Cooperative Research Centre for Sustainable Rice Production

Report on a study tour to examine symptoms of rice diseases in southern USA and California

Dr Eric Cother
Principal Research Scientist
NSW Agriculture
Agricultural Institute, Orange NSW

July – August 2002
Executive summary and recommendations

Overseas Travel Report

Dr Eric Cother, Orange Agricultural Institute, Orange

Examination of disease symptoms in rice, of likely importance to Australia, in the southern states of USA and California.

This was the first visit by a pathologist to look at rice diseases overseas since the industry started over 80 years ago. The purpose of my visit to the USA was to become familiar with current research into, and symptoms of, those diseases of rice that are potentially important in Australia.

Whilst my major interest was in rice blast, I took the opportunity to look at all diseases that were present at the time.

Blast

This disease is important in the southern states at all stages of growth. It is less important in the (comparatively) drier states of Mississippi and Missouri. It has been present in California since 1996 although its occurrence is very weather dependant. It was not observed this year in California until mid September because of drier conditions. While the relative humidity in the southern states is more conducive to fungal diseases than in Australia, questions were raised as to the effect of water depth (deeper in NSW) on plant physiology and blast resistance. It may occur that deep water would protect seedlings in NSW from blast infection – panicle infection will be dependent on free water at panicle emergence. Canopy structure and RH will govern whether conditions are conducive for spore germination.

Given the ancestral relationship between a lot of the NSW germ plasm and that of Californian cultivars, it is expected that our germ plasm will be susceptible to the blast pathogen. The molecular marker research being carried out at Southern Cross University will be important for establishing the current status of our cultivars. However, there are differing opinions in the USA rice fraternity as to the robustness and practicality of large scale marker testing.

Strategies and recommendations

Blast remains the single biggest threat to the Australian rice industry. Even though our research has shown that the pathogen could initiate multiple disease cycles if introduce, the exact behaviour of the disease will not be known until it occurs. It is possible that our agronomic practices are such that the disease will be of minor importance. The recent arrangement with California to screen NSW germplasm in their disease nursery is an important step.

However, as prevention is the best strategy, our quarantine must be maintained. Should the disease be observed on a limited scale, options for crop destruction must be seriously considered. In California, the infected crops were harvested in the first year of detection and there was no hope of containing the pathogen.
Bakanae

Strategies and recommendations

Because of the climatic similarities between California, Japan and Australia, this disease is potentially as dangerous as blast. All seed imports should be screened in quarantine for this disease as it is easily passively transmitted. It is emerging as the greatest threat to the Californian industry, more so because it is not as influenced by environmental conditions, as is blast. Strict adherence to a pure seed scheme in Australia should prevent the spread of the disease should it occur here.

Rhizoctonia diseases

*Rhizoctonia solani* competes with the blast fungus as the most important pathogen in the southern US rice crops. Although we have *R. solani* in soils in southern NSW, it is not presently known if these are pathogenic to rice or are genetically similar to the US isolates. I believe that this pathogen is unlikely to pose a threat to our crops given the (comparatively) lower ambient RH in NSW and more open canopies.

Aggregate sheath spot (caused by *R. oryzae-sativae*) has recently been detected in NSW. It is important in California and cultivars are routinely screened for susceptibility to this disease. It is literally swamped by *R. solani* in more humid environments. The research by Vincent Lanoiselet at Charles Sturt University should assist in clarifying the potential of this pathogen to cause loss of yield or quality.

Strategies and recommendations

A watching brief on aggregate sheath spot should be maintained. It is easy to be complacent about the apparent lower virulence of our isolates of this and the stem rot pathogen but this could change by mutation or new introductions of the pathogens.

Stem rot

Since its discovery in the MIA in 1994, stem rot (caused by *Sclerotium oryzae*) has remained a disease of minor importance and has not been reported in some years. It is a serious disease in southern USA and in California. Given that our cultivars have some lineage in common with Californian cultivars, it is assumed that the isolates of the fungus in NSW are less aggressive than those elsewhere. Hence it remains a threat should more pathogenic strains of the fungus be introduced.

Strategies and recommendations

As for aggregate sheath spot

Bacterial grain rot

Although it is likely that this disease has been present in the US for a number of seasons, it has only recently been characterised in that country. It has been present in Asia for over a century and introduced to America on seed. It has potential to severely affect grain yield and quality but the development of this disease is probably dependent on high RH.
**Strategies and recommendations**

We have been warned to be aware of this pathogen and it is worth keeping a watching brief in NSW for this disease. The symptoms are very similar to those associated with glume blotch (caused by *Pseudomonas syringae* pv. *syringae*). Severe symptoms of glume blotch should be investigated to characterise the bacteria associated with the diseased glumes to rule out the possibility of *Burkholderia glumae* being involved. This should be particularly so for crops that are harvested for seed.

