RISK ASSESSMENT OF EXOTIC PLANT DISEASES TO THE AUSTRALIAN RICE INDUSTRY, WITH EMPHASIS ON RICE BLAST

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ISBN 1 876903 03 1

Risk assessment of exotic plant diseases to the Australian rice industry

ABSTRACT

A pest risk assessment was carried out using all available information found in the literature and also two softwares developed by the CSIRO, CLIMEX and DYMEX. CLIMEX was used to assess the suitability of the climate in Australian rice growing area for each pest/disease and then when necessary and possible, a pest/disease model was created with DYMEX and run with Australian climatic data.

The Australian climatic conditions and/or the rice growing practices were found to be unfavourable for the majority of the exotic diseases. However, two diseases of rice (rice blast and kernel smut) and one plant parasitic nematode genus (root nematodes) were identified as having the potential to threaten the Australian rice industry if ever introduced in south eastern Australia.

KEY WORDS:

Disease risk assessment, model simulation, rice diseases.

INTRODUCTION

The rice industry in south eastern Australia has enjoyed a lifetime of freedom from serious diseases. None of the debilitating diseases of rice crops that are found elsewhere in the world have been recorded in the region. However, there are over 50 important diseases of rice crops in Asia, India and other tropical and temperate rice growing areas that lower yields and increase production costs.

Diseases in rice crops are caused by fungi, bacteria, viruses and nematodes. All these pathogens pose a serious threat to the high levels of productivity and the comparatively low levels of pesticide use of Australian rice crops should they ever be introduced to Australia. The introduction of disease is quite possible because more than 30 diseases can be transmitted on or inside rice grains. Because of its current relative protection, there is neither varietal selection program nor fungicide registered for any of the main rice disease. As a result, any introduction of a major rice disease may have dramatic effect on the rice industry of Australia. Each disease that becomes established in a new environment has a potential two-fold impact: a potential reduction in yield and an increased cost of production associated with control or processing procedures. Disease also incurs a cost to the industry through losses in export revenue due to declining production and additional entry restrictions or prohibitions that may be imposed by importers of Australian rice.

This disease risk assessment outlines the potential for disease problems to develop in the rice crops of south eastern Australia and describes existing diseases of rice in southern Australia. Some of the more serious, exotic diseases that could become established if inoculum was introduced to the country are outlined.

1. THE POTENTIAL FOR DISEASE PROBLEMS

The isolation of Australian rice growing regions from other rice growing areas of the world; rotations with upland crops and pastures; and strict quarantine regulations have helped to protect the south eastern Australian rice industry from crop diseases. In addition, the Mediterranean climate of the region with its hot dry summers is not conducive to the buildup and spread of many diseases.

Despite the conditions that have prevented the outbreak of diseases in rice crops in south eastern Australia, the rice industry should not become complacent about its disease status. Australian rice crops are particularly vulnerable to introduced diseases for several reasons.

1.1 Lack of varietal resistance

There is no known disease resistance or tolerance in the current rice cultivars grown in south eastern Australia. Breeding for disease-resistance can be a complex and lengthy task, and can only be carried out in the presence of the disease-causing organism.

1.2 Increased risks of pathogen introduction

The mobility of people around the world, increasing trade pressure to expand global markets and increased importation of rice and rice products, and increasing contact between rice growing regions increase the risks of pathogens being introduced to the industry.

1.3 Improved environment for disease buildup

The intensity of rice growing increased markedly in the 1990s. Rotations were shortened and in some cases are almost non existent. Rice following rice is a common practice, with some rotations including rice in as many as 9 crops in 12 years. Intensive crop rotations provide a more conductive environment for disease infections to establish and spread throughout the industry.

1.4 Diseases can adapt to local conditions

Disease pathogens, like all living organisms are capable of evolving and adapting to changed environments. Diseases, which may be of minor importance in the country of origin, could be a more serious threat in the new environment of south eastern Australia.

1.5 Poor awareness of disease risks

Australian rice growers generally are not aware of the risks that diseases may pose to the industry and its growers.

Quarantine regulations and vigilant crop monitoring are the two major defences available to the Australian rice industry to prevent serious disease outbreak in rice crops. Additionally, ongoing research helps give greater understanding of the disease risks to the industry, and the likelihood and conditions conducive to an outbreak of disease. Technological developments such as the use of molecular markers will provide information to enable breeding programs to develop varieties that could cope with the major disease threats to the Australian rice industry.

2. THE IMPORTANCE OF QUARANTINE

The quarantine protocols that have been in place for decades under the Commonwealth Quarantine Act 1908 have kept imported rice products and by-products, capable of carrying the inoculum of many fungal and bacterial diseases, out of the rice-growing areas of south eastern Australia. The disease inoculum, in the form of fungal spores and resting structures and dried bacterial exudates, can be present in paddy rice and, to a lesser extent, adhere to milled grain. Rice straw, in the form of packing material or processed as floor coverings, can similarly contain disease-causing inoculum. Strict enforceable quarantine regulations have assisted in keeping this material out of the rice-growing areas or ensuring that suspect material is adequately treated.

The isolation of the rice-growing regions has assisted in maintaining the relative disease-free status of Australian rice crops. However, this isolation is under threat. A previously insignificant danger, but one that is gaining increasing importance, is the risk of introduction of disease by growers returning from visits to rice crops overseas or by the increasing number of tourist groups that travel to study the Australian rice industry. The ease of international travel increases the potential likelihood that pathogens could be introduced on the clothing and footwear of visitors. Fungal spores in particular can adhere to clothing in numbers that could initiate an infection under the right environmental conditions. Although tourism poses a smaller risk than other avenues of pest introduction (e.g. importation of second-hand harvesters), the numbers of people travelling compounds the relatively small risk to a level that growers should be aware of. The increased mobility of Australian rice growers and visitors to the Australian rice industry, make the adherence to quarantine regulations all the more important. The occurrence of rice blast in California in 1996 (Greer et al. 1997) illustrates that area isolation does not guarantee continued freedom from a particular disease threat.

The virulence of different pathogenic strains of a fungus can vary and the existence of a particular disease, even if its presence is relatively benign, should not lead to complacency in the industry. Stem rot, for example, does not cause the same level of crop destruction in south eastern Australia that it does elsewhere (Cother & Nicole 1999). This may be due to the resistance of the cultivars grown in the region but more likely, it is the result of less virulent strains of the disease being present in the region. Should more aggressive strains of the stem rot pathogen be introduced to south eastern Australia, through a lapse in quarantine procedures or unintentional transmission, the seriousness of the disease and resulting crop losses could increase very quickly.

2.1 Crop monitoring

The price of healthy crops is eternal vigilance. Growers should report any suspected disease symptoms to their district agronomist. Early, accurate identification may prevent a serious disease outbreak.

2.2 Disease resistant varieties

Because we are free of the most important diseases, the susceptibility of our cultivars to any of the main rice disease is not known with the exception of the stem rot disease.

3. DISEASES RISK ASSESMENT FOR SOUTH EASTERN AUSTRALIA

3.1 Disease risk analysis

The aim of a disease risk analysis is to assess and manage the risks due to exotic pests and diseases. A pest risk analysis is generally composed of three different stages. The first stage is to identify the hazard (risk identification or quarantine pests). The second stage is to realise the risk assessment by characterising the risk of entry and establishment of the target pest. The third stage is to do a risk management by reviewing the management options to reduce the risk of introducing the target pest (Phillips et al. 1994). The term "pest" is defined, in accordance with the definition of the International Plant Protection Convention (IPPC 1979), as any form of plant or animal or any pathogenic agent, injurious or potentially injurious to plants or plant products. In this study we will focus on all the major rice pathogens which are already quarantine pests.

3.2 Disease risk assessment

A risk assessment is carried out by characterising the risk of entry and establishment of the target pest. The amount of information available to assess the pest risk will vary for each pest. In some cases, little or no information may be available and research may be needed to obtain the missing information. Finally, the assessment will be limited by the amount of information available on the biology of a particular pest (FAO 1996).

In our study the pest risk assessment was carried out using two softwares developed by the CSIRO, CLIMEX and DYMEX. CLIMEX for Windows Version 1.1 (Sutherst and Maywald 1985; Sutherst et al. 1995) enables users to perform climate comparison by calculating the climatic similarities between any location contained in its database. The CLIMEX meteorological database contains 2031 locations worldwide including 229 locations in Australia. DYMEX is a modular-modelling package composed of a builder and simulator that enables the user to develop and run deterministic population models of biological organisms. The user creates the model under the builder mode then runs it with the simulator (Maywald et al. 1997).

First step - The software CLIMEX was used to assess the suitability of the Australian rice growing area climate is for each pest/disease.

Second step - When the Australian rice growing area climate was found to be suitable for the pest/disease then a pest/disease model was created under DYMEX and run with Australian climatic data.

4. MOST LIKELY EXOTIC RICE DISEASES TO OCCUR IN SOUTH EASTERN AUSTRALIA

4.1 Rice blast (caused by Magnaporthe grisea)

Rice blast, caused by the fungus Magnaporthe grisea, is generally considered the most important disease of rice worldwide because of its destructiveness under favourable conditions. In 1981, a survey from the Commonwealth Mycological Institute reported rice blast in 85 countries. California was among the only rice growing areas remaining free of this

disease (Ou 1985). Since September 1996, California is no longer free of M. grisea. Australia still remains free of this disease.

Lifecycle

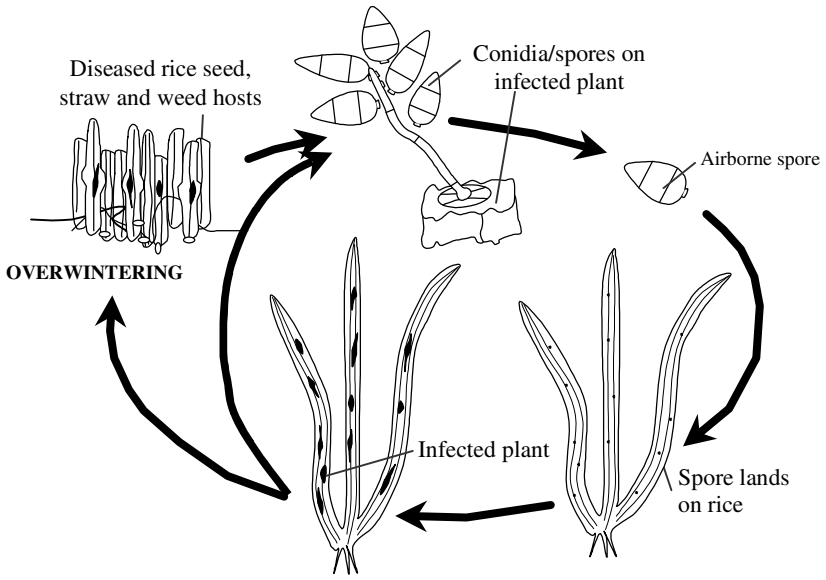

Figure 1: Disease cycle of Magnaporthe grisea

The disease cycle begins when the airborne rice blast spores land on rice plants, infect then produce lesions. After six days, conidiospores are produced and release new airborne spores called conidia. Rice blast is a polycycle disease dependent on favourable weather conditions. Many disease cycles can occur during the rice-growing season. The fungus overwinters as mycelium or conidia on diseased rice stubble, seed or in living plants (Ou 1985). The fungus may also overwinter on winter cereals and weed hosts, but the role of these hosts in the disease cycle remains unclear (Borromeo et al. 1993). Generally, the primary inoculum comes from overwintered rice straw. Conidia are most commonly disseminated by air or water (Ou 1985).

Symptoms

M. grisea can infect and produce lesions on most of the rice plant including leaves, leaf collar, stem, nodes, panicles and grain but not the leaf sheath. The fungus produces lesions or spots. However, their shape, colour and size can vary depending on varietal resistance, environmental conditions, and the age of the lesions (Ou 1985). Five different symptoms of rice blast can be observed on rice plants.

4.2 Leaf blast

On the leaves, the spots are elliptical or diamond shaped with more or less pointed ends. The lesions are 1-1.5cm long, 0.3-0.5 cm board with a grey or whitish centre and a brown or reddish brown margin (Ou 1985).

Figure 2: Leaf blast symptoms (D. E. Groth)

Leaf blast can occur and cause the death of susceptible cultivar from seedling to tillering stage (Ou 1985).

4.3 Collar Rot

Collar rot symptom occurs when the lesions develop at the junction of the leaf blade and sheath.

Figure 3: Collar blast symptoms (D. E. Groth)

In the worst scenario, it can cause the death of the flag or second to last leaf which significantly reduce the yield potential.

