CHAPTER 10

PEST AND DISEASE MANAGEMENT

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Wild plant species were adapted prior to the establishment of all major human cultures. Wheat, rye and barley originated in the Mediterranean and proximal areas; rice in India; bananas in south-east Asia; corn in south and central America and potato in South America. Organisms associated with these plants can be placed on the continuum from mutualism through to parasitism/predation, depending on their interaction with the plant. As these plants were domesticated and grown, increasingly in monoculture, the potential damage caused by their co-evolved pathogens/predators was realised. These pathogens were distributed with the crops as they were adopted in different parts of the world. Additionally, the crops were exposed to a range of other potential pathogens/predators in these new locations.

A pest can be defined as anything that people consider a threat to themselves, their crops, animals or property. A definition of pests must include nematodes, insects, weeds, molluscs, bacteria, fungi, phytoplasmas, viruses and viroids. Weeds are excluded from this consideration as they are discussed in Chapter 9.

HISTORICAL PERSPECTIVES

The ravages of pests on crops have been recorded since the earliest times in civilisation. The Romans created a god, Robigo, to whom they made sacrifices in the hope that he would protect their crops from stem or red rust (*Puccinia graminis* f.sp. *tritici*) (Agrios, 1997). In addition to appeasement of the gods, more conventional approaches to crop protection have been employed for over 4000 years.

The earliest farmers, by selecting seed from the healthiest plants and by retaining seed to sow from one year to the next, were already practising plant selection for resistance to a number of diseases. In 300BC, the Greek philosopher, Theophrastus, noted that diseases of crops occurred more regularly in the lowlands and that different diseases affected different crops (Agrios, 1997). However, due to the microscopic nature of most causes of plant diseases, they were largely associated with the wrath of God prior to the invention of the compound microscope in the mid-1600s.

The earliest examples of the use of insecticides can be traced to the Sumerians who used sulphur compounds to control insects and mites from 2500BC onwards. The Chinese used botanical insecticides as early as 200BC and are also credited with the use of mercury and arsenical compounds to control body lice (Dent, 2000). The development of chemicals to control plant diseases lagged by some 2800 years when in the mid-1600s farmers in the south of England noticed that wheat grown from seed salvaged from a shipwreck had a reduced level of bunt when compared to other crops. This led to the recommendation that wheat seed be soaked in brine prior to planting. The most important breakthrough in chemical control of plant diseases was made by Millardet in France in 1882. He observed that grapevines sprayed with a mixture of copper sulfate and lime (used to deter school children from stealing grapes) also reduced levels of downy mildew (Schumann, 1991). This mixture was refined by Millardet and became known as 'Bordeaux Mixture'. Bordeaux Mixture was also combined with a dye, 'Paris Green', to give protection against Grape Phylloxera.

An increase in reliance upon pesticides began in the 1890s with the use of lead arsenate and in the early 1900s with the addition of pesticides based on natural products such as pyrethrum and nicotine. However, the widespread adoption of pesticides in agriculture began with the discovery of DDT

(dichlorodiphenyltrichloroethane) by Paul Müller in 1939 while working for Geigy Chemical Company. The low cost, persistence, low mammalian and plant toxicity and broad-spectrum activity ensured its widespread adoption and use. The discovery of other compounds such as aldrin, heptachlor and chlordane was spurred by the success of DDT. These chemicals were so successful that research into alternative methods of pest control was often downgraded or abandoned. This had a marked effect on areas of research such as biological control. The publication of the book *Silent Spring* by Rachel Carson in 1962, and the subsequent reports of widespread pesticide resistance, have served to turn attention to the use of biological controls in recent times. Unfortunately, however, many people still expect biologicals to act in a fashion comparable to synthetic pesticides. Carson's book led to the withdrawal of DDT and a global ban on the use of the chemical. However, more recently it has been strongly argued that DDT should still be used against vectors of malaria in prone regions (Kapp, 2000; Attaran *et al.,* 2000).

Biological control also has a long history as a component of pest management in crops. Predatory ant colonies were used in China and Yemen to control caterpillar and beetle pests (Dent, 2000). These farmers constructed bamboo bridges between trees to facilitate the movement of ants. A pathogen of insects (*Metarhizium anisopliae*) was used as control of the sugar beet curculio in 1884 by the Russian entomologist, Elie Metchnikoff. The use of fungal spores to control a rot of pine was first reported in 1963.

IDENTIFICATION OF THE CAUSE

In addition to biological crop pests, the crop protection practitioner is often faced with symptoms on plants caused by non-biological (abiotic) causes including temperature extremes, nutrient deficiencies and sub-lethal effects of herbicides. These latter non-biological or non-infectious conditions, although strictly not plant diseases, must be eliminated as potential causes of plant disease early in any quest to identify a pest problem. Therefore, the crucial first step in any crop protection strategy is to identify the current pest(s) of concern or the potential pests. This identification will lay the foundation for any future decisions to be made.

Most plant diseases are caused by microorganisms (fungi, bacteria, phytoplasmas, viruses, viroids) and are therefore, usually, too small to be identified with the naked eye. Some of the fruiting bodies of fungi may be large enough to be used as a means of identification, (eg. sclerotes of species of *Sclerotinia* or stem rust pustules), however, many infections are largely internal and by the time some of the fruiting bodies are large enough to be seen with the naked eye, it is too late to implement a management program. Plant diseases cause alterations to the plant that can be seen as symptoms or signs. Signs of plant disease are defined as the pathogen, its parts or products seen on or in a host plant. If the pathogen can be seen then this helps in correct disease identification.