It should be noted that, notwithstanding the presence or otherwise of particular pathogens, the disease susceptibility of our rice crops will be influenced by changes in the genetic structure and agronomy of the crop. Climate warming, change in canopy structure, and agronomic changes to reduce water consumption and tillage can all combine to alter the environment of the crop and hence its disease resistance. Continued vigilance for the occurrence of both local and exotic pathogens is important.

20 September 2002
# Overseas Travel

## Itinerary

<table>
<thead>
<tr>
<th>Date</th>
<th>Activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>18 July 2001</td>
<td>Depart Sydney for Houston, Texas</td>
</tr>
<tr>
<td>22 July</td>
<td>Robert Millar, RiceTec, Alvin, Texas – blast sheath rot and stem blight research</td>
</tr>
<tr>
<td>23 July</td>
<td>Dr Anna McClung, Dr Bob Flellstrom, Robert Shank USDA-ARS Rice Research and Extension Station, Beaumont, Texas – molecular biology of blast resistance, markers, screening</td>
</tr>
<tr>
<td>24 July</td>
<td>Professor Don Groth and Dr Chuck Rush, Louisiana State University, Rice Research Station, Crowley, Louisiana – pathology of blast and stem rot, general pathology of rice</td>
</tr>
<tr>
<td>25 July</td>
<td>to Stuttgart, Arkansas</td>
</tr>
<tr>
<td>26 July</td>
<td>Dr Georgia Eizenga, Dr Yulin Jia and Dr Fleet Lee, Dale Bumpers Rice Research Centre, Stuttgart – blast research</td>
</tr>
<tr>
<td>29 July</td>
<td>Dr Gabe Sciumbato University of Mississippi, Delta Research and Extension Centre, Stoneville, Mississippi – stem diseases</td>
</tr>
<tr>
<td>30-31 July</td>
<td>Professor Alan Wrather, University of Missouri, Portageville, Missouri - general rice pathology</td>
</tr>
<tr>
<td>1-2 August</td>
<td>Drive to Minneapolis</td>
</tr>
<tr>
<td>5-8 August</td>
<td>University of Minnesota, Grand Rapids – biocontrol of Alismataceae and diseases of wild rice.</td>
</tr>
<tr>
<td>8 August</td>
<td>Fly to Sacramento, California</td>
</tr>
<tr>
<td>9 August</td>
<td>County agent, Yuba City, California – Growers management of diseases</td>
</tr>
<tr>
<td>12 August 2002</td>
<td>Dr Jeff Oster, Cooperative Rice Experiment Station, Biggs, California – blast screening, stem diseases and emerging problems</td>
</tr>
<tr>
<td>13 August</td>
<td>Dr Carla Thomas, FieldWise, Yuba City, California – grower support</td>
</tr>
<tr>
<td>14 August</td>
<td>Depart for Sydney</td>
</tr>
</tbody>
</table>

This report outlines the major diseases of rice in continental USA with comments on severity and current research programs. It is not written as a travelogue diary but rather the emphasis is on disease, and not on the personnel with whom I met.
BLAST

Rice blast is caused by the fungus Pyricularia grisea. Research in NSW by Lanoiselet et al., 2002 has shown that this disease, if introduced, poses a real threat to the southern Australia rice industry.

At RiceTec, a private breeding company at Alvin, Texas, the emphasis is on a hybrid-breeding program for yield and disease resistance. Lines are imported through quarantine, especially from China and Asia. Observations are made on the F1 generation, not on the parental characteristics. There are 5 plant breeders and a staff of 150 on a 400-acre property.

Photos Various symptoms of leaf infection by the blast pathogen

Seedling nurseries are used to rate germ plasm for blast. The pathologist rates seedlings for blast resistance as this translates into adult resistance. The aim is to achieve durable resistance, not dominant single gene resistance that is common in
Asia. In Texas, multiple races of the pathogen (up to 5) are used in the inoculum. Blast inoculum overwinters in Texas on weeds, whereas in Arkansas, inoculum is usually seed-borne. Bad blast infections occur in Texas 1 year in 5, R.solani (causing sheath blight) 1 year in 2 and straighthead occurs yearly.

Blast isolates are grown on rice-polish agar, inoculated onto autoclaved (2 hr) rice stem nodes, colonised for 2 months then stored in the freezer.

Dr McClung, at Beaumont believes markers are more reliable than the blast nursery. In field testing, resistance may be confused with escapes or the environment may not be conducive to infection and subsequent disease development. Their research is looking for background quantitative resistance. In the nursery, the presence of one gene can mask the presence of another so markers are preferred as they identify all genes present. Gene expression may be related to the environment. They have developed PCR-based micro-satellite markers that are being used to advance new varieties that have unique combinations of blast resistance genes for broad-spectrum resistance. The Pi-ta gene from a Vietnamese variety gives the widest resistance [see next page]. Beaumont is aiming to fingerprint all varieties. Germplasm is also screened for their ability to yield under drought conditions and under ‘organic’ culture.