4.4 Neck Rot and panicle blast

Infected part of the panicles turn brown or sometimes black. Attacks below the panicles cause neck rot which can result with the death of the all panicle if it occurs early (Ou 1985).

Figure 4: Neck rot symptoms (D. E. Groth)

Figure 5: Panicle blast symptoms (D. E. Groth)

4.5 Node infection

The infected nodes appear brown-black, dry, resulting with the death of the plant (Ou 1985). Climate and more particularly moisture are mainly responsible for these differences in symptom expressions.

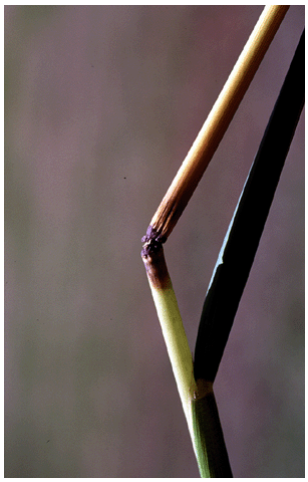

Figure 6: Node infection symptoms (D. E. Groth)

Leaf blast is more commonly observed in temperate rice growing area whereas in dry and hot climate, M. grisea only infect nodes causing the so-called Iraqi 'Shara' disease. Neck rot appears less specifically (Ou 1985).

Damage

Rice blast is considered as one of the most serious disease of rice. If an early rice blast outbreak occurs from the seedling to the tillering stage, the rice plants are generally killed. Later attack almost always results in yield and quality losses (Ou 1985).

Favourable conditions

The disease is favoured by long periods of leaf wetness, high relative humidity and temperatures around 24oC and 28oC. The attack severity is also correlated with high nitrogen applications (Hori 1898) and drought (Hemmi & Abe 1932).

Control strategy

An integrated management program is the best solution available for rice blast control. It includes good cultural practices and chemical control. Resistant varieties are readily available but it seems that the type of resistance available could lead to development of new and damaging races of the blast fungus (Ou 1985).
Growers are also advised to use uncontaminated rice seed. Buying certified seed is generally the best guarantee but it still won't protect the rice from other inoculum sources. Excessive nitrogen applications are favourable to rice blast so nitrogen should be well managed. Permanent moderate to deep water flooding without field drainage can reduce the impact of the disease. Benlate(r) (Benomyl) is the only registered fungicide in southern USA. A new fungicide from Zeneca called Quadris(r) (Azoxystrobin) seems to control the disease as well as, or better than Benlate(r). In contaminated areas, rice stubble and straw should be burnt in order to reduce the overwintering inoculum. A significant reduction of the primary inoculum will only be achieved if the vast majority of rice paddocks are burned (Greer et al. 1997).

Probability of entry

Wind dispersal

Rice blast is usually distributed throughout rice growing areas by air currents picking up the conidia to infect neighbouring fields. M. grisea has already been observed in New South Wales infecting several different weeds, therefore, spores could be blown and dispersed from these weeds to rice fields. The ability of isolates of M. grisea from weeds to infect rice crops and vice versa is subject to controversy. This controversy is mainly explained by the genetic diversity of the fungus, the hosts and the variation in the experimental conditions, such as soil fertility (Ou 1985). It is not known if virulent strains of M. grisea could be introduced to northern Australia by tropical cyclones as the maximum distance that conidia can travel remains a controversial issue (Teng et al. 1991).

Transport of infected plant material

In temperate areas, rice blast mycelium and conidia can overwinter on diseased crop residue, seed and living plant tissue. In rice paddies the main source of inoculum is from rice straw and stubble. Under dry conditions at room temperature, conidia are able to survive for more than a year and mycelium for almost three years (Kuribayashi 1928). Due to its lengthy survival, importation or transportation of contaminated seed, weeds or souvenirs made of rice straw by tourists could be a way for blast to be introduced to the rice growing area of Australia.

Travellers

Philips et al (1992) estimated the potential number of M. grisea conidia introduced to Australia on the clothes of international travellers to be 240,000 million for 1988 alone. M. grisea is already present in several Australian states, including NSW, therefore, domestic travellers could also involuntary introduce conidia to the rice growing regions.

Probability of establishing

The rice-growing areas of Australia are situated in southern New South Wales (NSW) and northern Victoria on fertile plains irrigated from the Murray and Murrumbidgee Rivers (Figure 1). Rice is grown in rotation with other cereals or pasture and is sown during October-November and harvested in March-April. Rice blast, caused by the fungus Pyuricularia grisea (T. T. Herbert) Yaegashi & Udagawa (anamorph Magnaporthe grisea (Cooke) Sacc. syn. P. oryzae Cav.), is generally considered the most important disease of rice worldwide because of its extensive distribution and destructiveness under favourable conditions. Infection of rice plants occurs from airborne conidia and symptoms appear as lesions or spots (Ou 1985). In 1981, a survey from the Commonwealth Mycological Institute reported rice blast in 85 countries (CMI 1981). Although M. grisea is favoured by humid and wet climatic conditions, this pathogen is characterised by a great adaptability to environmental conditions. Thus, blast has been reported under the hot and dry climate of California (Greer et al. 1997) and Iraq (Ou 1985). South eastern Australia is the only rice-growing area in the world free of the disease but the potential risk for rice blast is uncertain. Rice blast has never been observed in the Australian rice-growing area but M. grisea has been reported on weeds in several Australian States including NSW (Priest, personal communication). Nevertheless, none of the reports of M. grisea were within, or close to, the rice-growing area of NSW.

M. grisea is already present on Australian weeds but its virulence on the rice varieties currently grown in NSW has not yet been determined. Even if Australian strains were unable to infect rice, virulent strains from overseas might be accidentally introduced to Australia.

Methods

In this study, two software programs, CLIMEX and DYMEX (CSIRO, Brisbane, Australia) were used to investigate the risk M. grisea represents to the Australian rice industry. The predictive bio-climatic modelling software CLIMEX was used to assess the suitability of the Australian climate for development of M. grisea. A rice blast model was then created with the software DYMEX to predict the behaviour of the pathogen in the rice-growing areas of Australia.

4.6 Climex

As climate is one of the major factors limiting the distribution of diseases and pests, it can be useful to predict the probability of a disease successfully spreading into a new area. CLIMEX for Windows Version 1.1 (Sutherst and Maywald 1985; Sutherst et al. 1995) enables users to perform climate comparison by calculating the climatic similarities between any location contained in its database.
The climate of the Australian rice-growing area was represented by Deniliquin. Deniliquin's climate was then compared with the rest of the world and locations shown on a map. To only

compare the climate during the rice-growing season (October to April), the comparison was limited to the October-April period. CLIMEX can automatically compare locations from different hemispheres by displacing the data from the southern hemisphere by six months. Maximum and minimum air temperatures, total rainfall, rainfall pattern and relative humidity were used for the comparison. The product of these comparisons were expressed on a world map as "Match Indices", scaled between 0 (no similarity) and 100 (a complete match). To obtain a reasonable number of match locations, the Match Level was set at 0.6. This means the only locations shown on the map had (Figure 7) a Match Index equal to or above 60%. The four Australian locations (Table 1) were then compared with a modified and updated world distribution map of M. grisea.

Table 1: Weather stations used to test and run the DYMEX model of rice blast.

Station number	Station name	Latitude	Longitude
#32	Colusa A.A	39.14 (N)	122.1 (W)
#61	Orland2.A	39.42 (N)	122.9 (W)
074253	Finley, CSIRO	35.64 (S)	145.56 (E)
075028	Griffith, CSIRO	34.32 (S)	146.07 (E)
075031	Hay, Miller Street	34.52 (S)	144.85 (E)
074133	Yanco, Exp. Farm	34.60 (S)	146.40 (E)

4.7 Dymex

DYMEX is a modular-modelling package composed of a builder and simulator that enables the user to develop and run deterministic population models of biological organisms. The user creates the model under the builder mode then runs it with the simulator (Maywald et al. 1997).
The rice blast model was created from data in published research and was run with meteorological data (daily minimum and maximum temperature, minimum and maximum relative humidity and rainfall) obtained from four locations in the southern NSW rice-growing area (Table 1).

Model : description (See Appendix 1 & 2)

The rice blast model is composed of six standard modules plus the Lifecycle module that contained the disease cycle information for M. grisea. The Module Timer performs the timekeeping functions of the model. The Module Meteorological DataFile Reader (MetBase) is a specialised data file module containing the meteorological data. The module Latitude is a QueryUser module necessary for the Daylength module.
The module Daylength calculates the number of hours between sunrise and sunset given the latitude of the location and the date. The Module daily temperature cycle (circadian) generates a variable that describes the diurnal change in temperature. The most accurate way

to simulate the temperature cycle is the cycle shape Composite (Sine+Exponential). The Module daily relative humidity cycle (circadian) generates a variable that describes the diurnal change in the value of the humidity. A Sine cycle shape was selected to calculate this module.
The Module Lifecycle is used to create the disease lifecycle. Our rice blast lifecycle was composed of two lifestages, spore and disease.

Data for the spore lifestage

Spores of M. grisea are easily killed by UV radiation (Manibhushanrao and Suryanarayanan 1971) so spore mortality in the model was simulated by a step function with a threshold of two days (all spores die 2 days after sporulation).
The infection process of M. grisea requires adequate temperature, humidity and free water. Hashioka (1965) found that a minimum of 10 hours at 32oC, 8 hours at 28oC or 6 hours at 24oC was required for conidial germination and the penetration of germ tubes into the host cells. Spore germination and formation of appressoria occur between 10 and 33oC (Suzuki 1975) therefore in the model the infection process can only occur between 10 and 33oC. The influence of temperature on the infection process was simulated under DYMEX with a three segment linear function. Firstly, the above data had to be changed into a rate per 24 hours (infection rate of development). This was calculated as follows; at 10oC the infection rate of development was 0 (10oC ® no infection = 0 days for 1 infection, at 10oC the infection rate was 1/0 = 0); at 24oC, the infection rate of development was 4 (at 24oC it takes 6 hours for infection = 6 hours/24 hours = 0.25 days for 1 infection, the infection rate is 1 day/0.25 days = 4); at 28oC the infection rate of development was 3.03; at 32oC it was 2.43 and at 33oC it was zero. The slopes in the three segment linear function were 0.285 between 10oC and 24oC, -0.196 from 24ºC to 32oC, and -2.43 from 32oC to 33oC.
High relative humidity (Hemmi and Imura 1939), and free water (Ou 1985), are required for conidial germination. Therefore a step function with a threshold of 100% humidity was created in the model. In the model, infection could only occur if the relative humidity was equal to 100%.
The transfer stage from spores to disease required that the temperature and relative humidity were combined as a product meaning both had to occur simultaneously for infection to occur.

Data for the disease lifestage

Temperature conditions reported for disease development were similar to those reported for infection (Ou 1985). This was simulated by the same three segment linear function used in the spore lifestage.
Kato and Kozaka (1974) found that spore production was correlated with temperature and an IRRI study (unpublished data) found that a typical lesion can generally produce up to 84,000 conidia. In the model, the lesion fecundity (the total number of possible spores that can be produced per lesion) was therefore set at 84,000.
Kato and Kozaka (1974) reported, over a 30 day period, a spore production of 2962 spores at 16oC, 11975 spores at 20oC, 8148 spores at 25oC and 3950 spores at 32oC. The influence of temperature on the sporulation process was simulated under DYMEX with a three linear segment function but firstly the above data to be changed into a rate per 24 hours (sporulation rate of development). This rate was calculated as follows; at 16oC the sporulation rate of development was 98 spores/day (16oC ® 2962 spores / 30 days = 98 spores produced per day); at 20oC the sporulation rate of development was 399 spores/day (11975/30); at 25oC, the infection rate of development was 271 spores/day (8148/30); at 32oC the infection rate of development was 131 spores/day (3950/30). The slopes in the 3-segment linear function were 75.25 between 16oC and 20oC, -25.6 from 20oC to 25oC, and -20 from 25oC to 32oC.

Kato and Kozaka (1974) also demonstrated that lesions produce spores for about 30 days after a latent period of about 6 days. The latent period was simulated by a pulse function with a threshold of 6 days and a pulse width of 30 days. Therefore in the model, the lesions can only sporulate 6 days after the infection event and for a maximum of 30 days. As mature lesions are capable of sporulating when relative humidity is higher than 89% (Hemmi and Imura 1939), a step function with a threshold of 89% humidity was created in the model. In our model the lesions can sporulate only if the relative humidity is = 89%.
The sporulation required that the temperature and relative humidity and chronological age of the lesions were combined as a product meaning all these conditions had to occur simultaneously for lesions to sporulate.