The most common disease signs include rusts, smuts, downy mildews and powdery mildews. The rust sign is the spores of the fungus erupting through the epidermis of the plant, whereas a powdery mildew is the fungus mycelium and spores on the plant surface. Downy mildews, on the other hand, appear as a white or grey bloom of fungal spores that protrude through the stomates of the host. Smut fungi replace parts of the plant (usually the floral organ) with spores. All of the fungi responsible for these signs are biotrophic (*i.e*. require a living host). Symptoms are the changes to the plant in response to infection. Some symptoms are characteristic in certain crops such as reddening of the xylem vessels in fusarium wilts or mosaic patterns on leaves due to viral diseases. Others are an indication only and require the isolation of the pathogen for further identification.

- Bacterial identification is through standard bacteriological tests as well as fatty acid analysis or sequencing of the repetitive gene sequences such as the 16S rDNA region. This is the region of DNA that occurs between genes coding for the ribosomal apparatus.

- Viruses are usually identified using commercially available ELISA (enzyme-linked immunosorbent assay) techniques, using electron microscopy and/or polymerase chain reaction and sequencing.
- Nematodes may be extracted using a whitehead tray, misting or a Bauermann funnel. Only pathogenic nematodes possess a stylet. Identification is based on morphological characters or sequencing of the 18S rDNA region (this is similar to the region encoding the ribosomal region but in eucaryotes).
- Insects can be identified using standard morphological characters or DNA barcoding. However, the most important aspect is the mouthparts, either sucking or chewing, as this determines the type of injury the insect causes.
- Following the correct identification of the pest, the extent of population must be determined.

Loop-Mediated Isothermal Amplification (LAMP)
LAMP is a relatively new molecular method which is becoming more widely available. It is based on the designing of six DNA primers which are based on the region or gene of interest. It has a number of advantages over traditional PCR in that the reaction is isothermal (it doesn't require thermal cycling), it is very robust due to the enzyme involved (and so is not as sensitive to contamination), it is highly specific and more sensitive that traditional PCR. A number of systems have been designed for use in the field. The output from the reaction is similar to the output from a quantitative PCR (Figure 10.1).

MONITORING POPULATIONS

Monitoring of pest populations is undertaken to determine the geographic distribution and importance of a pest, the effectiveness of control measures or the development and implementation of a forecasting system. This last objective provides information that may be used in either strategic or tactical pest management. For example, these forecasts may provide farmers with information on the likely impact of soil-borne diseases on succeeding crops or the use of crop insecticides in cotton production. The accuracy of these predictions is heavily dependent on the sampling technique employed.

The pattern of distribution of a pest within a crop is rarely regular or uniform. This is especially true in soil-borne pests (Campbell and Noe, 1985; Miller *et al.,* 1997). This pattern may influence the sampling strategy used and the accuracy of the data collected. Furthermore, the pattern may indicate the source of inoculum in the case of plant diseases. The aggregated distribution of pests may, for example, indicate a slow moving pathogen following an initial introduction in seed or on machinery. The density of the pathogen may also affect the distribution. For example, *Anguina agrostis* (a nematode which causes seed

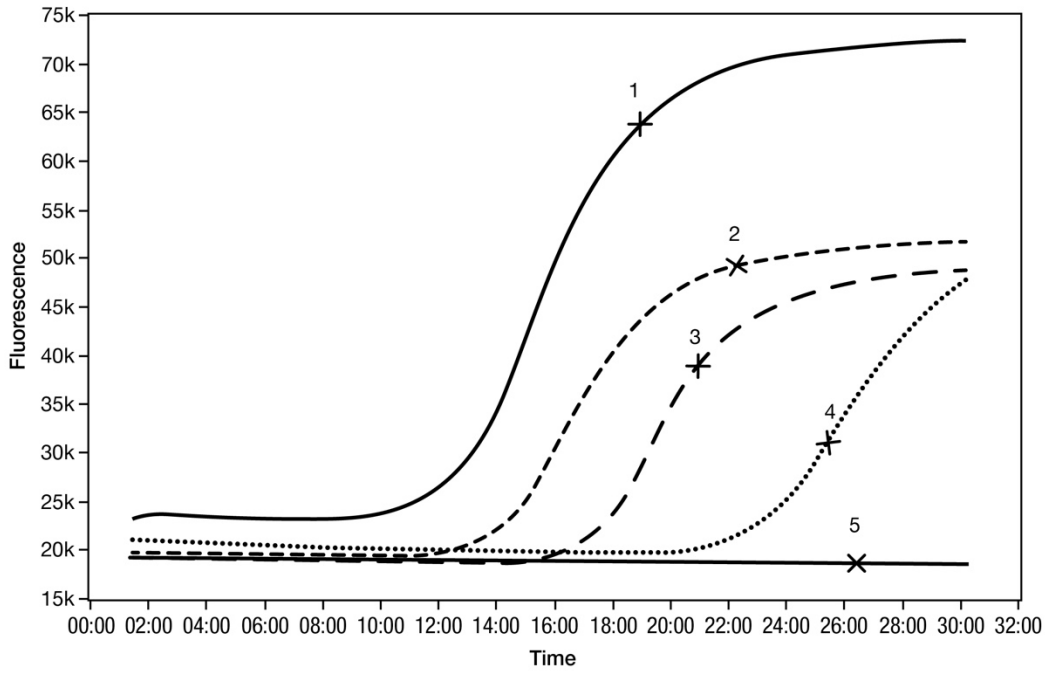


Figure 10.1 Example of loop-mediated isothermal amplification (LAMP) detection of phytoplasma showing positives in the heads of field-collected insects. (1) = positive control; (2) = *Zophiuma pupillata*; (3) = *Lophops saccharicida* (nymph); (4) = *Colgar* sp.; and (5) = negative control (Gurr *et al*., 2016)

gall in colonial bentgrass), the distribution of galls was found to be aggregated at high inoculum densities (Pinkerton and Alderman, 1994). Gradients of diseases or insects, on the other hand, may indicate the distance from the source of inoculum.

