problems. For the 2003 and 2004 harvests, failure to meet contracted carrot seed germination standards the deleterious effects because on of germination seed-borne Alternaria radicina resulted in an increase in abnormal seedling development, consequent rejection of seed lots and a significant loss in export revenue (C. Green pers. comm. 2005). The problem is that seed health testing bears a cost, and funding for any type of survey work alone is very difficult to achieve. While the seed industry has been prepared to pay for 'after the event' seed health testing to determine why there was a seed lot quality problem, the need is for knowledge of the health status of the seed lots to be sown, so that management decisions can be made which may well avert, or at least reduce, the problem in the first place.

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