Model: simulation

Climatic data required to run the model were the daily rainfall, minimum and maximum temperatures, and the minimum and maximum relative humidity. Meteorological data from Finley, Griffith, Hay and Yanco were purchased from the Australian Bureau of Meteorology to run the model. Any missing data were estimated by the estimation function of DYMEX. The latitude module was initialised for each station (Table 1). The model was run from 01/10 to the 15/04 (rice-growing period) for each year between 1988 and 1999. The simulation was initiated with 100 lesions (starting 01/10 for Australian locations and 01/05 for California locations). The number of infection events (1 event = 1 day) were recorded on an annual basis. The month of the last infection was also recorded every year.

Model: validation

In order to validate this model, two meteorological stations (Colusa and Orland) in the Sacramento Valley, California (Table 1) were chosen. The Sacramento valley was chosen due to its similarity (61%; see CLIMEX results) with the Australian rice-growing area of Deniliquin and because before 1996, California was the only other rice-growing area free of rice blast (Webster, personal communication). Rice blast was reported in California for the first time in 1996 (Greer et al. 1997). The outbreak was located south east of Willows (Glenn County) and north west of Colusa (Colusa County), on 7000 acres. The figure increased to 57,000 acres the following year. In 1998, 1999 and 2000, the infection levels were recorded as low. The met station of Orland was chosen as no rice blast outbreak has ever been reported in this area (Mutters, personal communication).
In California, rice is generally sown in late April and harvest begins at the end of September. Meteorological data from Colusa was retrieved from the Internet (http://www.imp.ucdavis.edu/WEATHER/wxretrieve.html). Data from 1984 to 1999 were used to validate the model over a period from 01/05 to the 30/09. The number of potential infection events (1 event = 1 day) were recorded on a monthly and annual basis.

Results

CLIMEX: Deniliquin climate projected to the rest of the world

The projection of the climate of Deniliquin to the rest of the world with a Match Level (60% is displayed in Figure 7. When compared with Deniliquin, 91 locations worldwide (excluding Australia) had a match index (60%.

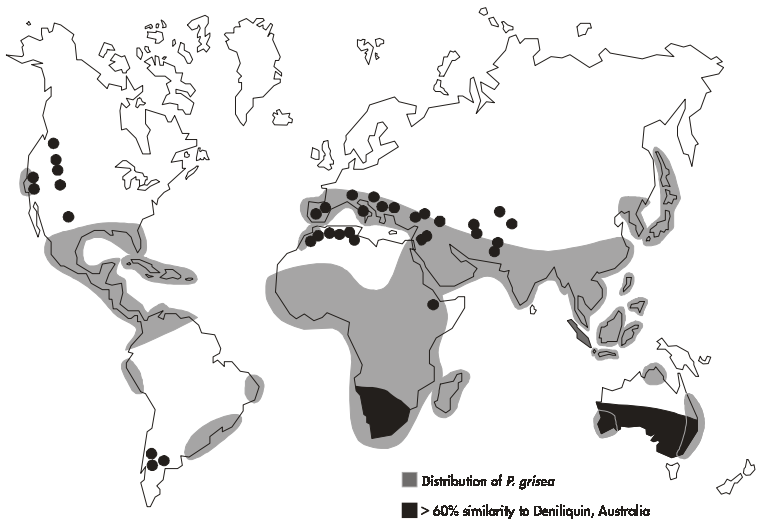

Figure 7: Worldwide locations with a (60 % similar climate to Deniliquin, Australia (October to April) overlayed with a distribution map of Magnaporthe grisea.

The vast majority of these 91 locations were within the known distribution area of M. grisea (Figure 7). The climate of Sacramento was found to have a 61% index similarity with the climate of Deniliquin. (Figure 7). During their respective rice-growing season, the temperatures are almost identical in both Deniliquin and Sacramento but the climate is much drier in Sacramento (Table 2).

Table 2: Long term climate averages of Sacramento, USA and Deniliquin, Australia. Source (CLIMEX long term database).

Station	Sacramento	Deniliquin
Summer (Jan) max/min temp.	32.2ºC/14.4ºC	31.5ºC /15.9ºC
Winter (July) max/min temp.	11.1ºC/3.9ºC	13.9ºC/3.3ºC
Mean temp. during -		
the rice-growing season	20.8ºC	20.2ºC
Total rainfall	473mm	409mm
Total rainfall during -		
the rice-growing season	31mm	218mm

DYMEX: Model validation

In Colusa, simulated infection events showed that conditions for M. grisea were favourable from 1995 to 1999. The number of infection events increased each year from 1994 to 1996, it

decreased to six in 1997 and then peaked in 1998 where 29 infection events were recorded. The minimum number of infection events (three) occurred in 1995 (Figure 8).

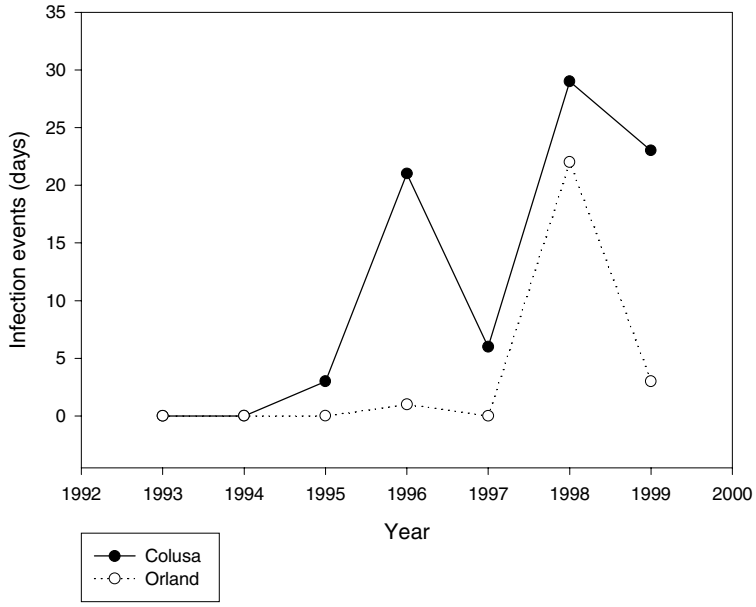

Figure 8: Model validation. DYMEX simulated yearly infection events of Magnaporthe grisea for Colusa and Orland, USA from 1993 to 1999.

In Orland, the model showed that the climatic conditions were largely unfavourable for M. grisea from 1993 to 1997 as only one infection event was predicted by the model. With respectively 22, three and one infection events, 1998, 1999 and 1996 were the only three seasons favourable (= 1 infection event) for M. grisea (Figure 8).

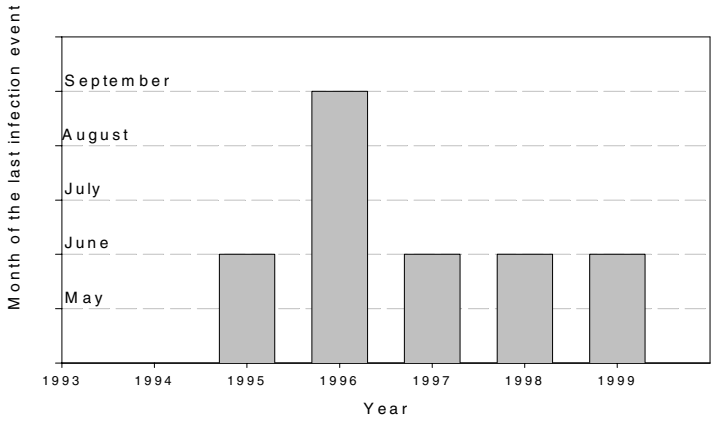

Figure 9: Model validation. DYMEX simulated month of the last infection event of Magnaporthe grisea in Colusa, USA from 1993 to 1999.

In Colusa, for the majority of the years, the last simulated infection event occurred within 2 months of sowing (Figure 9). However, in 1996, the last simulated infection event in Colusa occurred in September meaning that infection conditions were favourable throughout the entire rice-growing season.

DYMEX: Model simulation

In Australia, simulated infection events showed that Yanco was the most favourable location (9 years having (1 infection event) for M. grisea whereas Griffith was the least favourable location (2 years having (1 infection event).

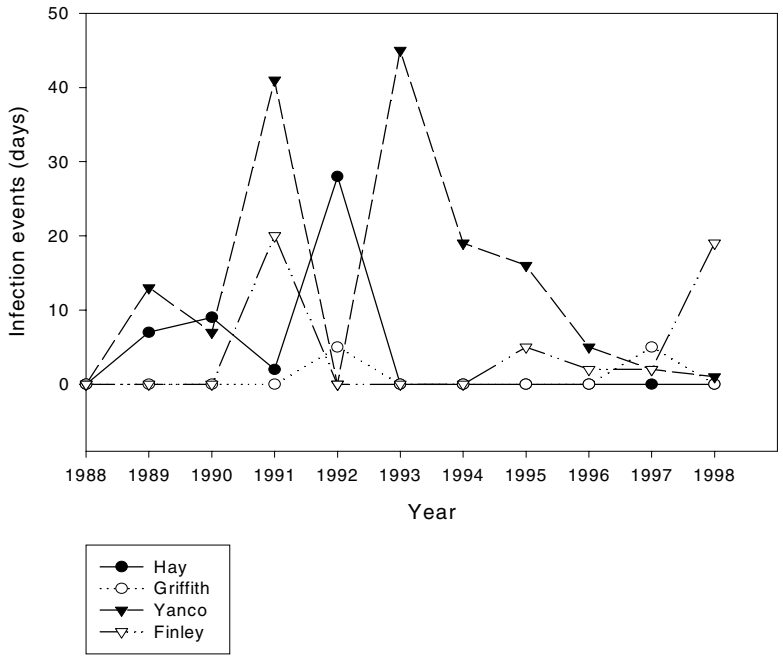

Figure 10: Model simulation. DYMEX simulated yearly infection event of Magnaporthe grisea in southern NSW from 1988 to 1999.

The maximum number of infection events occurred in 1993-94 at Yanco (45 infection events) in 1991-92, at Yanco (41) and in 1992-93 at Hay (28). However, 80% of the years displayed had less than 10 infection events (Figure 10).

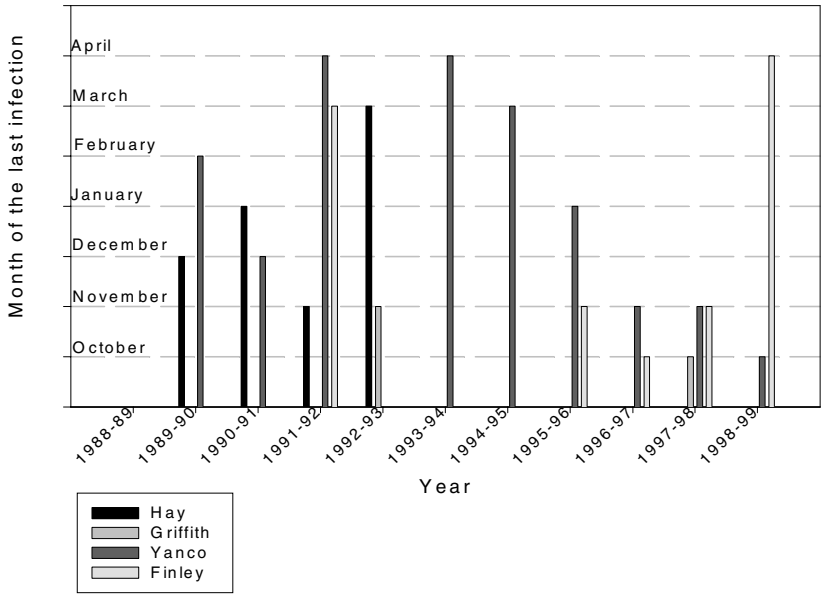

Figure 11: Model simulation. DYMEX simulated month of the last infection event of Magnaporthe grisea in southern NSW from 1988 to 1999.

For those years and locations that had favourable conditions for M. grisea, 80% of the last simulated infection events occurred within 6 months of sowing. The last simulated infection event for Yanco in 1991-92 and 1993-94 and for Finley in 1998-99, occurred in April, meaning that infection conditions were favourable thoughout the entire rice-growing season (Figure 11).

Discussion

CLIMEX has been successfully used in Australia and in the USA to assess the potential establishment of weeds, fungi and insects (Julien et al. 1995; Venette and Hutchison 1999). This is the first report of the use of both CLIMEX and DYMEX to conduct a comprehensive risk assessment of an exotic plant pathogen.