When assessing a disease or insect in the field, the distribution becomes very important. If the disease or insect is highly aggregated into 'hot spots', the assessor may easily miss the disease. However, to assess every plant within a field is obviously impractical. Therefore, there are ranges of different sampling patterns that may improve accuracy of disease assessment. Partial field assessment is very quick, but a non-random disease could be easily missed. Whole field sampling gives a better estimation of the disease present and is a compromise between partial field and stratified random sampling. Systematic sampling requires the taking of samples at fixed intervals that are determined using random number tables. In stratified random sampling, the field is divided into smaller sub-units and a random sample(s) is taken from each unit. This last sampling method delivers the highest accuracy but also the highest cost. Stratified random sampling only differs from systematic sampling in that the position within each stratum varies in stratified random sampling.

In addition to the position of the sample within a field, the number of samples to be used will have an effect on the accuracy of measurements as well as the cost in terms of time and labour. The number of samples required is dependent upon sampling variability. The higher the variability, the more samples that will need to be taken to reduce the error to below the generally accepted threshold of 5%. This requires prior knowledge of the variability of the pathogen/pest or a pre-test sample. In this case, with random sampling, the following formula can be used to determine the sample size (n)

$$n = S^2/cy$$

where S = standard deviation of the sample, y = the mean of the sample estimate and c = the pre-determined level of error (*e.g*. 0.05).

The spatial distribution or pattern of a pest can be studied using the variance-to-mean ratio (Piloeu, 1977) which is also known as the Taylor Power Law (Taylor, 1961; Southwood, 1966). When the variance exceeds the mean the pattern is said to be aggregated, whereas if the ratio of variance-to-mean is approximately one, the pattern is considered to be random (Poisson distribution). Variance-to-mean ratios of less than one indicate a uniform pattern.

If the spatial distribution of a pest or pathogen is known then sequential sampling systems can be designed (Madden and Hughes, 1999). Sequential sampling involves collecting samples and adding the number of individuals to the tally until either an estimate of the mean can be made or the number of pests or diseased units can be said to be above or below a predetermined threshold. Although time consuming to develop, sequential sampling systems are useful tools in determining the incidence of pests quickly and easily (Turechek *et al.*, 2001).

In addition to in-crop pest assessment, assessment of initial inoculum of insects and diseases may be used in some systems to forecast the potential for damage in succeeding crops. Propagules of *Sclerotinia* species may be counted following sieving or separation from soil or seed. Nematodes may be separated from soil and the numbers determined or a bio-assay used to determine the numbers. This bioassay technique may also be used for some soil borne diseases such as take-all of wheat (see Case Study 1: Herdina and Roget, 2000). The accurate determination of the levels of some soil borne diseases and nematodes is now being made using polymerase chain reaction (PCR) techniques, a DNA probe or both (Bateman *et al.,* 1997; Goodwin *et al.,* 1995; Klassen *et al.,* 1996; O'Dell *et al*., 1992; Ophel-Keller *et al.,* 1995). For these techniques the total DNA can be extracted from the soil or soil organic matter and levels of pathogens determined using specific primers and quantitative PCR or a slot blot technique.

Case Study 1: Prediction of take-all disease risk in field soil using a rapid and quantitative DNA soil assay (Herdina and Roget, 2000)

Take-all of cereals is caused by the fungal pathogen *Gaeumannomyces graminis* (Sacc.) Arx & D. L. Olivier var. *tritici* (*Ggt*). It is considered one of the most damaging root diseases of cereals (Cook, 1994). To study the precision of sampling strategies required for commercial assessment of *Ggt* a wide range of field soils were collected from throughout Australia. DNA was extracted from the soils and soil organic matter using the technique of Herdina *et al.,* (1997) and compared to soil bioassays for *Ggt* (Herdina *et al.,* 1997). Variability attributed to sample size, within field variability and source of DNA (either total soil or soil organic material) were studied so that a DNA-based assay could be combined with a regression model for the relationship between growing season rainfall and yield loss at various *Ggt* levels (Roget and Rovira, 1991). This could be used by farmers to manage their take-all risk. Variability could be reduced by increasing sample size and by extracting the total soil organic matter prior to DNA extraction. A sample size of 500g/ha was sufficient to differentiate risk categories with a probability of 95%. Although the correlation between the DNA-based system and the soil bioassay was poor the level of take-all disease severity (yi) can be predicted using the equation

$$yi = 0.88\ xi - 3.55$$

where xi = pg of *Ggt* DNA in 0.1 g of soil organic matter. The levels of *Ggt* in the soil organic matter of <30, 30-50 and >50 pg correlated to take-all disease levels of <20% (low), 20-40% (moderate) and >40% (high) of seminal roots with lesions. The expected yield losses from crops with these levels of take-all would be <10%, 10-30% and >30%. The knowledge of the risk of take-all allows farmers to change their management (eg rotation with a non-host, fungicide treatment or delayed sowing) prior to planting the crop. This technology is currently being used by farmers to detect and predict take-all (Herdina and Roget, 2000).

In the case of *diseases*, the levels of the pathogen itself may be determined or an assessment is based on the extent of symptoms or signs associated with the disease.