IC17, IE1K and 1B49 are the most important races of the blast fungus because the present cultivars of rice do not have resistance to them. Crossing southern varieties with Californian varieties hasn’t given random blast resistance. There may be some relationship between cold tolerance and blast resistance as Texas cultivars with resistance don’t grow well in California. They have found no relationship between days to flowering and blast resistance.

There is no evidence that blast races are evolving. There is not enough severe pressure each year that would cause the pathogen to shift in pathogenicity. The mix of cultivars being grown could even be selecting for weaker pathotypes.
Green house screening for blast looks at the plants' genetic reaction to a particular race. Breeding lines are excluded if lesions appear on the leaves – those lines passing this test go onto the field nursery where percentage leaf reaction (an IRRI scale of 0-9) and whole plant reaction are scored. Specific race testing is done at the early generation stage.

Indica cultivars are good sources of blast resistance. Each gene confers resistance to a unique spectrum of races. The Pi-K gene may already be in Australian cultivars as it is present in all Californian lines. The gene ta\(^2\) gives good field resistance.

Marker testing – 100 samples per day can be tested for the presence of markers by capillary electrophoresis. Micro-satellite marker RM144 is on chromosome 11, RM244 is nearby. Pi-k is completely linked to the gene (0.0cM whereas RM144 is 3.6cM away; ie 3.6% of 100 recombinants would have an error in transferring this gene). There is a high association of markers with blast resistance. There may be several genes for resistance in the region near the markers. Markers are not always true for indica types. The decision on which marker to use is dependent on parentage. Most markers are independent of each other. Flanking markers help to keep track of other characteristics that may be associated with resistance. Pi-b and Pi-k\(^2\) lie at the end of a chromosome and these may be easier to incorporate into germplasm than ta\(^2\) which is in the centre of a chromosome. RM208 shows excellent association with Pi-b across a wide range of germplasm.

In Louisiana, the semidwarf medium grain cultivar Bengal is very susceptible to blast. Flooded field conditions are not favourable for blast. If there is a need to increase blast severity in research plots, the crop is drained for 3-4 days.
Cold winters help to control inoculum carryover. Cool nights and hot, humid days in summer result in an increase in blast. Blast needs heavy dews; heavy rains don’t necessarily result in increased disease. In the blast nursery, too much water can wash the germinating conidia off leaves.

Photo. Effect of water level on blast disease. Plants on LHS were grown in shallow water and have sterile panicles.

Blast control by increasing water depth is not compatible with rice water weevil control, which requires draining of the crop.

Red rice is the number one weed problem. Rotation with soybean helps because of the herbicides that are used in soybean cultivation. Herbicide resistant cultivars are being developed because of problems with red rice [Clearfield mutant and Liberty®-transformed cvs]. 100 000 acres of Clearfield rice have been planted in AR this year. The bleaching herbicide, Command, is very cheap compared with propanil and is used where propanil resistance is present. It turns rice white but the crop grows out of this with no yield loss. Cv Cypress was transformed for tolerance to the herbicide Liberty®. It is a broad-spectrum herbicide isolated from an actinomycete and registered for non-crop use. The chemical also works in vitro against blast and Cercospora. Herbicide tolerance genes and Rhizoctonia solani resistance genes are linked. Germ plasm is sprayed with Liberty; this suppresses the R. solani and takes out the herbicide sensitive crosses – survivors are resistant to R. solani.
Quadris® fungicide is good for blast and R. solani control. Tolerance to this fungicide has been recorded in blast but not in Rhizoctonia, probably because Rhizoctonia propagates vegetatively.

Field plots for blast resistance screening rely on natural inoculum of blast. Plots are drained for 5 days and 46lbs of N/acre are applied to stimulate disease development.

In Louisiana, the blast nursery for screening early generation material relies on sandy soils and high N, together with misting nozzles for overnight humidity, to create ideal disease conditions. Survivors in the seedling nursery are 80% likely to have neck blast resistance. There is sufficient natural inoculum in the field so no more is added. The outside block of M201 is planted 2 weeks before the other crosses. A rake shaped VVVVVV makes rows in the soil – seed is sown using an improvised spreader that is fast and saves bending over. The spreader is the length of the row, about 50mm wide and funnel is about chest height. Seed is poured into the funnel, hits the rubber bung and is more or less evenly distributed along the row.

In Arkansas, the 1985 blast epidemic led to the release in 1989 of cv Katy containing a cluster of resistance genes. This was followed by Kaybonnet and Drew. Crossing and incorporation of resistance genes is easy because they are near the centromere. Work is in progress to discriminate between resistance genes – it is thought that Pi-ta² and Pi-ta may be the same gene. Fundamental Nucleotide Polymorphisms are being examined as a single protein change may be involved in changing a phenotype from resistant to susceptible. The basic material for US breeders for blast resistance is coming from the wild species. O.barthii is thought to be the progenitor of African cultivars and O. rifipogon the progenitor of japonica lines. Wild species are easy to germinate if dehulled and placed on agar. Blast is a niche disease and its occurrence emphasises the classic disease triangle (host, pathogen, environment) and mostly one factor is missing. Yield and quality drive the breeders but new germplasm is usually disease susceptible. 45% of the rice area in Arkansas is sown to cultivars that are susceptible to blast.