The Deniliquin climate projection to the rest of the world revealed that most of the matching locations were within the known distribution area of M. grisea, including Sacramento, the latest rice-growing area to be infested by the pathogen. ***The climatic conditions prevailing in the Australian rice-growing area can, therefore, be considered as favourable for M. grisea and the potential for rice blast to occur within the Australian rice-growing areas appears to be relatively high.***

Some validation of the blast model was achieved with the Californian data as it detected the 1996-99 Californian infection events of M. grisea near Colusa (Figure 8 & 9) and did not, with the exception of 1998, display many infection events for the disease-free area near Orland, Glenn County (Figure 8). In Colusa, the model recorded several infection events in 1995 whereas no disease was reported from California. It is not known if the pathogen was present before 1996 but it seems probable that it took at least one year for the pathogen to increase and disease to become noticeable. The model also displayed a maximum of potential infection events in 1998 whereas in the field, 1996 and 1997 were the most severe years (Mutters, personal communication). Field events may have been reduced in 1997, 1998 and 1999 as preventive measures aiming at reducing the incidence of rice blast were taken by

Californian authorities just after the 1996 rice blast outbreak. From 1996 to 1998, rice growers were allowed to burn limited acreage of infected rice fields to try to prevent the spread of the disease by reducing the amount of inoculum. Then, in July 1997, the California Department of Pesticide Regulation temporarily granted the registration of the fungicide Quadris(r) (azoxystrobin) for the control of rice blast (Mutters, personal communication).
In Colusa, the model showed that for most of the years when the disease was seen in the Californian rice fields, the last infection event occurred within 2 months of sowing (Figure 9). This tends to indicate that a 2 - month period of favourable conditions is enough for the disease to become noticeable in the field. In 1997, a 2 - month period (with 6 infection events) was enough for the disease to become noticeable in the Californian rice fields (Figure 9). When applied to Australia, this low threshold would mean that the disease would have been noticeable during six seasons at Yanco, three at Hay, two at Finley but none at Griffith. The model simulation showed that for 10 out of 11 rice-growing seasons, the climatic conditions were favourable for rice blast to occur in at least one of the four locations of south eastern Australia studied in this paper. The disease was found to be able to occur generally over several months. In three years (Yanco 1991-92, Finley 1998-99 and Yanco 1993-94), the disease would have been able to occur over most of the rice-growing season. Those same years and locations also experienced a very high number of infection events, so that the disease incidence would have probably been very important in the field. The most favourable simulated location for rice blast was Yanco whereas Griffith was the least at risk. This is because the main trigger for M. grisea sporulation and infection is the level of relative humidity, and the level of humidity was generally higher in Yanco than for the three other locations during the 11 years investigated.
The meteorological data used to run both models came from meteorological stations remote from actual rice fields. The meteorological conditions in a rice paddy have been found to be different from the data recorded in a standard meteorological station, especially in relation to the relative humidity under the rice canopy (Webster, personal communication). If the relative humidity under the rice canopy is higher than ambient, then infection and spore production events would be higher and more frequent. To assess the microclimate of waterlogged rice fields, data loggers should be placed in rice fields to record more realistic meteorological data needed to run the model. A rice blast forecasting system has already been developed to use real time climatic information from data loggers installed in rice fields (Park & Kim 1995). Other models have also been developed to forecast epidemics, potential yield losses (Luo et al. 1997) and severity of the attack (Hashimoto et al. 1984). Such data loggers were placed in a rice field in Yanco by the authors during the 2000-01 rice-season. Important differences were found between the Stevenson screen placed on the rice field bank and the rice canopy. It was found that the average humidity was at least 20% higher in the rice canopy than in the Stevenson screen. The results were also found to be quite variable which indicates that the difference in humidity is probably influenced by other factors such as windspeed, rainfall and evapotranspiration. Long term data collection would be necessary and other parameters potentially influencing humidity should also be recorded. If these relative humidity differences are confirmed in the future, our study might underestimate the potential risk for rice blast to occur in south eastern Australia.
In any modelling exercise there are some inherent shortcomings, which include the unavailability of accurate in-crop climatic data. As an example, leaf wetness data were unavailable and had to be estimated by the relative humidity. Another major problem in disease forecasting is the validation of the disease forecasting model. With our model, a complete validation would require comparison of our model prediction with the actual number of events that occurred in Colusa. To the best of our knowledge, the actual number of infection events has never been recorded in California. Despite these limitations, the rice blast model provides a method for estimating the threat that rice blast represents to south eastern Australia.

Results from both CLIMEX and DYMEX underlined the potential for rice blast to occur and threaten the rice industry of south eastern Australia. Rice cultivars should be tested to estimate the threat posed by local Australian strains of M. grisea to rice crops and strict quarantine vigilance on M. grisea (and other quarantine organisms) should be maintained to preserve the competitiveness of the Australian rice industry.

Total risk

The risk of rice blast to establish and to occur in the Australian rice-growing region appears quite high.

4.8 Kernel smut (caused by Tilletia barclayana)

Kernel smut was reported from Japan (Takahashi) in 1896 and from the USA (Anderson) in 1899 (Ou 1985). The nomenclature of this fungus is still a subject of controversy. It is commonly referred as Tilletia barclayana (Bref.) Sacc. & Syd. In Sacc. but some authors refer to it as Neovossia horrida. This disease is now known to occur in most Asian countries, in Central America and in Western Africa (Ou 1985). The disease is now widespread in California's rice growing areas (Webster, personal communication).

Lifecycle

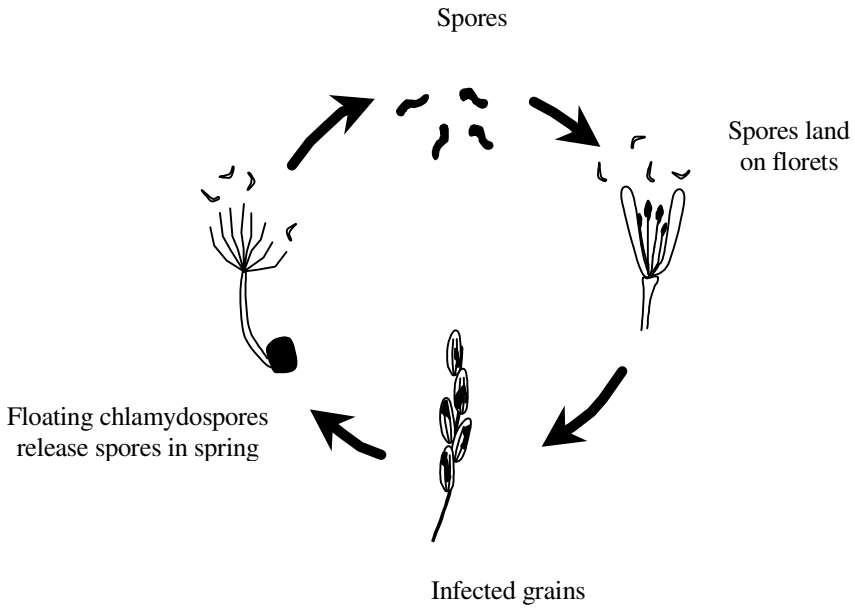

Figure 12: Disease cycle of Tilletia barclayana

Kernel smut survives as chlamydospores in infected grains, crop residue and in the soil. Chlamydospores have been found to survive at least 3 years in stored grains. In spring, they float on the water surface, germinate and produce primary sporidia. Secondary sporodia are produced and projected in the air. Wind helps this second inoculum to reach the rice panicles. Ovaries can be infected if the florets are open. Contrary to other diseases caused by Tilletia species, kernel smut is not borneseed (Ou 1985).

Symptoms

The smutted grains are loaded with mass of black spores. The symptoms are more noticeable in the early morning when dew causes the infected grains to burst and release their loads of chlamydospores (Ou 1985).

Figure 13: Rice grain presenting symptoms of kernel smut (left) and healthy rice grains (right) (D. E. Groth)

Damage

Kernel smut only infects the florets. The disease reduces both yield and quality. Even if yield losses as high as 15% have been recorded, kernel smut is generally not considered as an economically important disease of rice (Whitney 1992).

Favourable conditions

Excessive nitrogen applications and excessive flood depth favour the disease (Templeton 1963).

Control strategy

The use of tolerant cultivars is the only known method to control kernel smut. In the USA, surveys showed that short-grain cultivars appear to be the most susceptible whereas short grain cultivars are the most resistant (Whitney 1992).

Probability of entry

Kernel smut has been reported on rice at Mareeba, Queensland (Tonello 1980). The disease is now widespread in California but is considered a minor disease of rice. Used farm equipment from overseas, transportation of rice material and farmers travelling to other rice growing area pose the greatest threat of introducing kernel smut in Australia.

Probability of establishing

According to the bioclimatic program software CLIMEX, the climate occurring in the Californian rice growing is relatively similar to the climate of southern NSW/northern Victoria. T. barclayana has never been reported in either NSW or Victoria but an accidental introduction of this pathogen in the rice growing regions would probably lead to kernel smut outbreaks.

Total risk

The risk of kernel smut to occur in the Australian rice growing regions is high.

4.9 Root nematodes (Hirschmanniella spp.)

At least five species of the genus Hirschmanniella are known to be of economic importance for rice crops (Prot 1992).

Life cycle

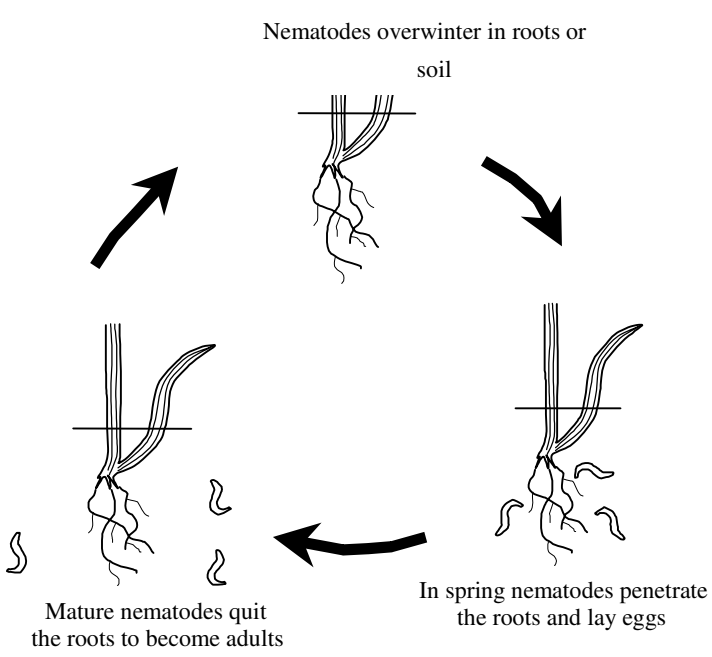

Figure 14: Disease cycle of Hirschmanniella spp.

Shortly after sowing, the nematodes enter the seedlings and migrate to the root system where the female nematodes lay they eggs. Juveniles hatch and develop in the root system then some of them migrate to the soil around the panicle heading stage. Some species of nematodes have one generation but others have two generations per year (Prot 1992).

Symptoms

There is no specific symptom of a root nematode attack except a reduction of plant growth. The number of tillers can also sometime be reduced (Prot 1992).

Damage

Yield losses are estimated to be ranging from 10 to 35 % (Prot 1992).

Favourable conditions

Excessive nitrogen applications favour these nematodes (Ou 1985).

Control strategy

The use of tolerant rice varieties and applying nematicides are most successful methods available to control Hirschmanniella spp (Prot 1992). The susceptibility of the Australian rice varieties to these nematodes is unknown.

Probability of entry

To our best knowledge, Hirschmanniella spp. has never been reported in Australian rice crop. Nematodes can be disseminated by various means such as tools and machinery, soil, wind, irrigation, animal and infected plants.

Probability of establishing

Hirschmanniella spp. are present in most of the rice-growing areas of the world. The rice-growing practices in south eastern Australia appears to be favourable for Hirschmanniella spp., therefore the probability of establishing in the Australian rice-growing area is probably medium to high.

Total risk

It is not easy to classify the total risk root nematodes represent to the Australian rice-growing as little is known about Hirschmanniella spp. The total risk would probably be between low to medium.

5. DISEASES PRESENT IN SOUTH EASTERN AUSTRALIA

5.1 Damping off (caused by Pythium and Achlya spp.)

Water mould, also called damping off, is the most important disease in the Australian rice growing regions. The disease is generally caused by a complex of fungi such as Achlya spp., Pythium spp. and Fusarium spp. In Australia, the disease has been attributed to Pythium infection and more particularly P. arrhenomanes and P. irregulare (Cother and Gilbert 1992).