Similarly, *insect population estimates* may be based on absolute or relative estimates. Absolute estimates of insect numbers use actual insect counts that may be from the number of eggs of *Heliocoverpa* on a sample of cotton plants or the number of pupae/m^2. The accuracy of this method of sampling depends on the sampling efficiency (a factor of insect density and size) and errors made in the assessment. This form of assessment is time intensive. Relative estimates of insect numbers are used more widely and consist of counts of insects that do not relate to a pre-defined sampling unit. Therefore, this type of estimate is only directly comparable under the same sampling conditions. The methods most often used are based on traps. Traps can either affect an insect's behaviour (such as pheromone traps) or be mechanical (*e.g*, pitfall traps or suction traps). Interpretation of trap data depends on the various factors that affect the efficiency of the trap, e.g. wind speed on pheromone traps and moonlight on light traps (Bishop *et al.,* 2000). Both methods are dependent on the insect phase effect (specific insect behaviour or physiological state of the insect).

The inoculum of *air-borne pathogens* can be quantified using techniques and instruments that are used to quantify pollen or dust in the air. Most air-borne pathogens are fungi as this group of organisms is best evolved for air-borne dispersal. Rust fungi, for example, can be dispersed over large distances. A number of species of rust fungi have been found in New Zealand shortly after their discovery in Australia, indicating long distance travel (Brown, 1997). The identification of the pathogen depends upon direct observation of the fungal spore or by growing the pathogen and examining cultural and spore morphology. Spore traps may be as simple as glass slides with adhesives that are exposed to the air for a set period of time or may consist of coated rods that are moved through air. Suction traps move known volumes of air over a surface. In the case of a Burkard spore trap (Figure 10.2), the spores impact onto a clear film. The spores can then be examined microscopically for fungal identification. The Anderson spore trap uses a cascade impactor to separate particles or spores of different sizes onto prepared agar on which the pathogens can be grown and identified. These methods produce large volumes of information, however the collection and identification of the spores is tedious, time consuming and requires a great deal of expertise. Such methods may be used if the disease develops steadily under uniform weather conditions (Kerr and Keane, 1997) but are not used widely in commercial disease monitoring situations.

More commonly, plant diseases are assessed based on the incidence or severity of disease symptoms. The methodology of plant disease assessment is highly dependent on the type of disease or plant part affected, the type of crop and the rationale for the disease appraisal. Disease assessment can be based on the plant organ, whole plant or on the plant population being examined (Anon., 1948; James, 1971; 1974)

Disease severity is usually expressed as a percentage of the plant part affected. Cobb (1892) was the first to publish pictorial keys to indicate the severity of rust on leaves of wheat in Australia. He used a scale that ranged from one to fifty% severity, but indicated that pustule area probably did not exceed 37% of the leaf area. Melchers and Parker (1922) and later Petersen *et al.,* (1948) modified the Cobb scale by multiplying all percentages by 2.7027 (effectively making Cobb's 37% equivalent to their 100%) and by adding a further diagram for 65% severity. Petersen *et al.* (1948) continued to add further divisions to this scale to make it linear. They also used varying pustule sizes in their keys. Horsfall and Barratt (1945) incorporated aspects of the Weber-Fechner Law that states that the eye distinguishes according to a logarithmic scale of light intensity. Below 50% severity the eye sees diseased tissue, whereas above this it distinguishes disease-free area. Both Chester (1950) and James (1974) criticised the seemingly large gaps between severity levels in logarithmic disease assessment keys and suggested the use of further divisions. They both realised however that, although there would then be more divisions, approaching a linear scale, these could not be distinguished by the human eye.

Figure 10.2: Burkard spore trap

In *disease assessment*, it is also important to indicate the sampling unit and to give an indication of the physiological age of the plant at the time of assessment (Cobb, 1892; Large, 1966; James, 1971). Growth stage keys such as those of Large (1954) and Zadoks *et al.* (1974) are frequently used for cereals. The time spent sampling may be reduced if (i) relationships between incidence and severity can be established (Rayner, 1961; James and Shih, 1973; Seem, 1984), (ii) a sequential sampling technique can be developed (Cole and Gaunt, 1984), (iii) reduced sample size is used (Analytis and Kranz, 1972) or (iv) the number of plant organs sampled can be reduced (Cobb, 1892, Khan, 1987). The application of these methods requires a detailed knowledge of the disease progress of the pathogen and the potential losses it may cause.

Once serial observations of disease have been made, these can be plotted against time to produce a disease progress curve. These progress curves usually take the form of a growth (frequency) curve or a cumulative curve. The former curve has both an increasing and a decreasing phase. The cumulative progress curve is most used by plant pathologists and is formed by progressively summing the disease assessments. This most often leads to a sigmoid curve which has logarithmic exponential and transitional phases and a plateau or asymptote (Baker, 1971). According to Madden (1980), quantification of disease progress curves can be used in evaluation of control strategies, prediction of future levels of disease and verification of plant disease simulators or forecasters.

ECONOMICS AND YIELD LOSS RELATIONSHIPS

Economic threshold

To reach an economic crop yield it is often necessary to tolerate some level of damage caused by the pest or pathogen. The criterion most often used to determine the amount of damage (or level of pest population) which can be tolerated is the economic threshold (Stern *et al.,*1959). Above this threshold the cost of control of the pest is out-weighed by the potential losses due to the pest.

Carlson and Main (1976) considered economic thresholds to be based upon:

- the cost of control, which includes the cost of the chemical, the application costs and any damage resulting from application of the chemical;

- the value of crop loss which could have been prevented, and
- forecasts of disease intensity, so that timing of control can be determined.