The fungicides Quadris and Gem are used for blast control in Arkansas but are expensive and don’t always give 100% control. Timing, growth stage and weather are very critical for successful fungicide application. BASF 500 or “Headliner” is a
potential fungicide close to commercial release. Quadris and other fungicides can be used as seed treatments. Normally seed is only treated with fungicides if seedling diseases are anticipated.

The pathogen overwinters in standing rice straw, primarily in the nodes, but it is a poor competitor if rice is ploughed in. It is possible to isolate the fungus from last year’s nodes in the field up until about June. The main inoculum for blast in Arkansas is infected seed, either internally or as adhering conidia. When the collar becomes infected, this lesion provides inoculum to contaminate grain. A locally bred cultivar, LaGrue, develops many fewer blast lesions on lower leaves than the Californian cultivars but the disease is most noticeable at panicle emergence. However, panicle infection is partly dependent on weather conditions

Water levels are much shallower than in Australia. Decreasing water level for 5-14 days increases the severity of blast. Anaerobic conditions due to deep water affect plant physiology and increase the plants’ resistance to blast. Clay soils and open country in Arkansas usually have fewer problems with blast.

On a leaf, the vein is a continuation of nodal tissue and hence is more susceptible to infection and the lesion follows the vein.

There are 20 blast races in the US but only 4 are regularly found in growers’ fields in Arkansas and of these, 1 or 2 predominate. IB49 is the main race but this changes as the germplasm changes. One generation of the fungus under ideal glasshouse conditions can cause a change of race – the fungus loses an avirulence gene as its pathogenicity changes.

In Mississippi, the blast inoculum (conidia) is blown up from the southern states though there may be some weed carryover. It is possible to map the movement of blast from south to north as the northern areas are planted later. Medium grain cultivars are more susceptible to blast than long grain cultivars.

The blast races in MS are the same as present in AR and early sowing helps reduce the incidence of blast. The plant passes through a young susceptible stage, into a more resistant phase and then becomes more susceptible at panicle emergence. Collar rot symptoms are not common, in contrast to Arkansas. In Mississippi, the panicle is infected by airborne conidia or from conidia on lower leaves if lesions are present, which is not always the case. When the Arkansas-bred cv, Newbonnet, was a more popular cultivar, it had no leaf lesions but it rapidly developed neck blast at panicle emergence from infection by airborne conidia. It is generally too hot for severe blast in Mississippi. The cultivars grown are mostly different from those grown in Arkansas, which has sandier, lighter soils than MS.

Farmers are reluctant to spray for blast and there is little use of Quadris. If used, blast sprays are applied twice, costing $US30-35/acre/application. Gem® has more activity against blast but is not as long lasting as Quadris, which can give control
for 2-3 weeks. Quadris was the first foliar fungicide recommended for blast control, as others didn’t give an economical return.

Blast, although it occurs, is not as big a problem in Missouri because of the dry weather – they receive less rain than Stuttgart. Main race is IB49. Blast inoculum is windborne and there is little, if any, overwintering of inoculum. Some northwest Arkansas seed is brought in but growers use mostly locally grown seed because of the weed problems, especially red rice, associated with AR seed.

Professor Wrather at Missouri State University is interested in providing detailed weather data for us to use in the DYMEX model to see if we can predict the relatively light infection rates that occur in Missouri.

In California, dew does not occur until August, so there is less rapid spread of blast. The Cooperative Rice Experiment Station (CRES) at Biggs tests 5-10 000 lines per year in seedling trays in the glass house. Testing is done after the crop in the field is harvested and again before seedlings emerge in spring. Testing is mainly for major gene resistance and this does not necessarily reflect other types of resistance that cultivars may have. Another limitation is that it doesn’t distinguish between any differential resistance of seedlings and adult plants. Also, screening in the glasshouse does not simulate multiple disease cycles. Lesion number and size give little information on multi-cyclic effects in the field. Lesions are subjectively scored as brown spot only = Resistant; spot has discrete centre = Susceptible. CRES wants to develop a system that will allow disease to develop more in the glass house so that a scale, such as the 0-9 IRRI scale for blast resistance, can be used. However, it is difficult to extrapolate any S rating through to field reaction, as the middle part of the scale doesn’t extrapolate well.

Seeds are placed on the surface of the soil in seedling trays using a template and not covered with soil (to save time). The seed is pushed into the moist soil using another template. Plants are inoculated at 10 day old and ~7 cm high. Plants are placed in the dew chamber and then spray inoculated – this prevents disturbance of inoculum which would occur if inoculated prior to placement in the chamber. Seedlings are kept inside for 16-24 h and results are read 10 days later. Presently looking at pre- and post-inoculation RH to maximise lesion development in the greenhouse. Seedlings can undergo a shock when removed from the dew chamber so are covered with cloth for 3-4 h when moved to the greenhouse to allow them to acclimatise – higher RH may overcome this problem.

