

Effects of some diseases on the quality of culinary soybean seed

Malcolm Ryley

Agency for Food and Fibre Sciences, Department of Primary Industries, PO Box 102, Toowoomba Qld 4350 Australia

Introduction

It has been estimated that diseases reduce the world's annual soybean production by 11% per year (\approx 15 million t). The most serious diseases are considered to be, in order of importance, soybean cyst nematode caused by *Heterodera glycines*, stem canker (*Diaporthe phaseolorum*), brown spot (*Septoria glycines*), charcoal rot (*Macrophomina phaseolina*), purple stain (*Cercospora kikuchii*) and sclerotinia blight (*Sclerotinia sclerotiorum*) (Sinclair and Hartman, 1999a). Australia is free from soybean cyst nematode and brown spot.

Some diseases, such as phytophthora stem rot caused by *Phytophthora sojae*, cause yield reductions primarily by reducing plant populations (Ryley et al., 1989), although this disease has been reported to also reduce the protein content of seed (Caviness and Walters, 1971). Other diseases such as downy mildew and purple seed stain can cause a direct visual effect on seeds, while others have a more subtle effect by reducing seed size, protein, and perhaps tofu potential. In this paper, I discuss the biology, importance and management of some diseases which affect, or have the potential to affect, the quality of edible soybeans in Australia. All of these diseases are caused by pathogens which either infect the seed and/or plants before harvest. A summary of the effects of these diseases on soybean seed quality, and potential management options are summarised in Table 1.

Phomopsis seed decay

Phomopsis seed decay is primarily caused by the fungus *Phomopsis longicolla*, but other species of *Phomopsis* and *Diaporthe* are often isolated from affected seed. The fungus belongs in a complex which causes pod and stem blight (*Diaporthe phaseolorum* var. *sojae*) and stem canker, caused by two other varieties of *D. phaseolorum*. This fungal complex is considered to be the most important pre-harvest disorder of soybean seed worldwide (Sinclair, 1999). These pathogens are seedborne, and most occur on other crops and weeds. Rainfall or moisture after maturity is thought to be the key factor determining the extent of damage by this complex (Rupe and Ferriss, 1986). Delayed harvesting due to rainfall or other factors usually leads to higher infection levels (Stovold and Francis, 1987).

Severely infected seeds are shrivelled, elongated, cracked and may appear white and chalky, but others might be infected but not show symptoms (Kulik and Sinclair, 1999). Infected seeds cause poor emergence, damping off and plant death (Abney and Ploper, 1988). Oil, meal and flour derived from affected seeds are of lower quality than those from non-infected seeds (Roy, 1976), and enzymes released from the fungi degrade seed coat proteins. Allen et al. (1992) found that some Australian isolates of *P. longicolla* were very toxic to sheep and rats, due to production of cytochalasin compounds.

The disease is found on soybeans most commonly from the north coast of New South Wales (Stovold and Francis, 1987), and only occasionally in other Australian production areas. Practical control measures include rotations with non-legumes, ploughing-in of residues, and the use of

weathering resistant varieties. Varieties with poor weathering tolerance tend to have higher levels of infection. Some resistant sources have been identified.

Downy mildew

This worldwide disease is caused by the fungus *Peronospora manschurica*. Before 1999 this disease occurred occasionally on the north coast of NSW, but in that year it was found for the first time in southeast Queensland and on the Darling Downs after a cooler- and moister- than normal summer. The pathogen has been recorded on native *Glycine* species throughout these regions (DPI and NSW Agriculture, unpublished records). Infection occurs during high humidity and low temps (20-22°C). Symptoms are pale yellow spots on the upper leaves, with a downy light purple growth on the opposite leaf surface. Severely affected leaves can die prematurely. Affected seed is partly or completely covered with a white crust consisting of the resting state (oospores) of the pathogen, often has cracks, and is smaller and lighter in weight (Phillips, 1999). In trials conducted in the United States in mid 1980's on susceptible varieties, yield losses (using fungicides to minimise infection) ranged from 10-25% (Dunleavy, 1987) due mainly to a reduction in seed weight.

The presence of downy mildew can be used as a quarantine restriction on soybean imports from Australia. New Zealand has a nil tolerance for downy mildew on soybean seed entering their country. We have found that 7 of the 53 samples of seed intended for export to New Zealand which we have tested since mid-1999 have been positive for the presence of downy mildew (QDPI, unpublished records). Most current varieties grown in northern NSW and southern Queensland are resistant to the pathogen. Residue burial, and rotation for at least 2 years, can reduce the threat of infection in subsequent crops.

Purple seed stain

Purple seed stain, caused by the fungus *Cercospora kikuchii* is considered to be the 5th most important yield-depressing pathogen in the world (Sinclair and Hartman, 1999a). As the name suggests affected seeds are discoloured, with pink to dark purple areas occurring as small blotches or covering the entire seed coat. Symptoms also occur on leaves (purplish discoloration of the upper leaf surfaces and lesions on the petioles) and stems (elongated lesions), and in severe infections premature leaf death can occur (Schuh, 1999). Symptoms are not usually visible until mid podfill, by which time infection has already occurred. Infection can occur over a wide range of conditions, but is favoured by increasing periods of dew later in the season, with spores originating from infected residues.

The major problem with this disease is a reduction in the visual quality of the seed, making it unsuitable for many sectors of the industry. Purple seed stain is an occasional and minor problem in most Australian varieties, with the incidence depending on weather conditions during crop growth. Most varieties released through the DPI over the past 20 years have cv. Davis in their background, which has acceptable levels of resistance to the pathogen (Schuh, 1999). Little is known of the inheritance of resistance to *C. kikuchii* in soybeans. Release of new varieties with resistance to the pathogen is the only viable management strategy.