In some cases, the pest may be allowed to cause some damage as long as the crop has the potential to compensate for these losses. The compensation point, or tolerance level, is dependent on the growth stage of the crop when the pest becomes evident, as well as crop management practices, geographic location and climatic factors (Main, 1977). In addition, the timing of control is dependent on the properties of the pesticide (Rowell, 1985) and the susceptibility of the host to the pest (Brown *et al.,* 1986; Rowell, 1973).

Yield reduction

Pests may reduce yield in three primary ways, that is, by reducing the plant population, diverting nutrients from the produce or destroying the marketable product.

In the establishment phase of crops, *losses of individuals* can occur through the activity of insects such as plague locusts (*Chortoicetes terminifera*) or army worm (*Pseudaletia convecta*). Seedling pathogens such as species of *Pythium, Phytophthora* and *Rhizoctonia* may affect establishment. High inoculum levels of pathogens such as *Puccinia striiformis* and *Leptosphaeria maculans* may also destroy established seedlings.

Once the crop becomes established, pests may *divert nutrients* from the crop. Sucking insects such as aphids and grazing pests such as mites may act at any stage of plant development. Diseases like rusts (caused by *P. recondita, P. striiformis*), blackleg of canola (caused by *Leptosphaeria maculans*), Septoria leaf blotches (caused by *Septoria tritici*) and cereal cyst nematode (*Pratylenchus thornei*) may all affect crop yields by competiting for nutrients or reducing leaf or root growth (Ash and Brown, 1990, 1991; Wellings *et al.*, 1985; Bailey *et al.,* 2000; Cavelier and Couvreur, 1995; Khangura and Barbetti, 2001; Nicol *et al.,* 1999).

Insects may directly *attack the marketable product* such as is the case when cotton bolls are destroyed by species of *Heliocoverpa* (Wilson *et al*., 1972). Some diseases, notably smuts and bunts (caused by *Ustilago tritici*, *U. nuda* and *Tilletia spp.*), may also reduce yields (Brown, 1975), although losses from these diseases are sporadic.

When assessing insect intensity, development stages that have a similar effect can be grouped together. In the case of aphids, for example, adults and fourth-stage instars are equivalent in terms of damage, however first to third instar larvae produce one-third the damage or 0.33 adult equivalents (Wratten *et al.,* 1979).

Yield loss relationships

A crucial element in any pest management system is the relationship between disease intensity and crop loss (Teng *et al.,* 1980). Once it has been established that a particular disease does cause sufficient yield loss to warrant control, attempts should be made to quantify the disease-loss relationship (James, 1974). Disease-loss models can be categorised into three main types, empirical, conceptual and explanatory (Teng, 1979). Empirical models are usually considered to be either of critical point, multiple point or area under the disease progress curve types (James, 1974). These types of models do not take account of the causal relationships between variables and as such are purely descriptive (Teng, 1979).

Critical point models are of the form

$$Y = a_0 + a_1X$$

where *Y* is the percentage yield loss, *X* is the measure of disease present at a critical point, a_0 is the Y-intercept and a_1 is the slope of the relationship. Critical point models assume that neither the infection rate nor the shape of the epidemic progress curve is important in determining the final yield loss, and as such are best suited to modelling short, late epidemics (James, 1974). Critical point models have been successfully used to model the relationship between yield loss in a variety of cereals due to disease (James *et al.,*1968; Slootmaker and van Essen, 1969; Romig and Calpouzos, 1970; Eyal and Ziv, 1974; Kingsolver *et al.,* 1984). The relationships derived by Doling and Doodson (1968), Mundy (1973) and Brown and Holmes (1983) were established using field trials whereas those of King (1976) were derived using single tillers from commercial fields. The two main criticisms of critical point models are that they ignore the physiological basis of disease loss and they are usually not applicable outside the situation in which they are developed.

Multiple point models, as the name implies, are derived from a series of disease evaluations throughout the growing season. These types of models take the form

$$Y = a_0 + a_1X_1 + a_2X_2 + \ldots + a_qX_q$$

where *Y* is the percentage loss in yield and $X_1 \ldots X_q$ are sequential disease recordings. James (1974) considered the use of multiple point models appropriate to situations where there was variability in infection rates, early or long season epidemics or where yield was accumulated over a relatively long period. He also concluded that inclusion of more than one point even when there is a short, late epidemic may improve the accuracy of the model. Notable examples of multiple point models are those that have been developed for wheat leaf rust (*P. recondita*) in the United States (Burleigh *et al.*, 1972) and barley leaf rust (*Puccinia hordei* Otth.) in New Zealand (Teng *et al.,* 1980). Brown (1988) found that the use of a multiple point model increased the accuracy of yield loss prediction of stripe rust on wheat (as determined by the coefficient of determination) but considered this slight increase in accuracy did not warrant the use of this technique in practical situations.

Area-under-the-curve model,s as advocated by Van der Plank (1963), try to relate the area under disease progress curve (AUDPC) to yield loss. James (1974) considered AUDPC models as intermediate between critical point and multiple point models. Their main advantage over critical point models is that they can distinguish between the losses caused by epidemics of different duration but the same disease rating at a critical point (James, 1974). Buchenau (1975) found this model the most appropriate for modelling the relationship between stem and leaf rusts to disease loss. Seck *et al.* (1988) also found that this model gave good results when relating yield loss in wheat to leaf rust intensity. Other workers have found that critical point and multiple point models were more accurate than AUDPC models in predicting yield loss (James *et al.*, 1972; Schneider *et al.*, 1976; Arnold, 1977; Teng *et al.,* 1980). Romig *et al.,* (1969) found that the AUDPC from high incidence-short duration stem rust epidemics did not correlate well with yield loss in spring wheat. The multiple point and AUDPC models, even though they may increase the accuracy of the prediction in many cases, may not be warranted because of the over-riding concern of sampling costs. In all cases the suitability of the model depends on the reason for developing the model originally (Teng *et al.,* 1980).