Introducing southern US varieties with blast resistance into the Californian breeding system has introduced ‘yield drag’. They are not adapted to the Californian environment, show no cold tolerance and they are susceptible to stem rot. Tom Tai, UC Davis, Geneticist at UC, Davis is developing markers along the lines of Anna McClung’s group in Texas but CRES is not keen yet to embrace the technology. The Pi-z gene can be detected in long grain lines but not in medium grain lines where it is known to exist - why this is so is not known.
Markers won’t replace conventional screening because of the number of lines involved. They would be more widely used if there were no reliable screening techniques available. Markers are a tool only. Markers for bacterial blight have been developed that identify lines with supposed resistance. However, these genes are not expressed in the field for unknown reasons and the lines are susceptible to the disease.

No blast was seen in the field in California in 2002 until early September when the disease was identified on 2 farms, previously infested with blast. Usually blast symptoms occur mid-August when there are 8-10 h of dew; the dews are dependent on moist airflow from the coast. When the disease occurs, collar blast is quite common because free water stays longer in this region of the plant. The most common symptoms are neck node and collar infections. Rice crop residues are the most important source of primary inoculum but the role of secondary hosts is unknown.

Weather patterns have changed in the past 4 years and, together with changes to the cultivars grown (M201 replaced by M202 and then M204), this has led to less blast. The geographic area affected by blast has, however, broadened due to use of infected seed and, in one known instance, the use of contaminated harvesting equipment. The move to earlier maturing cultivars was made to avoid the boot stage forming late in the season when heavy dew forms at night (late August) and night temperatures are higher. FieldWise have 10 weather stations in the rice areas automatically updating to the base computer every 15 minutes. FieldWise developed their own model for blast based on a modified peanut Sclerotinia model from North Carolina. Disease forecasts are provided by this company and are accessed by growers via the Web. FieldWise trapped blast spores for 3 years as part of a PhD project but stopped when no blast was present in past 2 years. Rotarod samplers on timers are used to trap spores but looking for lesions is more reliable and gives earlier warning of the disease. The lesions must be mature to develop conidia and spore traps are only collected and examined weekly. Thus spray application based on spore sampling could be too late for effective control.

Prior to any pesticide being applied, a Pesticide Certified Adviser (PCA) must inspect a field to determine the presence of disease and write a prescription that is lodged with county authorities (can be via the Internet). A grower can be a PCA himself.

Dr Thomas thinks that the weather data we used in our Dymex model may underestimate the RH values and she is interested in collaborative work to compare her model based on temperature and hours of leaf wetness with our model which is more rainfall/temperature based.
Sterile panicles from blast infection

Nodal infection by blast
RHIZOCTONIA

There are 3 species of Rhizoctonia of importance to the rice industry in America. R. solani causes sheath blight in the southern states where it is the most important of the 3 pathogens. However in California, aggregate sheath spot, caused by R. oryzae-sativae, is more important and boarded sheath spot caused by R.oryzae is of minor importance.

Rhizoctonia solani on stems and flag leaf of rice at panicle emergence.

The sheath blight (R. solani) nursery at Alvin, Texas grows 200 cultivars from breeders in all States and these are rated for disease reaction. Similar uniform nurseries are grown in each State. There are no yield measurements. The trials are observational only and are not replicated. Each breeder has a specific number of cultivars of their choosing that can be included. The plots are irrigated overhead in the late afternoon and morning to extend the dew period. R.solani isolates are kept as sclerotia on dried agar plates with no loss of virulence. Inoculum is grown on 50% rice hulls/cracked rice. Inoculum is spread only down the middle row of a 6-row plot. Only 1 strain is used, which also infects soybean and this crop is grown in rotation to build up inoculum. Onset of symptoms and disease development (the disease severity curve) varies with cultivar.

An infection peg of the fungus forms directly from the hyphae so disease progression up the stem is rapid in humid conditions. For reasons not known, Californian germplasm is resistant to R.solani in Texas.
Sterile and disease panicles on plants heavily infected by *R. solani*

Early stages of formation of sclerotia on infected stems

Stem lesions can cause weak stems.
Leaf lesions caused by Rhizoctonia solani

In Louisiana, short growing season cultivars of 75-80 day-maturity are grown, so ratooning is possible. 60% of cultivars grown in the south come from the LSU program at Crowley. R. solani kills the tillers, so consequently severely infected crops are not ratooned. Ratooning is not a problem with blast. Ratooning occurs from Crowley west into Texas but no further north due to the shorter season with insufficient heat units. About 50% of crops are ratooned, yielding 25-50% of the first harvest. R. solani is the main pathogen in rice because soybeans are used in the rotation and they are very susceptible.