Lifecycle

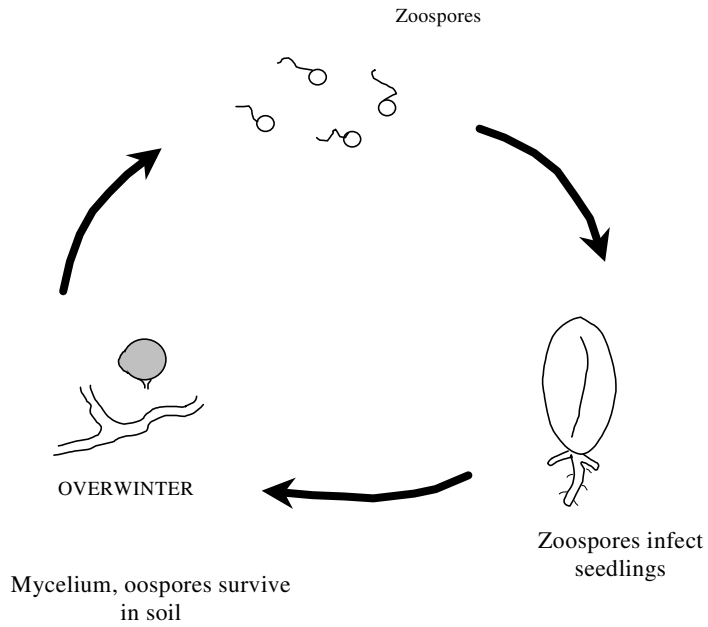

Figure 15: Disease cycle of Pythium spp.

Between rice crops, the fungi survive in the soil as mycelium or oospores. In spring, rice field flooding induces zoospore production. The zoospores reach the rice seeds and infect the radicle or the endosperm through seed cracks (Ou 1985).

Symptoms

Damping off results in poor seedling establishment. The attack causes the death of the seedling before emergence or death of the coleoptile within a few days of emergence. Infected seeds and seedlings are covered with a whitish cottony like mycelium (mould). After germination infected seedlings become yellowish, growth is reduced and death generally follows (Ou 1985).

Figure 16: Damping off symptoms on rice seedlings (D. E. Groth)

Damage

In Australia the rice sowing rate of around 600 seeds/m2 generally results in only 300 established plants (Cother & Gilbert 1993). It is thought that damping off is mainly responsible of this low establishment rate. Cother & Gilbert (1993) found that P. arrhenomanes could reduce shoot and root growth by 48 and 70 % respectively.

Favourable conditions

Damping off is favoured by any factor reducing germination and growth. Cold/cool temperatures at or after sowing are generally the main factor leading to the disease. Slow growing and/or non-vigorous cultivars are more susceptible to damping off (Ou 1985).
In Australia, P. arrhenomanes and P. irregulare are more frequently present in heavy soils such as black or grey soils (Cother & Gilbert 1992).

Control strategy

Selecting vigorous cultivars and using dusted seed as well as good sowing techniques reduce the impact of damping off (Rush 1992).

5.2 Stem rot (caused by Sclerotium oryzae)

The stem rot fungus was discovered in Italy by Cattaneo (1876) and given the name of Sclerotium oryzae. In the 1910s, 1920s the disease was reported in Asia and in the USA. Stem rot is serious disease of rice, which can occur in both tropical and temperate regions. It now occurs in most of the rice growing areas of the world (Ou 1985).
Stem rot disease was first observed in New South Wales in the summer of 1995/96 (Watson & Priest 1998). The main rice varieties grown in Australia and two breeding lines were tested for their susceptibility to S. oryzae by Cother & Nicol (1999). Even if three cultivars (Kyeema, Langi and Millin) had their yield reduced up to 30%, the strain of S. oryzae currently present in Australia was not considered particularly virulent.

Lifecycle

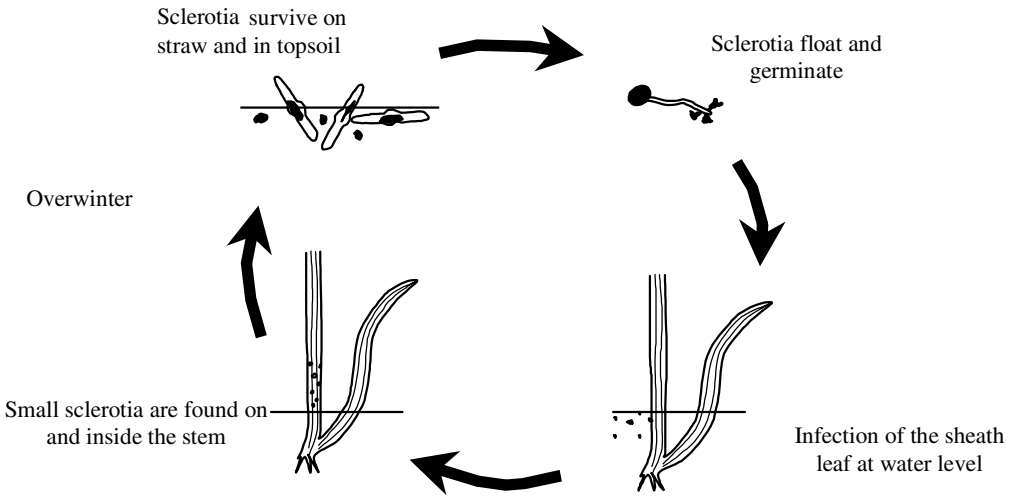

Figure 17: Disease cycle of Sclerotium oryzae

Sclerotia overwinter and can survive several years in rice straw or in the soil. Tullis & Cralley (1941) demonstrated that a small percentage of buried sclerotia were still viable even after 6 years in the soil. In spring, when the rice paddy is flooded, the sclerotia float on the water surface and infect the rice sheaths at the water line level. The fungus produces conidia and ascospores that may induce additional infections. A large quantity of sclerotia is produced on the different diseased tissues (Ou 1985).

Symptoms

Irregular black lesions on the outer sheath leaves located at the water level are generally the first symptoms observed in a rice field. These symptoms do not most of the time appear before the mid-tillering stage. The fungus progresses through the leaf to finally reach and rot the culm. Many small black sclerotia are produced on the infected areas and also inside the culms (Ou 1985).

Figure 18: Young stem rot symptoms (D.E. Groth)

Figure 19: Aged stem rot symptoms (D.E. Groth)

The intensity of the attacks generally increases and reaches its peak during the harvest time. In the tropic the fungus still continue to grow and to produce sclerotia after the harvest. Depending on the severity of the attack, the damages range from unfilled grain to the death of the tiller (Ou 1985).

Damage

The disease attacks the sheath and the culm of rice plants. The attack leads to lodging, which mainly reduces the weight and the quality of the grain. In the Philippines, Hernandez (1923) reported yield losses as high as 80%. Generally, the yield losses caused by the stem rot range between 10 to 75% (Webster 1992).

Favourable conditions

Krause and Webster (1973) demonstrated the correlation between disease incidence and inoculum density. Fertilisation influences disease severity. Cralley found that nitrogen overfertilisation increase the disease severity. Phosphorus had the same effects as Nitrogen whereas a high level of potassium counter balanced the nitrogen overfertilisation effect. Kobari (1961) found a positive correlation between stem borer attacks and stem rot severity.

Control strategy

The amount of inoculum can be partly reduced by burning or ploughing straw stubble after the rice harvest (Webster et al. 1976). Rice varieties differ in susceptibility so growers are recommended to choose an appropriate cultivar in infested area. Good nitrogen management can also reduce the risk of a severe stem rot attack (Ou 1985).

5.3 Glume blotch (caused by Pseudomonas syringae pv. syringae)

Pseudomonas oryzicola was first found on the leaves sheathing the panicles of rice by Klement (1955) in Hungary. In 1971, Visnyovsky et al demonstrated that P. oryzae was in fact P. syringae pv. syringae. The disease has been reported in China (Fang & Ren 1960), Japan (Funayama & Hirano 1963), and Indonesia (Goto 1965). Cother reported the first attack of the bacteria in Australia in 1974.

Lifecycle

Little information is known of the lifecycle of P. syringae pv. syringae on rice.

Symptoms

The bacteria can be found on the leaves sheathing the panicles, on the glumes and also on the stem. On the sheath, the bacterium causes water-soaked, dark green, elongated spots which can turn dark brown - red. On the glumes, discolouration occurs from early to late maturing. Small dark brown spots surrounded by green to brown tissue are noticeable on the florets of the panicle. In severe case, the stem may collapse (Cother 1974).

Damage

The disease can reduce the yield and quality of the grain but is considered as a minor disease of rice in Australia (Cother 1974).

Favourable conditions

The favourable conditions for the disease are unknown.

Control strategy

None.

5.4 Downy mildew (caused by Sclerophthora macrospora)

Downy mildew has been recorded in many countries worldwide (Ou 1985). In Australia, this disease was first found on rice in Yanco in 1930 (Noble et al. 1935). It occurs sporadically since then. Downy mildew has a wide host range but it is generally considered as a minor disease of rice.

Lifecycle

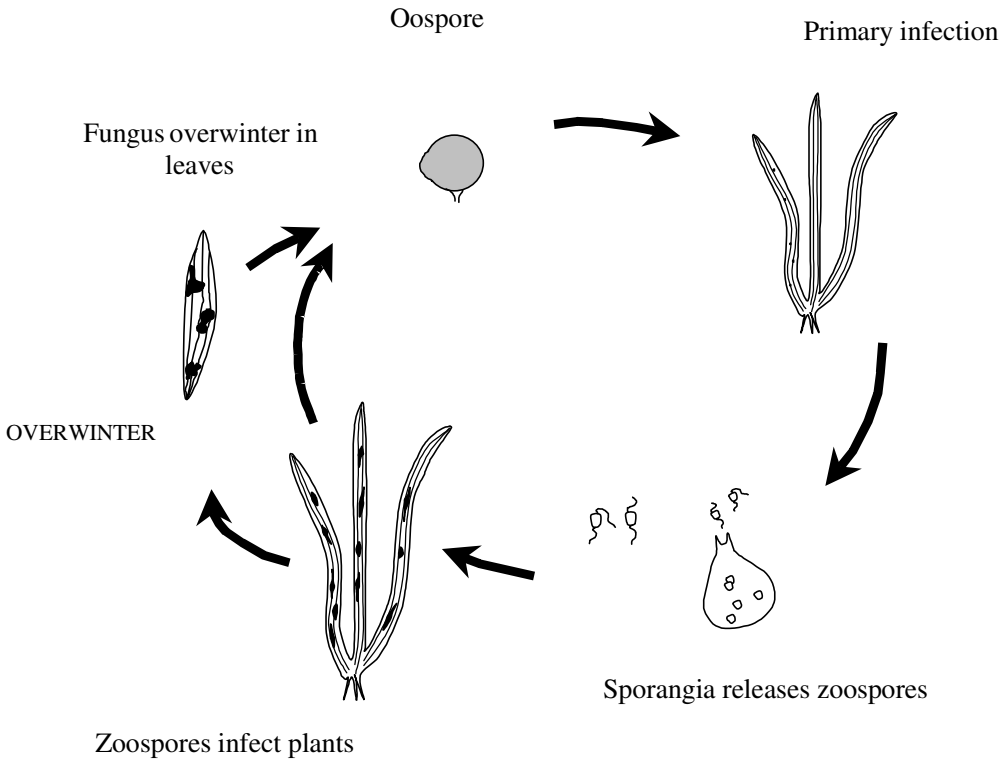

Figure 20: Disease cycle of Sclerophthora macrospora

S. macrospora overwinters as oospores in infected rice residue and also in host plants including rye, barley, maize, millet, oats, wheat and many grasses. In spring oospores germinate and infect rice seedlings. Secondary infections are caused by zoospores released by sporangia (Ou 1985). Host plants are generally considered as an important source of primary inoculum (Katsura 1965).

Symptoms

Seedling can be readily infected but symptoms are generally more noticeable near the flowering stage. Infected leaves present white to yellow spots and can be twisted in severe

case. Panicles are unable to emerge completely, they remain green and generally fail to produce grain. Downy mildew is considered as a minor disease of rice (Ou 1985).

Damage

The disease has a wide range of hosts but downy mildew is considered a minor fungal disease of rice (Ou 1985). Although downy mildew has been recorded in south eastern Australia (first at Yanco in 1930), the hot dry climate does not generally favour its development (Cother, personal communication).