Final yield of crops is dependent on the interaction between the yield determinants such as tiller number or head number, seed weight, spikelet number pod size and/or grain number per spikelet. Each of these parameters is differentially affected by the timing of the disease. *Conceptual response models* try to account for this by relating the timing and the severity of the disease epidemic to the final yield loss. Conceptual response models in cereals were first used by Calpouzos *et al.* (1976) who related the timing of the epidemic and the epidemic slope to the yield loss in wheat caused by stem rust. Teng and Gaunt (1980) also used a conceptual response model to relate loss in barley to leaf rust intensity in New Zealand. Most of these models can be quantified by the use of simple split-plot field trials using varying spray regimes and cultivar resistance.

Explanatory models are much more complex models that not only explore the relationship between a particular pathogen and yield loss but also include soil, environment and crop information. These types of models are best illustrated by simulation models (Teng, 1979) where yield loss relationships are incorporated into crop growth models such as those of Johnson *et al.,* (1986) for potato, Thorton and Dent (1984) for barley leaf rust and Rabbinge and Rijsdijk (1983) for cereal pests and diseases in Europe. Synoptic crop-loss models (Stynes *et al.,* 1979; Veitch and Stynes, 1979; 1981; Veitch and Stynes, 1982; Stynes *et al.,* 1981) are seen as intermediate between multiple point empirical models and explanatory models. These types of models are derived from multiple regression equations but strive to incorporate realistic estimates of the impact of environmental factors, soil characteristics and disease on yield.

Remote Sensing

Remote sensing, the acquisition of data by a recording device not intimately in contact with the plant/pest, is being developed as a means of acquiring information about the extent and severity of a pest infestation. The platform, which supports the remote sensing device, may range from hot air balloons through to airplanes, helicopters and satellites. The sensors they support generally collect part (or parts) of the electromagnetic spectrum. The majority of remote sensing systems are mounted on airplanes or satellites and collect radiant electromagnetic radiation (EMR) in the visible through to the infrared part of the spectrum (Campbell 1996; Jensen 1996). Remote sensing systems that collect EMR in this range are known collectively as optical systems. Even within the optical range of remote sensing systems there are many different types of sensors and platforms in use. All produce different types of remote sensor data.

The value of remote sensing systems can be measured in terms of their resolution. 'Resolution' refers to not only the smallest object detectable in an image (spatial resolution) but also to the number, range and width of spectral capture (spectral resolution), time between repeated captures (temporal resolution), and the ability of a sensor to detect variations in signal strength (radiometric resolution) (Campbell 1996; Jensen 1996).

Moran *et al* .(1997) found that remote sensing technology has greater potential for detecting and identifying crop diseases based on the associated physiological effect the disease has on leaf and canopy elements. Research has shown that disease induced stress caused general physiological changes to the plant resulting in changes to the spectral response (Toler *et al.,*1981; Lorenzen and Jensen, 1989). For example, necrotic disease can cause a darkening of leaves in the visible spectrum and cell collapse that would decrease near infrared reflectance. Chlorosis induced by diseases of fungal and viral origin causes marked changes in the visible reflectance (similar to N deficiency). Other diseases may be detected by their effects on canopy geometry (wilting or decreases in leaf area index) (Everitt and Nixon 1986; Carter and Miller 1994) and the pattern of spread within the field.

Fouché and Booysen (1996) found remote sensing using an airborne platform useful in capturing reflectance differences between diseased and healthy trees. They examined rootrot, caused by *Phytophthora cinnamomi* in an avocado orchard, and evaluated a fungicide treatment in cashew nut trees. Fouché and Booysen (1996) used a classification technique supported by field observations to characterise the difference between diseased and healthy lemon trees. Significant differences were found in all cases between the two classes.

Remote sensing has been used to examine the indirect effects of insect damage on plants. Moran *et al.* (1997) found few studies on insect-induced stress, but discovered that remote sensing has been used to detect insect habitat (Hugh-Jones *et al.,* 1992), the growth and yield of plants (Vogelmann and Rock 1989), or changes in plant chemistry. Peñuelas, *et al.* (1995) found the visible and NIR wavebands useful in identifying the effect mite populations had on apple trees. They found that increasing mite pressure reduced the leaf chlorophyll. The results of this were first identified in the visible spectrum

(Peñuelas *et al.,* 1995). Incidence of a disease can be used as a rapid method of assessing disease. In some cases, such as floral smuts of wheat, incidence equals severity. However, in most other cases, incidence and severity are linked by a relationship which is determined by the pathsystem; the spatial distribution of the pathogen or disease stage of the epidemic and the controls applied (Ash, 1990).