R. oryzae is worse in dry years when R. solani is suppressed. R. oryzae can be seen in a harvested crop by removing trash lying on remaining stems; orange/salmon coloured mycelium is noticeable in the tissue under the humid trash covering.
At the Dale Bumpers National Rice Research Centre, Stuttgart, Arkansas, wild relatives of cultivated rice are being screened as possible sources of resistance to Sheath Blight caused by R. solani. They can’t be screened in the field because the canopy structure is not conducive to disease development and mature plants shed seed too easily. Dr Eizenga developed a glasshouse technique for inoculating tillers of accessions and F1 progeny. Dr Yulin believes there is no simple resistance to R solani and breeders will have to rely on the GM approach.

Arkansas isolates of R. solani are identical with those from the Philippines. They have offered to check our isolates for us; although the pathogen is present throughout our irrigation area, it has not yet been isolated from rice. Isolates could be collected from soybean grown in rotation with rice, so we could at least know the relationship between our isolates and those causing disease in rice elsewhere.

Photo Early symptoms of infection by R. solani

The tolerance of a cultivar to R. solani regulates the rate of movement of the fungus up the plant. The plant can tolerate the presence of the fungus in lower leaves but if it moves to the top two leaves, the effect of disease increases markedly. If the flag leaf is infected, yield loss can exceed 70%.

In Mississippi, R. oryzae is only a nuisance to the rice industry. Symptoms caused by this fungus are masked by the more aggressive R. solani. Night temperatures in the 80’s (F) influence movement of the fungus into the upper canopy, as does water depth. (12-15cm is normal). In July, water can be in short supply and this increases the incidence of R. solani. Changing the water up and down can drown sclerotia and reduce the inoculum source.

Some soybean cultivars are susceptible to R solani and this rotation increases the number of sclerotia in soil. Price governs the crop grown in rotation with rice (soybean, cotton or maize) but in heavier soils there is not much choice for farmers and a 2-year soy/1-year rice rotation is used for red rice and weed control. Incidence of R. solani is not influenced by soil type. Rice straw burning is being phased out.

R. solani is less of a problem in Missouri because it is drier. They may get some basal lesions but the fungus doesn’t move up the canopy. Farmers rarely spray for this disease, although some fields traditionally have a problem. There have been no bad years for R. solani since the 1980’s. This is thought to be due to the weather
getting drier. Any initial infections don’t progress. Rotations are with soybean, sometimes corn, rarely cotton, which needs lighter soils and are principally for red rice control. Very rarely get aerial web blight on soybeans, (caused by R.solani), though it will occur if late July rain falls. Soil populations of R.solani are variable under soybean; they sometimes increase and sometimes decline.

There is no problem in California with R. solani, presumable due to lower RH within the rice canopy.

In California, R. oryzae-sativae is more aggressive than R. oryzae and spreads up the plant faster than this pathogen. R. oryzae is not often seen in California. Basidiospores are sometimes produced on the upper stems. R. oryzae produces sclerotia infrequently and doesn’t overwinter well. R. oryzae-sativae produces 10+ sclerotia per tiller in air spaces in the leaf tissue.

Sclerotia appear white at first and are elongated because of the shape of the airspace.

Some discolouration is associated with lesions – this doesn’t become necrotic and nothing can be isolated from this tissue. In the field, dead heads on R. oryzae-sativae-infected plants appear yellowish compared to those on stem rot infected plants which are pinkish in colour. Infection on the flag leaf can be very damaging. The mid-range of fertility levels does not influence R. oryzae-sativae as much as it does stem rot, which increases as nitrogen levels increase. N and K must be balanced. High N and low K will aggravate the incidence of R. oryzae-sativae.

Quadris gives some control of R. oryzae-sativae. Timing of application must be 1-2 weeks earlier than for blast control when it is applied at the boot split stage. While toxic to fish, the lockup period for water management is such that no water can be removed from the crop between application and harvest.

It is possible to have 100% incidence of aggregate sheath spot in a field but for it to be of low impact on plants. The relationship between pathogen and cultivars has
not yet been established. The stem rot pathogen (*Sclerotium oryzae*) can be replaced over several seasons by the more biologically aggressive *R. oryzae-sativae*. The yield effect of aggregate sheath spot is less than that of stem rot but it has a greater impact on milling quality.

Lesions caused by *R. oryzae-sativae*. The dark line within the lesion is characteristic of the disease in California but has not been seen in infections in Australia.
BAKANAE

Known in Japan for over a century, Bakanae was discovered in California in 1999 and has slowly increased in incidence and importance. Some fields this year will have a 30% incidence with as many as 80% of fields having the disease. The disease, caused by the fungus Gibberella fujikuroi, was probably introduced on seed illegally imported from Japan. Infected seedlings are pale and elongated and the elongated sheaths later develop brown spots. The primary leaf stands out at a greater angle than in uninfected plants. Infected seedlings usually die. Those that grow to maturity produce no panicles or they are sterile. Plants infected with Bakanae flower 5–7 days earlier than healthy plants.