Favourable conditions

Downy mildew is favoured by cool temperatures (18-23oC) and a high relative humidity therefore, the hot dry climate of southern NSW does not favour this disease.

Control strategy

None

5.5 Cochliobolus leaf spot (Cochliobolus sativus)

Leaf spot caused by Cochliobolus sativus has been recorded for the first time in south eastern Australian rice fields in 1966 (Walker et al. 1968). Little is known about this disease but it can be suspected that the biology of C. sativus is relatively similar to C. myabeanus (causing brown spot)

Lifecycle

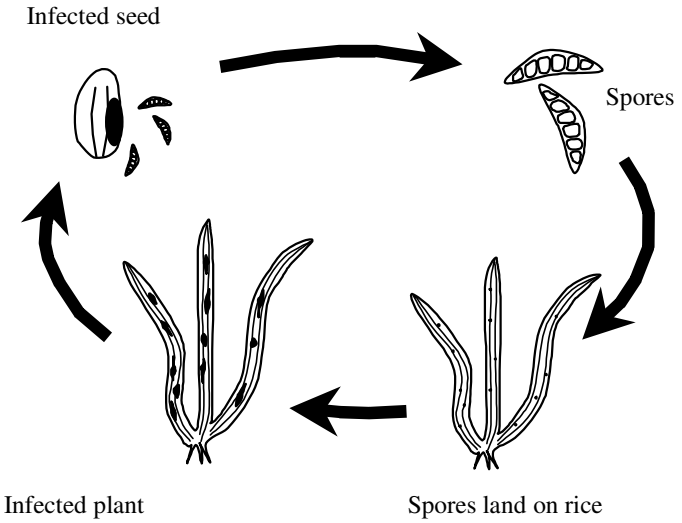

Figure 21: Supposed disease cycle of Cochliobolus sativus

Leaf spot is a seed born disease. The fungus overwinters in rice seeds and also in infected crop residue. Primary infection is thought to be caused by infected seeds. Secondary infection is caused by airborne conidia and results in leaf lesions. In the tropic, inoculum is generally found all year round (Ou 1985).

Symptoms

Leaf spot can attack rice plants at any stage of growth. On young plants symptoms are small brown to purple brown circular spots. On older plants, spots appear as brown oval spots with a grey-white centre. Spots can be found on almost the entire plant and are generally uniformly distributed. The length of the lesion ranges from 1 to 4 mm but can reach 1 cm on susceptible varieties (Ou, 1985).

Damage

Heavily infected seed cause blight of seedlings. Several authors have reported important seedling losses due to blight (Ou, 1985). On older plants, the disease reduces the kernel weight and the number of grain per panicle. In 1943 a brown spot attack caused a famine in Bengal (Padmanabhan 1973).

Favourable conditions

Brown spot attacks are correlated with nutrient soil deficiency. The disease is rarely reported when rice is grown in good soils. Free water, high humidity and hydric stress favour the disease (Ou 1985).

Control strategy

Growing rice in fertile soils and/or providing adequate fertilisation is the best method to control brown spot as it is more a nutritional or physiological disorder than a pathological disease (Ou 1985).

6. DISEASES RECENTLY DISCOVERED IN SOUTH EASTERN AUSTRALIA

A rice disease survey was conducted in summer 2001 in the MIA, CIA and in the Murray Valley. Many plants showing previously unobserved disease symptoms were collected and investigated in the plant pathology laboratory of Charles Sturt University. Two pathogens were isolated from the lesions and successfully infected rice plants in glasshouses. After investigation, Waitea circinata was found to have already been discovered in 1995-96 (Watson, personal communication) but this the very first time Rhizoctonia oryzae-sativae, causal agent of the aggregate sheath spot disease, is discovered in the Australian rice crops.

6.1 Sheath spot (caused by Waitea circinata)

Sheath spot was first reported in Australia in the southern NSW rice growing area during the 1995-96 season and it was recorded again in both summers 2000 and 2001. Waitea circinata (anastomosis group WAG-Z) is the teleomorph of R. zeae whereas W. circinata (anastomosis group WAG-O) is the teleomorph of R. oryzae.

Life cycle

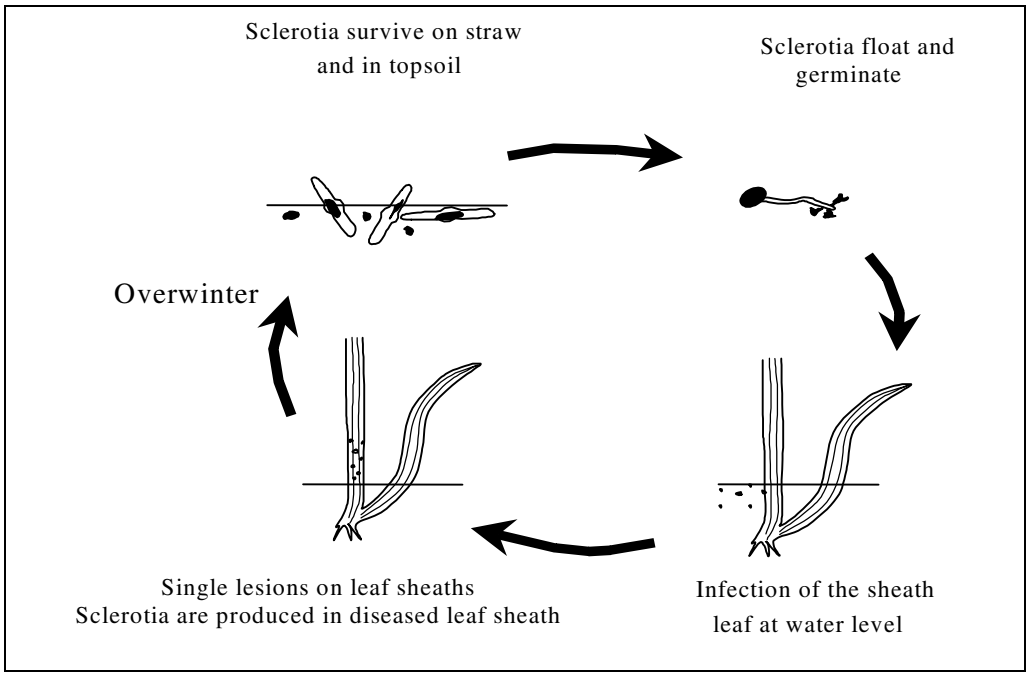

Figure 22: Disease cycle of Waitea circinata

The fungus survives as sclerotia on infected rice straw or in soil. In spring, when the rice paddy is flooded, the sclerotia float on the water surface, germinate and then infect the rice sheaths at the water line. At the end of the season, cylindrical sclerotia are produced in the diseased leaf sheaths (Rush 1992).

Symptoms

The disease causes spot type lesions on the leaf sheath midway up the tiller. Typical lesions are oval, 0.5-3.0 cm long. The spot is generally grey-white surrounded with a reddish-brown border. In contrast to the very similar aggregate sheath spot disease, the lesions are usually separated and do not coalesce (merge). The first symptoms are generally found near the water line and then progress up the leaf sheaths. Favourable conditions for the fungus can cause the death of the infected leaves (Rush 1992).

Damage

Sheath spot disease can cause lodging on very susceptible cultivars but it is generally considered of minor importance (Rush 1992).

Figure 23: Sheath spot symptoms(V. Lanoiselet)

Figure 24: Sheath spot symptoms (V. Lanoiselet)

Favourable conditions

Semi-dwarf rice cultivars could be more susceptible than tall cultivars (Rush 1992).

Control strategy

Straw removal, stubble burning and a longer rotation are the current control methods for aggregate sheath spot (Rush 1992). At the time of publication no rice varieties that are resistant to the disease have been identified or developed in Australia.

6.2 Aggregate sheath spot (caused by Rhizoctonia oryzae-sativae)

Aggregate sheath spot, also sometimes known as brown sclerotial disease, was first reported in Australian rice crop by the author during the rice disease survey in summer 2001.

Lifecycle

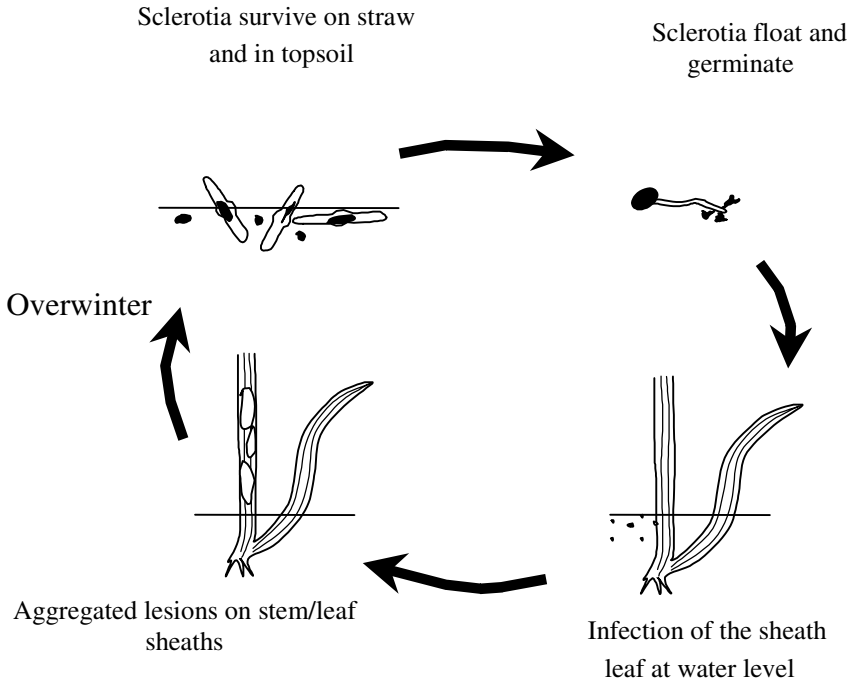

Figure 25: Disease cycle of Rhizoctonia oryzae-sativae

The fungus survives as sclerotia on infected rice straw or in soil. In spring, when the rice paddy is flooded, the sclerotia float on the water surface and infect the rice sheaths at the water line. The first symptoms appear during the tillering stage as small black lesions on leaf sheaths. The disease then grows upward and reaches the upper leaf sheaths. Secondary infections progress up the stem and may spread to the flag leaf and panicle. Leaves of infected sheaths turn yellow and then generally die. The fungus can reach and infect the culm. The fungus then produces sclerotia on the surface and in the different infected tissues of the rice plant (Gunnell & Webster 1984).

Symptoms

Aggregate sheath spot can be easily mistaken as sheath blight (R. solani) and sheath spot (R. oryzae). The first lesions appear on the rice plant near the water line. Lesions are oval and can be grey-green or yellowish coloured surrounded by a brown margin. Their size range from 0.5 to 4 cm in length. The disease grows upward reaching the upper leaf sheaths. Leaves of infected sheaths turn yellow and then generally die.

Figure 26: Aggregate sheath spot symptoms (V. Lanoiselet)

Figure 27: Aggregate sheath spot symptoms (V. Lanoiselet)

The culm may be attacked and infected but this symptom rarely occurs in California (Gunnell & Webster 1984). Young sclerotia appears whitish as they are covered with mycelium, then take a dark-brown colour overtime. Their size range can be greatly variable.

Damage

The death of infected sheath leaves by reducing the photosynthesis process can lead to grain yield losses. Culm or stem attack generally lead to grain sterility or can reduce the filling and the quality of the grain (Gunnell 1992).

Favourable conditions

Gunnell and Webster (1984) suggest that Californian semi-dwarf rice cultivars are more susceptible than tall cultivar. On the contrary to sheath blight (R. solani), high nitrogen application does not seem to favour aggregate sheath spot (Gunnell 1992).

Control strategy

The disease can be managed by reducing the amount of inoculum present. Straw removal, stubble burning and longer rotation are the current control methods. No resistant variety has been discovered so far (Gunnell 1992).

7. MAJOR EXOTIC RICE DISEASES UNLIKELY TO OCCUR IN SOUTH EASTERN AUSTRALIA

7.1 Virus diseases

There are more than 15 main rice diseases caused by virus. For the vast majority of these diseases, the virus requires tropical insect vectors. Virus diseases are therefore mostly a problem in tropical or sub-tropical regions (Ou 1985). Some of the vectors can be found in Northern Australia but would be unable to survive the climate of south eastern Australia (Stevens, personal communication).