THE ENVIRONMENT AND PESTS

The factors that are thought to affect plant disease are often conceptualised in terms of the disease triangle (Figure 10.3). In this concept the three factors - host, pathogen and the environment - interact to affect the potential for a disease epidemic. In a similar way, the environment can also have a direct effect on insects and their interactions with their host. Temperature is the most important influencing factor in insect development. The amount of heat required (physiological time) can be used to predict the time required for an insect to complete a developmental process or stage. This physiological time is calculated from the summation or accumulation of time by temperature and is expressed as day degrees (Southwood, 1978). To calculate physiological time, a determination of the threshold temperature for the insect must be determined. Although physiological time is often assumed to be fixed for each stage, it may be influenced by fluctuating temperatures, the insects' diet and host plant (Foley, 1981; Williams and McDonald, 1982). When using temperature to predict changes between stages, it is most difficult to define the initiation of the event (e.g. the conclusion of diapause) and so errors may be introduced into day-degree calculations (Pruess, 1983). The transmission of viruses can be affected by the mobility of vectors, which is affected by temperature and the interaction between the virus and its vector (non-persistent or persistent) (Jeger *et al.*, 1998; Irwin 1999).

Temperature can also be considered one of the most important direct determinants of disease progress. In most cases, the optimum temperature for disease development mirrors the optimum for the host and the pathogen. However, in some cases, the disease progresses more quickly when the plant is stressed. For example, the growth of *Fusarium oxysporum* f.sp. *lini* and the development of flax wilt (caused by this pathogen) are at an optimum SY 24°C. However, the optimum for disease development may be different in different hosts. This is graphically illustrated in Figure 10.4.

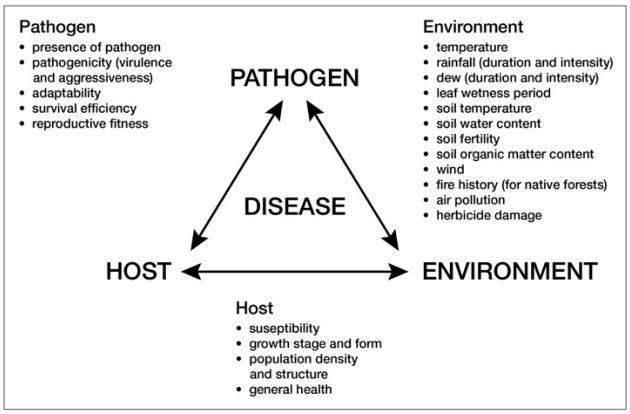


Figure 10.3 Disease triangle (Kerr and Keane, 1997)

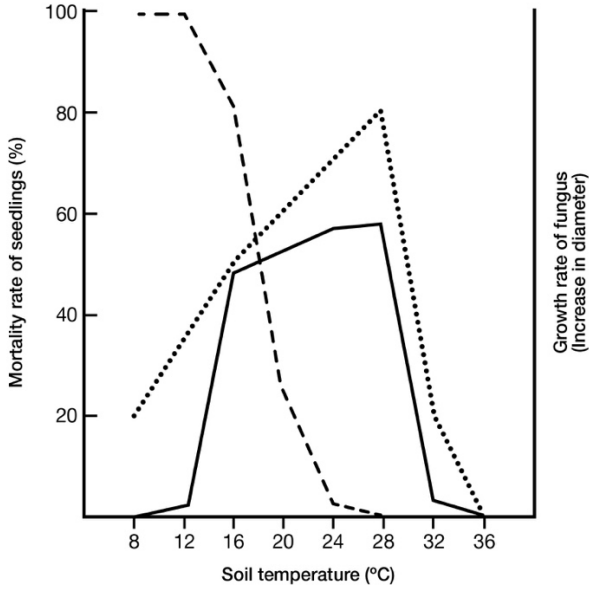


Figure 10.4: Relationship between soil temperature, the growth of *Gibberella zeae* in culture (dotted line) and development of disease caused by this pathogen in wheat (full line) and maize (dashed line) (Data from Dickson, 1923 as cited by Manners, 1982)

Temperature can also affect resistance genes within the host and so the outcome of the host:pathogen interaction. Park *et al.* (1992) and Ash and Rees (1994) described the effect of post inoculation temperature and the interaction of light intensity on the resistance of some Australian wheat varieties to stripe rust. Higher temperatures and lower light intensities were found to increase resistance in some varieties. Ash and Rees (1994) proposed that this may affect survival of *P.striiformis* f.sp. *tritici* (the cause of stripe rust of wheat) over the summer period in Australia and the inclusion of these type of data may improve regional forecasting of the disease (Ash *et al.*, 1991). Moisture (rain, dew, and irrigation) is also crucial to infection by most plant pathogens and plays a role in dispersal of many, such as the case in splash dispersal of *L. maculans* (West *et al.*, 2001). The interaction of dew period and temperature has often been used in the prediction of the severity of plant diseases. For example Webb and Nutter (1997) clearly demonstrated the effect of temperature and dew period duration on the epidemiology of rust of lucerne (Figure 10.5).

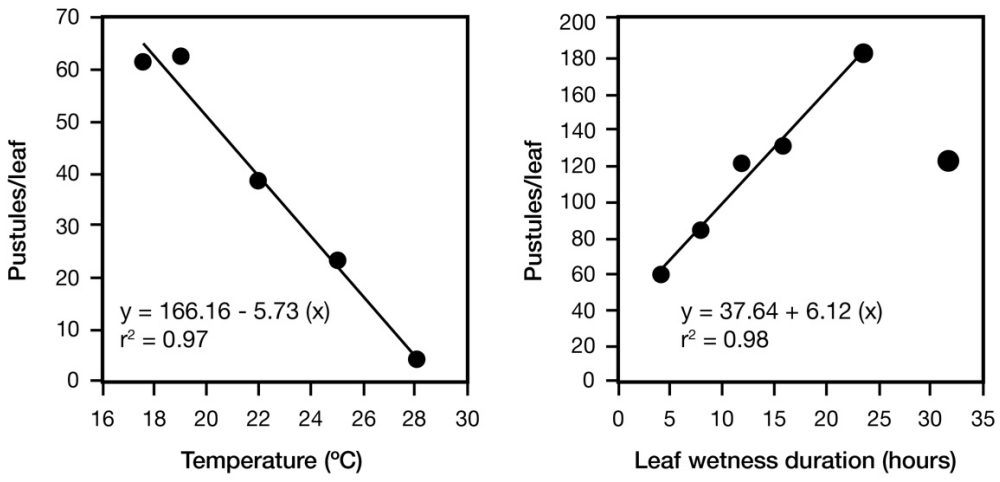


Figure 10.5: The effect of temperature (a) and leaf wetness duration (b) on pustule number in lucerne (redrawn from Webb and Nutter, 1997)