Infection by G. fujikuroi produces elongated, slightly yellow plants, with pronounced projection of the flag leaf. Infected plant (LHS) compared with healthy plant.
Spores are produced on infected plants at and above water level

The main inoculum is the spore load carried on paddy. Spores from the massive amounts of inoculum produced on dead plants adhere to seed during harvest. Farmers can mix contaminated seed 1:4 or 1:5 with clean seed and still get the same level of disease in the field due to the spore load. There is very little overwintering of inoculum. The industry can control the disease if the seed-borne levels of inoculum are maintained at zero levels by the use of clean seed. Although Bakanae may infect nodes of the plant in the field, the crown of the plant is the main infection point.

Bakanae is not as dependent on environmental factors for infection and spread as is the blast pathogen. Therefore, it poses a much higher risk to the rice industry.

If seed is allow to stand for 48 h following soaking instead of the normal 24, the incidence of Bakanae rises. Various seed treatments have been tried. Chlorine bleach at 0.1% after the seed has been soaked and drained appears successful. CRES are only testing chemicals that are, or have been, registered in California as it is too difficult to attempt registration of new compounds. A chlorine soak would be easy to register if the problem of disposal of the soak solution can be effectively handled. The most effective chlorine treatment gives a 20% reduction in seedling growth 2 weeks after treatment; the plants eventually grow out of this but in a field situation, this could interfere with the management of water, herbicide applications, etc. The systemic acquired resistance stimulator, Messenger, is ineffective as a seed treatment and its use actually increases the disease. It has no effect against stem rot.

Because Bakanae symptoms expression is greatest at 90-95 F (31-35°C), treatment experiments are planted later than usual to coincide with higher temperatures and to accentuate symptoms.

The fungus is variable and several different strains exist. These exhibit a variety of stunting or elongating effects, or none at all, on infected seedlings.
Characteristic sharp angle of the flag leaf displayed by Bakanae-infected plants

*Bakanae incidence is increased by zero or low water levels (water depth – early season, 2-4 inches, panicle initiation 8", normally 6" through most of the season) so water management can be used to improve disease severity, especially in the glass house, where no flooding is used.*

The best time to rate plants for the incidence of Bakanae is 30-35 days after sowing. This gives a good indication of what will develop later in the season even though the level of disease fluctuates through the season. With the sun at the right angle, as few as 1 plant per half-acre can be spotted in a field. Occasional diseased plants will appear through the season but the reason for delay in symptom expression is not known. Plants dying later in the season have a greater effect on final yield, as there is less compensation by neighbours. For every 10% of seedlings dying, the grower can expect about 1% level of dead heads at harvest.

If seed is harvested from an infected field, the grower can expect a 13-15 times increase in incidence of the disease the following year. Thus the disease can slowly build up from a very low incidence to become quite devastating. There are no
established standards for any disease in the California seed scheme; it is merely noted on inspection if there is disease present in the field.

In the past, disease management has included stubble burning. Currently, the 'safe harbour' provision in the Agriculture Burning Reduction Act will allow stubble burning if the grower can prove that disease is limiting his yield. He can then burn 25% of his acreage. The quite lenient test provisions are based on incidence, not severity, and burning is chosen by growers as a management option whenever possible. Bakanae, however, is not listed at present on the 'safe harbour’ list, even though burning is an effective option.

Stubble incorporation has shifted the effects of disease up to a higher plateau that has levelled off rather than continuing to rise. Climate and fertility then influence disease severity more. In the absence of burning, stubble incorporation has also increased problems associated with weed seed survival.

Bakanae could easily dominate stem rot and aggregate sheath spot as the most important disease if it is not controlled through adherence to a clean seed program.

Seed testing is best done by dilution plating of the washing from a seed lot rather than by plating individual seeds. This is because of the massive spore load and cross contamination.

Cultures of the fungus produce bright orange pigment on BBL PDA (LHS, less so on Difco PDA, RHS). The top of the culture is yellow/orange 4-5 days after plating and the colour then fades as the colony ages.
STEM ROT

Stem rot, caused by Sclerotium oryzae, is common in all States. In Texas, it is worst when water levels are kept constant; varying the level helps to control the disease.

Stem rot is the biggest problem in California, especially in taller cultivars as the disease increases lodging.

Some resistant cultivars are nearing commercial evaluation stage in California, especially for long grains. Markers are being developed to tag stem rot-resistant genes. Resistance is really needed in medium grain cultivars. Resistance is mostly from O. rufipogon although it has taken breeders from the 1980’s to remove undesirable characters of O. rufipogon and incorporate quality traits. Lines incorporating genes from O. rufipogon have, over time, been given a yield advantage. L205 is a susceptible cultivar that is infected up to the last leaf whereas in IY502, the disease only progresses to the 4th leaf.

A yield loss of 15-20% is incurred when the 3rd and 4th leaves are killed. Resistance is relative, in terms of fewer disease symptoms [cf. tolerance, where the same level of disease gives less yield or growth reduction].