Sclerotinia blight

Sclerotinia blight is caused by the soilborne fungus *Sclerotinia sclerotiorum*, which also attacks many other crops and weeds. Symptoms appear after flowering when the upper leaves wilt and die. At this stage there are water-soaked lesions on the stems which develop initially at the nodes and later spread along the stem. A white cottony growth of fungal mycelium grows over the

diseased tissues, and later black, elongated sclerotes develop on the mycelium and inside the stem tissue. Side branches and pods can also be affected. Infected pods are white on the outside, with mycelium and sclerotes on the inside. Infected seed range from white and mouldy to symptomless, and all are flattened and shrivelled. Research in the United States has shown that for every 10% increase in sclerotinia incidence, yield losses ranged from 150-260 kg/ha (Hoffman et al., 1998). Mean 100 seed weight, seed quality, and oil content decreased, and protein content remained unchanged as disease levels increased (Hoffman et al., 1998).

The pathogen is widespread in Australia (NSW Agriculture, unpublished records; DPI, unpublished records) having been found in all soybean production areas. Contamination of the seed with sclerotes during harvest is the major concern with seed quality. At present, the NACMA standards for edible soybeans include, under the foreign material/Admixture requirement, a maximum of 6 sclerotes per half litre (National Agricultural Commodities Marketing Association, 2001). Difficulty is experienced in separating sclerotes, particularly broken ones, from soybean seed.

Sclerotinia blight is favoured by high soil moisture, high humidity in the lower canopy, and moderate temperatures (Grau, 1988). Options for management include agronomic practices which reduce in-crop humidity, avoidance of affected fields, and host resistance. There is no active breeding for resistance to *Sclerotinia sclerotiorum* in Australian programs, although the variety Manta is believed to possess some resistance to the pathogen.

Rust

In Australia, soybean rust is caused by the fungus *Phakopsora pachyrhizi*. This disease usually appears late in the season (Kochman, 1977), being more severe when there are long periods of leaf wetness (such as dew periods), and a mean daily temperature of <28°C. In Australia, the humid coastal areas of Queensland and northern New South Wales, and the Atherton Tableland in north Queensland are the regions at greatest risk. However, in most years it is not considered to be a significant disease, and is rarely seen in the major production areas.

Small, angular, reddish-brown lesions develop on the undersides of leaves, in which pustules containing a multitude of spores erupt through the leaf surface. Pustules can also develop on petioles, pods and stems. Severe infection can result in leaf yellowing, premature defoliation and early maturity. The spores are windborne, but not seedborne, and other legumes including species of *Glycine* are alternative hosts (Keogh, 1975). Soybean rust can be found on some native *Glycine* species throughout the winter months in southern Queensland (Kochman, 1977).

The disease causes significant yield losses, particularly in southern Asia where losses of up to 90% have been recorded. The effect on the components of yield, including, pods/plant, seeds/pod, and mean seed weight depends on the time of first infection, rust severity and variety. In inoculated trials in the United States, Hartman et al., (1991) found that yield losses were higher and 100 seed weight was less, when infection occurred at R1 than at R6. When yield potential was high, the numbers of pods/plant and seeds/plant were less in rust-infected plants than in healthy plants. Significant reductions in yield, 100 seed weight (Ogle et al., 1979), the number of filled pods and seed, and oil content have been reported for rust-affected plants (Ogle et al., 1979).

Plant resistance is the only practical method of control. Four dominant, independently inherited genes for resistance (*Rpp1*, *Rpp2*, *Rpp3*, *Rpp4*) have been identified in soybean, and others are believed to exist (Sinclair and Hartman, 1999b). The QDPI recurrent selection program used the rust resistant parents PI200492 and Taichung 4 in the first intercrossing generation (Rose et al.,

1992). Material from the CSIRO breeding program is screened annually against the natural rust population in north Queensland.

Charcoal rot

The symptoms of charcoal rot, caused by the fungus *Macrophomina phaseolina*, appear after flowering during stress conditions. Infected plants rapidly wilt and die, and a silvery-grey discoloration develops in the epidermal and subepidermal tissue. Later small black dots (microsclerotes) develop in the affected tissue. These microsclerotes can survive for many years in the absence of a host plant. Charcoal rot reduces the quality of soybean seed through a reduction in seed weight, with seeds being small and shrivelled. Most other summer crops are susceptible to charcoal rot to some extent, and many weeds can be infected without displaying signs of infection. *M. phaseolina* is present in all agricultural soils in Australia, with the severity level depending on past paddock history and soil type. The pathogen can also be seedborne, but its importance is unknown.

Some varieties, such as, Davis (which is a parent of many of the DPI varieties) are known to have some tolerance to charcoal rot (Smith and Wyllie, 1999). Cereals and cotton are generally less susceptible than sorghum, maize, navybean and mungbean, and in badly infested fields rotations from the latter group must be at least 3 years. Agronomic practices that conserve moisture and minimise stress should be adopted.

Conclusions

The coastal regions of northern New South Wales, and the coastal cane-producing regions of Queensland which have considerable potential for soybean production, are at most potential risk from diseases which reduce the quality of soybean seed. Significant outbreaks require a specific set of weather conditions, and as a consequence they occur only occasionally. Very little can be done after harvest apart from grading, but there are preharvest management strategies which can assist in minimising the effects of these diseases on soybean quality.

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