7.2 Bacterial diseases

More than 10 diseases are known to be caused by bacteria. Most of these diseases are caused by only two genera of bacteria. Pseudomonas species causes diseases of the seedlings, sheath and grain whereas foliar diseases are caused by Xanthomonas species. Bacterial diseases are generally favoured by warm temperature, rain, humidity and storms. As a consequence, most of the bacterial diseases occur under tropical or sub-tropical climates (Ou 1985).
The most important worldwide bacterial disease is bacterial blight caused by Xanthomonas oryzae pv.oryzae (Ou 1985).

7.2.1 Bacterial blight (caused by Xanthomonas oryzae pv.oryzae)

Bacterial blight caused by X. oryzae pv. oryzae is the most important worldwide bacterial disease of rice. It can occur in both tropical and temperate region. This disease has now been reported in Asia (Ou 1985), Northern Australia (Aldrick et al. 1973), and in many other rice-growing countries (Ou 1985).

Lifecycle

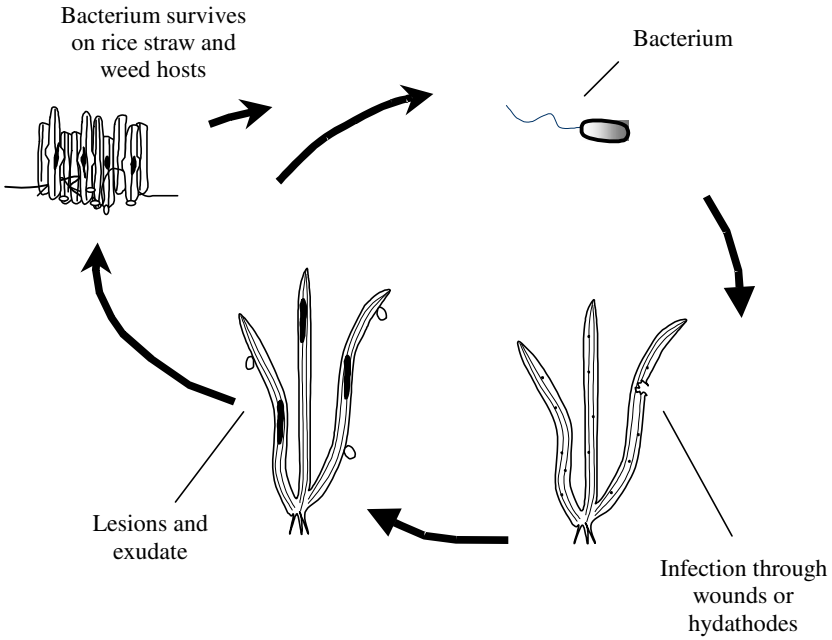

Figure 28: Disease cycle of Xanthomonas oryzae pv.oryzae

The bacterium survives mainly in rice straw and stubble and on weed hosts. It can also survive for a few weeks on infected rice seeds and in soil but it is generally not considered to be an important source of infection (Mizukami 1961). Unpublised studies from IRRI suggest that X. oryzae can be present all year round in the irrigated channel and in rice fields of tropical countries.
The bacterium infects the rice plant through the water hole of the hydathodes. The hydathodes are epidermal structures specialised for secretion of water. On rice plants, they are located along the upper surface of the leaf near the edges. Once in the hydathode, X. oryzae multiples in the epitheme before invading the vascular system of the rice plant. A large number of bacteria is then excreted by the water exudation system of the plant (Tabei & Muko 1960). The pathogen can also can also enter through leaf or root wounds. The pathogen can be spread by water, rain splashes, wind, machinery and tools or even by handling during rice seedling transplantation (Ou 1985).

Symptoms

X. oryzae can infect leaves and stems but symptoms are only noticeable on the leaves of rice plants. Three different symptoms of rice blast can be observed on rice (Ou 1985).

Kresek or wilt syndrome

This is the most serious form of the disease as it leads to the death of the plants. The Kresek symptom is generally found in the tropical rice growing areas where rice plants are transplanted from nursery to rice fields. The transplantation process creates numerous wounds on the leaves and roots, which lead to the infection and to the disease. The first symptoms are green water-soaked spots on the leaves. The infected leaves turn then greyish-

green and begins to fold up and roll along the midrib. The kresek symptom can sometimes be mistaken as a rice stem borer attack (Ou 1985).

Leaf blight

Leaf blight is the most common syndrome of the disease. In temperate regions it generally appears at the heading stage. The first symptoms observed are green water-soaked stripes on the leaves.

Figure 29: Leaf blight symptoms (D.E. Groth)

The size of the stripe increase and they then turns yellow then white. The symptoms may cover the entire leaf. On young lesions, drops of bacterial exudate may be observed generally early in the morning. In case of severe infection, lesions surrounded by a water-soaked margin can be found on the glumes (Ou 1985).

Yellow leaf

This is another syndrome found in the tropics. The youngest leaves are the only one to show the symptoms. The whole leaf becomes pale yellow or has a yellow or greenish-yellow board stripe. Whereas the leaves show the symptoms, the bacteria can only be found in the stem of the rice plant (Ou 1985).

Damage

In temperate regions, the disease mainly reduces the weight and the quality of the grain. The severity of the disease is generally greater in the tropics as the Kresek attack kills the plants. In Japan, yield losses are usually between 20 to 30% (Ou 1985).

Favourable conditions

In south east Asia, typhoon and storms are known to favour the occurrence of the disease by disseminating the bacteria and by causing wounds to the rice plants. This explains why bacterial blight is often considered as a post-typhoon disease or rain borne disease. The severity of the disease is correlated with heavy rainfalls, violent winds, flooding and high nitrogen applications. On the contrary, severe drought and heat reduce or suppress the severity of the disease (Ou 1985).

Control strategy

In infected areas, the use of resistant cultivars is recommended. Integrated management program also include, controlling weed hosts, avoiding high nitrogen applications, controlling flooding and plowing rice field after harvest (Mew 1992). Burning rice straw and longer crop rotation should be researched as a possible way of controlling bacterial blight.

Probability of entry

Bacterial blight is generally considered the most important bacterial disease worldwide. It is found in both tropical and temperate rice growing areas. The disease is currently occurring in many rice growing areas (Ou 1985). In 1973 bacterial leaf blight was reported for the first time in Australia on cultivated and on wild rice in the Northern Territory (Aldrick et al 1972). All Australian isolates were found to have low virulence compare to Asian isolates. Infected rice plant or seed transportation poses the greatest threat of introducing X. oryzae. It is illegal to carry rice material in the Australian rice-growing region so the probability of entry is low.

Probability of establishing

The climate prevailing in northern Australia is favourable to X. oryzae but the hot and dry conditions occurring in southern NSW/northern Victoria are unfavourable for bacterial blight. A bacterial blight outbreak in the current rice growing regions of Australia appears therefore unlikely.

Total risk

Bacterial blight is unlikely to occur in the current Australian rice growing regions (southern NSW-northern Victoria).

7.3 Fungal diseases

The majority of the rice diseases are caused by fungi and many of them are considered minor diseases of rice. The most important fungal diseases are rice blast (caused by M. grisea), sheath blight (caused by R. solani), stem rot (caused by S. oryzae) and seedling diseases (Pythium, Achlya spp.) (Ou 1985).

7.3.1 Sheath blight (caused by Rhizoctonia solani)

Sheath blight was first reported in Japan by Miyabe in 1910 The disease now occurs in most temperate and tropical rice growing areas of the world. Over the last 20 years sheath blight has become an important problem in intensive rice-growing area (Ou 1985).

Lifecycle

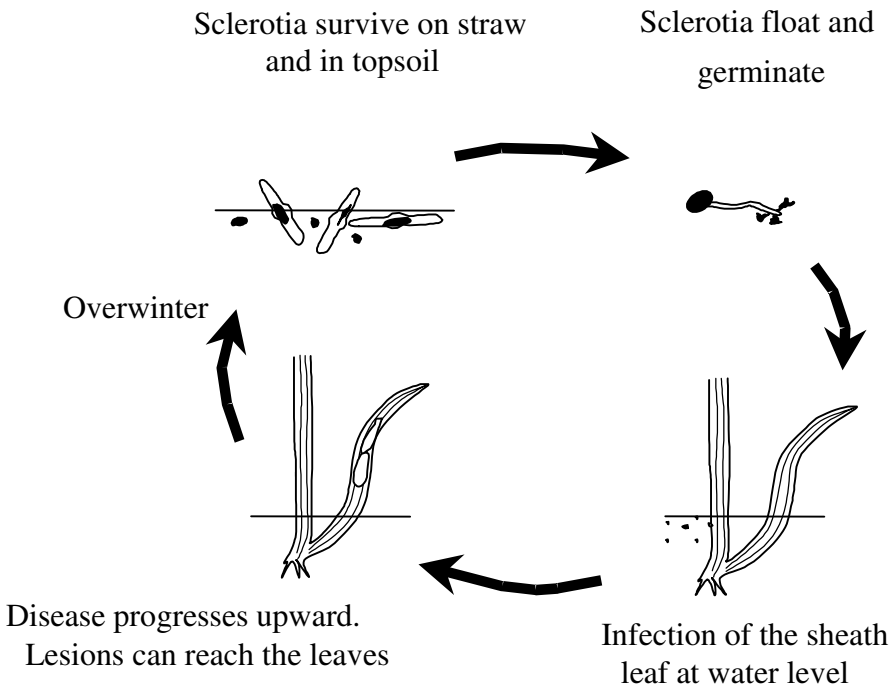

Figure 30: Disease cycle of Rhizoctonia solani

The fungus overwinters as sclerotia or as mycelium in crop residue and can survive several years in rice straw or in the soil. In spring, when the rice paddy is flooded, the sclerotia float on the water surface and infect the rice plant at water line level (Ou 1985).

Symptoms

Figure 31: Sheath blight symptoms on leaves (D. E. Groth)

Figure 32: Sclerotia on rice leaf (D. E. Groth)

One centimetre long, ellipsoid, green-grey spots on the leaf sheath are generally first observed near the waterline. Under favourable conditions the disease progress upward, reaching leaves and panicles (Ou 1985).

Damage

Sheath blight is generally considered the second most important fungal rice disease. Yield losses can reach 50% on susceptible cultivar (Ou 1985).

Favourable conditions

Humid and warm climate favours sheath blight. Semi-dwarf cultivars and high nitrogen fertilisation also favours the disease (Rush & Lee 1992).

Control strategy

US medium grain cultivars were found to be more tolerant to sheath blight than long grain cultivars. Fungicide applications are currently recommended in southern USA and growers. Growing host crop such as soybean and sorghum in rotation with rice increases inoculum level in soil (Rush & Lee 1992).

Probability of entry

The causal agent of sheath blight disease has already been recorded in the Northern Territory and also in Queensland. Infected rice plant, straw or soil transportation causes the greatest threat of introducing R. solani in south eastern Australia. It is illegal to carry rice material in the Australian rice-growing region so the probability of entry is low but not impossible.

Probability of establishing

The disease is favoured by humid and warm climatic conditions. Therefore the climate of south eastern Australia does not seem favourable for the disease.

Total risk

The total risk appears low and even if the disease was introduced and became established in south eastern rice crop, the climatic conditions would probably prevent the disease reaching severe economic levels. The total risk sheath blight represents to the rice industry is low.

7.4 Nematodes

Nematodes are microscopic worms generally living in soil. Among the 150 species of plant-parasitic nematodes found in rice soils, the only nematodes being considered of major importance are: Aphelenchoides besseyi, Dytilenchus angustus, Hirschmanniella spp., Meloidogyne spp., Heterodera spp. (Ou 1985). The climatic conditions prevailing in the Australian rice growing area are unsuitable for all of these nematodes except the root nematode (Hirschmanniella spp.).

8. RECOMMENDED MEASURES IN THE EVENT OF AN OUTBREAK OF THE FOLLOWING DISEASE OUTBREAK IN SOUTH EASTERN AUSTRALIA

8.1 Rice blast (caused by M. grisea)

In the event of rice blast being introduced and causing an outbreak in Australian rice, it may be useful for Australia to examine how California is handling M. grisea.

The Californian example

In September 1996, a rice blast outbreak occurred in California. This was the first time blast was recorded in California. A University of California survey of 508 fields found 33 infected fields, with 27 in Glenn County and 6 in Colusa County. The first observations indicated that Californian rice varieties were susceptible to the disease. By 1998, rice blast had already affected 867 fields or 58,000 acres in 3 different counties. The spread of this disease meant the local authorities relaxed their pollution regulations and allowed rice growers to burn the stubble of infected field. The Californian State recommended the following measures to try to stop the spread of rice blast:

- destruction of rice stubble and straw located in adjacent fields exposed to the infestation,
- cleaning of rice harvesters and other harvesting equipment,
- planting only certified seeds,
- use of seed treatments/dressing,
- avoid excessive nitrogen fertilisation,
- continued monitoring of rice fields.