Stem rot is a more predicably damaging disease than R. oryzae-sativa. However, yield loss data from stem rot over a number of years is not consistently related to severity levels. No fungicide is available that can be used as a research tool that would allow correlation of severity levels and yield.
For research purposes, sclerotia of *S. oryzae* are produced on autoclaved paddy rice; this is then milled and sclerotia screened out using normal equipment. Field plots are inoculated with sclerotia distributed using a backpack blower-mister. Resistant cultivars can yield up to 1000 lb/acre (~1100 kg/ha) more than a susceptible cultivar under high disease pressure. Resistance to stem rot is also related to resistance to *R. oryzae-sativae*.

Damage from stem rot at the 3rd leaf stage or higher up the plant at grain filling will lead to double digit yield loss and the earlier these leaves are killed, the greater the loss. All candidate lines for commercial release are screened for stem rot resistance through the disease nursery at F8+. If bred specifically for disease resistance, lines are tested at F3+.
PANICLE BLIGHT

Panicle blight (caused by Burkholderia glumae) is especially bad in Texas around Beaumont. Individual glumes or whole panicles can be infected. All crops were infected in 1995. The pathogen was discovered during biological control experiments when another species of Burkholderia was being used. It has been a problem in Japan for many years as contaminated seed rots when sown in transplant nurseries. It is carried on seed and there are no plant symptoms until the panicles emerge.

The condition was originally thought to be physiological. The pathogen is difficult to isolate and needs a semi-selective medium to recover the bacterium, as it is a slow grower. B. gladioli is also isolated on about 10% of occasions. B. gladioli from South America is also pathogenic to rice. Hot dry conditions have aggravated the disease and though bacterial, no ooze is seen in infected panicles. The bacterium produces a yellow colour, due to a toxin, on PDA (looks like Pseudomonas fluorescence on KB). A rifampicin-resistant strain is used for transmission studies. It is a very good antagonist to R.solani. LSU is developing a monoclonal antibody (ELISA) for seed testing. There are at least 2 strains of B. glumae present in rice fields. High night temperatures are the key to infection.

The bacterium moves up the growing plant as an epiphyte and infection occurs at flowering. Glumes become bleached or discoloured with the tip of the glume being a lighter colour than the (darker) base. Infection causes sterility and can be mistaken for straighthead; however, no ‘parrot beaking’ is present (see photographs). In field trials, inoculum is applied at 25-50% panicle emergence with 10⁶ cfu/ml giving good infection.
Discolouration of glumes caused by infection by *B. glumae*

Professor Rush at LSU strongly advises caution when bringing seed into Australia from USA or Asia as *B. glumae* is a common contaminant.

The incidence of *B. glumae* in Mississippi and Missouri is lower than in Louisiana but scientists admit that they are not aware of how serious the problem may be. Some cultivar resistance may be present.

In California, no bacterial seed infections have been found but they have not been specifically looked for, as most pathologists have a background in mycology but not plant bacteriology.

In Arkansas, *panicle blight* (*Burkholderia glumae* and *Pseudomonas fuscovaginae*) occurs occasionally after heavy storms and hail damage.
*Burkholderia glumae* infection of panicles (LHS) does not cause the ‘parrot beaking’ symptom (arrowed, RHS) associated with straighthead

Infection by *B. glumae* may be confused with glume blotch and we should be aware that introduction of *B. glumae* may be overlooked.
OTHER DISEASES

*Helminthosporium* can be periodically bad and in Louisiana, occasionally *Cercospora* attacks the base of the panicle at the internode (whereas blast attacks at the node). *Sarocladium* flag leaf infection can cause some sterility and is often associated with stink bug damage.

In Texas, *Ustilaginoidea virens* – false smut – green to bright orange pustules, turning black at maturity. There is sporadic occurrence everywhere. There is no yield reduction but contaminated grain cannot be milled because of ergot alkaloid.

In Louisiana, kernel smut (caused by *Tilletia barclayana* infection) can be high in some cultivars and false smut (*Ustilaginoidea*) is becoming a problem.

Kernel smut also occurs sporadically in Arkansas, though infected grain can be blended 2-5% for milling. Such grain not suitable for parboiling as it colours the product.

In Mississippi, kernel smut is a big problem and increases with increasing fertility. Toxin production by isolates in MS is less than that which occurs with isolates in Japan. One spray per year with Tilt® gives good control.

*Tilletia barclayana* occurs infrequently in California, although it was very bad 5 years ago. The disease requires free water and very high fertility.
Crown sheath rot caused by *Gaeumannomyces graminis*

*All new hybrids in Texas are highly susceptible to Gaeumannomyces graminis (crown sheath rot or black sheath rot). The pathogen colonises the nodes first and weakens them. Black sheath rot and stem rot (*Sclerotium oryzae*) can be distinguished in the field by pulling on the infected plant; *G. graminis* infected plants snap at the nodes whereas stem rot-infected plants break between the nodes.*

*This fungus is present in most wheat growing soils. It may pose a threat to rice growing if our watering regime was to change.*