A new fungicide from Zeneca called Quadris(r) (Azoxystrobin) was registered in California in 1997. This fungicide is a preventive treatment and should therefore be applied before symptoms such as collar and panicle infection occur. The Californian rice blast task force recommended all infected areas to be targeted for treatment. A single application of Quadris was estimated to cost about US $20 an acre.

Blast also occurs in several other American States (Arkansas, Mississippi and Texas). Most rice varieties grown in Arkansas are resistant to M. grisea but some susceptible varieties have higher yield and grain characteristics. Dr Lee Fleet from the University of Arkansas

recommends that farmers grow susceptible cultivars to choose a field that has no history of severe blast and plenty of available water. As moisture stress makes rice varieties more susceptible to blast, it is important to maintain good flood level in the fields.

8.2 Kernel smut (caused by Tilletia barclayana)

Control of kernel smut is not easy. The use of tolerant cultivars is the only known method to control kernel smut. Short-grain cultivars appear to be the most susceptible whereas short grain cultivars are the most resistant. Heavy applications of nitrogen and high flood depth during flowering are known to favour the disease.

Should a kernel smut outbreak ever reported, the following measures might help to contain/eradicate the disease:

- burning stubble and straw might reduce the amount of inoculum
- cleaning of rice harvesters and other handling equipment could reduce the dissemination of the inoculum
- high nitrogen fertilisation should be avoided
- high water level during flowering should also be avoided

8.3 Root nematodes (Hirschmanniella spp)

The introduction of nematodes can remain undetected for a long time as nematode damage is quite often mistaken as nutrition deficiency or diseases. The use of tolerant rice varieties and applying nematicides are most successful methods available to control Hirschmanniella spp. The susceptibility of the Australian rice varieties to these nematodes is unknown.

In the event of a localised establishment of root nematodes the following measures might help to contain/eradicate the nematodes:

- avoid growing rice in infested the paddock(s)
- growing trap plants during interculture
- applying mematicides on the infested paddock(s)

9. CONCLUSION - FUTURE PROSPECTS

The vast majority of the viral, fungal and nematode pathogens to rice plants were found to be unlikely to occur in south eastern Australia. However, the risk assessment has identified two fungal diseases (rice blast and kernel smut) and one plant parasitic nematodes genus (root nematodes) as having the potential to threaten the Australian rice industry if ever introduced in south eastern Australia. Among these three pathogens, rice blast represents the greatest threat to the rice industry in Australia. Rice blast caused by the fungus Magnaporthe grisea is generally considered the most important disease of rice worldwide because of its destructiveness under favourable conditions. Rice blast has never been observed in the Australian rice-growing area but M. grisea has been reported on weeds in several Australian States including NSW.
In the event of a rice blast outbreak, it would be usefull to follow the same measures undertaken by California following their rice blast outbreak in 1996. The two most important measures to undertake would be the destruction of rice stubble and straw located in adjacent fields exposed to the infestation and lobbying for the registration of a fungicide effective against rice blast such as Quadris(r) (Azoxystrobin).

The potential threat of exotic diseases being introduced and becoming established in rice growing area of Australia has been identified. It is, therefore, important that rice-growers are aware of the vulnerable nature of the Australian rice industry to new diseases and more pathogenic strains of existing fungal pathogens. Trash in second-hand farm equipment from overseas rice producing countries and travellers coming from other rice producing areas pose the greatest threats of introducing more virulent strains of pathogens into Australia. **The appearance of stem rot disease in 1994-95, sheath spot disease in 1995-96 and aggregate sheath spot disease in 2000-01 serves as a warning to the industry that its relative freedom from disease is tenuous and vigilance to avoid introducing new or more virulent pathogens must be maintained.**

ACKNOWLEDGMENTS

The authors would like to thank Mr G. Maywald (CSIRO Brisbane) for his knowledge of DYMEX, Professor R. Webster and Mr R. Mutter (University of California) for helping to validate the model with Californian data. Special thanks to Dr. L. Humphreys and Mr D. Smith (CSIRO Griffith), and Dr. R. Williams and Mr. A. Watson (NSW Agriculture, Yanco) for their various help. The assistance of Belinda Gersbach in preparation of the maps and financial support to Vincent Lanoiselet from the Cooperative Research Centre for Sustainable Rice Production are gratefully acknowledged.

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APPENDIX 1 THE RICE BLAST MODEL STRUCTURE

Environment - Common
Variables:

User-defined Function Templates
None specified

Module: Timer
Inputs: Not used [Equilibrium variable]
Outputs: Days since start
Day of year
Simulation date

Timestep: Daily

Module: MetBase1 [MetBase]
Inputs: Simulation date
Outputs: Minimum temperature
Maximum temperature
Rainfall
Max relative humidity
Min relative humidity

Module: Latitude [QueryUser]
Inputs: None
Outputs: Latitude variable1

Module: Daylength1[Daylength]
Inputs: Latitude variable 1
Day of year
Outputs: Daylength
Not used [Day length change]

Module: Daily temperature cycle [Circadian]
Inputs: Min temperature [Daily minimum value]
Max temperature [Daily maximum value]
Day length [Daylength]
Outputs: Daily temperature cycle [Daily cycle]
Cycle shape: Composite (Sine + Exponential)

Module: Daily RH cycle [Circadian]
Inputs: Min relative humidity [Daily minimum value]
Max relative humidity [Daily maximum value]
Day length [Daylength]
Outputs: Daily RH cycle [Daily cycle]
Cycle shape: Sin

Module: Lifecycle1 [Lifecycle]

Inputs: Day of year [Day of year]
Not used [Simulation date]

Cohort variables:
Chronological age [Local]
Density [Local]
Number [Local, Proportional (inverted)]
Physiological age [Local, Direct]

Lifestage: Spores
Environment: Global
Resource: Not specified
Outputs: Total number: total number of spores

Process: Continuous mortality (affect number)
r1: function
Driving variable: Chronological age
Function shape: step [F(x) = max(0, f(x))]
Parameter: threshold
Min: 2 Max: 2 Default: 2
Parameter: step height
Min: 1 Max: 1 Default: 1

Process: Spores - Transfer (affect stage transition to disease)
Combination rule: product
r1: function: temperature infection slope
Driving variable: Daily temperature cycle
Function shape: 3-segment linear
Hashioka (1965) found that a minimum of 10 hours at 32oC, 8 hours at 28oC or 6 hours at 24oC was required for conidial germination and the penetration of germ tubes into the host cells. Kahn and Libby (1958) reported that the minimum periods required to initiate infection were 10, 12 and 14 hours at 26.7oC, 21.1oC and 18.3oC respectively.

Parameter: line 1 x-intercept
Min: 10 Max: 10 Default: 10
Parameter: line 1 slope
Min: 0.285 Max: 0.285 Default: 0.285
Parameter: line x-value at intersection of line 1,2
Min: 24 Max: 24 Default: 24
Parameter: line 1 slope
Min: -0.196 Max: -0.196 Default: -0.196
Parameter: x-value at intersection of line 2,3
Min: 32 Max: 32 Default: 32
Parameter: line 3 slope
Min: -2.43 Max: -2.43 Default: -2.43

r2: function: humidity requirement for infection
Driving variable: Daily RH cycle
Function shape: Step [F(x) = max(0, f(x))]
High relative humidity, necessary for infection (Hemmi and Imura 1939), and free water, required for conidial germination (Ou 1985), were simulated by a step function with a threshold of 100% humidity.

Parameter: threshold
Min: 100 Max: 100 Default: 100
Parameter: slope
Min: 1 Max: 1 Default: 1

Reproductive lifestage: Disease
Environment: Global
Resource: Not specified
Outputs: Total number: total number of infections

Process: Continuous Mortality (affects number)
r1: function
Driving variable: Chronological age
Function shape: step [F(x) = max(0,f(x)]
Parameter: threshold
Min: 40 Max: 40 Default: 40
Parameter: step high
Min: 1 Max: 1 Default: 1

Process: Development (affects physiological age)
r1: function: temperature function
Driving variable: Daily temperature cycle
Function shape: 3-segment linear
The temperature favourable for infection are similar to those for mycelial growth (Ou 1985).

Parameter: line 1 x-intercept
Min: 10 Max: 10 Default: 10
Parameter: line 1 slope
Min: 0.285 Max: 0.285 Default: 0.285
Parameter: line x-value at intersection of line 1,2
Min: 24 Max: 24 Default: 24
Parameter: line 1 slope
Min: -0.196 Max: -0.196 Default: -0.196
Parameter: x-value at intersection of line 2,3
Min: 32 Max: 32 Default: 32
Parameter: line 3 slope
Min: -2.43 Max: -2.43 Default: -2.43

Reproductive process: Disease - fecundity
r1: Parameter: minimum amount of spores
Min: 84000 Max: 84000 Default: 84000
Fecundity is the total number of possible spores that can be produced per lesions. Kato and Kozaka (1974) found that spore production was correlated with temperature and that the minimum quantity of spores produced by a single lesion was around 84,000.

Process: Disease - Progeny production (affects progeny production to spores)
Combination rule: product
r1: function: rate of spore production
Driving variable: Daily temperature cycle
Function shape: 3-segment linear
Progeny production determines the rate at which the progeny are produced over time. The rate of spore production varies with the temperature (Kato & Kozaka, 1974).

Parameter: line 1 x-intercept
Min: 16 Max: 16 Default: 16
Parameter: line 1 slope
Min: 75.25 Max: 75.25 Default: 75.25
Parameter: line x-value at intersection of line 1,2
Min: 20 Max: 20 Default: 20
Parameter: line 1 slope
Min: -25.6 Max: -25.6 Default: -25.6
Parameter: x-value at intersection of line 2,3
Min: 25 Max: 25 Default: 25
Parameter: line 3 slope
Min: -20 Max: -20 Default: -20

r2: function: humidity requirement for sporulation
Driving variable: Daily RH cycle
Function shape: Step [F(x) = max(0,f(x))]
Mature lesions are capable of sporulating when relative humidity is higher than 89% (Hemmi & Imura, 1939).
Parameter: threshold
Min: 89 Max: 89 Default: 89
Parameter: slope
Min: 1 Max: 1 Default: 1

r3: function: latent period for sporulation
Driving variable: Chronological age
Function shape: Pulse
Kato and Kozaka (1974) demonstrated that lesions produce spores for about 30 days after a latent period of about 6 days.
Parameter: threshold
Min: 6 Max: 6 Default: 6
Parameter: slope
Min: 1 Max: 1 Default: 1
Parameter: pulse width
Min: 30 Max: 30 Default: 30

APPENDIX 2 FUNCTION TEMPLATE DETAILS USE IN THE DYMEX MODEL

Step function
A Step Function evaluates the relationship

f(x) = 0, for x < p1
f(x) = p2,for x >= p1

The function has two parameters, p1 and p2.

p1 - the Threshold - the value of the Driving Variable at which the step occurs.

p2 - the Step Height.

In DYMEX Function Template Syntax

y = if([x1]<[p1], 0) + if(not([x1]<[p1]), [p2])

Three linear segment function

View Three Segmented Linear Function Template
A Three Segmented Linear Function evaluates the relationship of 3 straight-line segments, as follows

$f(x) = (x - k1)*k2, \quad \text{for } x < k3$
$f(x) = (k3 - k1)*k2 + (x - k3)*k4, \quad \text{for } k3 <= x < k5$
$f(x) = (k3-k1)*k2 + (k5 - k3)*k4 + (x - k5)*k6, \text{ elsewhere}$

The function has 6 parameters:

k1 - the intercept with the X-axis of Line 1
k2 - the slope of Line 1
k3 - the value of x at which line 1 and line 2 meet
k4 - the slope of Line 2
k5 - the value of x at which line 2 and line 3 meet
k6 - the slope of Line 3

Pulse function

View Pulse Function
A pulse function evaluates the relationship described by the following equations:

$f(x) = 0 \quad \text{for } x < p1$
$f(x) = p2 \text{ for } p1 =< x < (p1+p3)$
$f(x) = 0 \quad \text{for } x >= (p1+p3)$

The 3 function parameters have the following meanings:
p1 - Threshold value
p2 - Pulse Height
p3 - Pulse width

In DYMEX Function Template Syntax

y = if(not([x1]<[p1]) & ([x1]<([p1]+[p3])),[p2])