PRINCIPLES OF CONTROL

There are two major areas of pest control, preventive and curative control. *Preventive control* methods attempt to reduce the number of contacts, or the effect of those contacts, between pest and host. *Curative control* methods attempt the destruction of some or all of the pests present with the host. Both methods involve the application of the principles of exclusion, avoidance, protection and eradication, applied either as regulatory, physical, cultural, biological or chemical methods of control.

With *exclusion* the intention is to keep pests out of previously 'clean' areas by the erection of artificial barriers. These barriers may be of a regulatory or of a physical character. Regulatory barriers include quarantine and inspection measures like seed certification; physical barriers include vermin-proof barriers such as the dog-proof fence separating Queensland and New South Wales, flyscreens, or chemical repellents and attractants used with chemical killing agents.

Cultural strategies such as time of sowing can be used either to keep the crop away from the main period of pest attack or to minimise its effects by *avoiding* conditions favourable for their activity. General crop management practices should aim at increasing crop vigour while, at the same time, making the environment unfavourable for the development of the pest species.

All management practices, which, in the presence of the pest, favour crop development, are implied in the principle of *protection*. Biological, physical, chemical and cultural control methods all have a role to play. General farm hygiene is essential. Where practicable, all refuse and diseased material must be destroyed. Roads, fence-lines and storage areas must be kept free of weeds; machinery and storage areas must be kept free of insect pests that attack grain.

The destruction of the pest in the presence of the crop is the function of *eradication* techniques. In this context the term is not confined to the complete removal of an established pest, but refers to any significant reduction in its population.

General methods of control

Quarantine and regulatory control

Quarantine refers to the isolation of one area from another by physical and commercial barriers to prevent the introduction and spread of pests and diseases. Experience has shown that introduced pests and diseases are frequently more damaging and wide-ranging in their activity in a new environment. This is partly due to their escape from the biological constraints present in the area of origin and partly because the new host populations are genetically susceptible as they have not previously been exposed to the pest or disease.

The exclusion of pests and diseases is an important step in control programs. Freedom from pests and diseases not only lightens the economic burden of producers but opens additional markets from which infested produce may be excluded. The huge volume of modern traffic in people and commodities prevents quarantine from being 100% effective but it does slow down the entry and spread of pests.

One difficult aspect of quarantine is the fact that the diseases present within Australia have not been totally catalogued. New diseases of existing crops in Australia are constantly being reported (Ash and Lanoiselet, 2001a; b; Lanoiselet *et al.,* 2001**;** Nobbs *et al.,* 2001; Washington and Pascoe, 2000). In implementing quarantine, the community through the action of the legislating government, recognises the need to protect established and developing agricultural industries. It is a function that requires legal sanction to be firmly applied. Failure to enforce quarantine restriction by inspections and legal sanctions leads to breakdowns in the system. This is because quarantine reaches into all human

activities and restricts the normal rights and privileges of a citizen. Many people find this irksome or economically damaging. Consequently adequate procedures must be developed to allow safe movement of articles from infested areas. Fumigation, dipping or other commodity treatments can be applied where appropriate.

Plant quarantine requirements have the effect of slowing plant introductions and thereby increasing the cost of breeding programs. With due cognisance of this disadvantage, however, Australian quarantine allowed entry of several maize, sunflower and wheat introductions. Since 1968 these methods have allowed the development, at Tamworth, of the Australian wheat collection with more than 14,000 introductions now available to all wheat breeders. Such genetic collections not only preserve material but reduce quarantine risks by rationalising plant introduction, thus alleviating costly and frustrating delays in plant improvement programs.

Principles behind imposing quarantine restrictions on imported plants and plant products into Australia apply also for inter and intra state quarantine. A number of important plant pathogens and insect pests, for example, are restricted to certain regions or states and there is good reason to apply quarantine to contain them.

Quarantine consists of four operational modes: exclusion, surveillance, containment and eradication.

Exclusion Most of our existing 'pest' problems are a legacy of the absence of quarantine in our early days of settlement. Australia is still relatively free, however, from the world's most harmful 'pests' (including rabies, foot and mouth disease, khapra beetle, screwworm fly, fire-blight disease). This is despite the tremendous increase in worldwide movement of people and goods, and is evidence of the effectiveness of our quarantine services. Exclusion of such harmful exotics is vital to the continued efficient production and protection of our export markets.

Surveillance The establishment of Australian National Animal Health Laboratories (ANAHL), the animal quarantine station on Cocos Island, and the upgrading of post-entry plant and animal quarantine facilities throughout Australia (capital expenditure in excess of $100 million) are indications of the Australian Government's commitment to maintain our relative 'pest'-free status by world standards. These developments also facilitate entry of genetic material of potential value for plant and animal improvement.

Surveillance is important for the early detection of new introductions or 'flare-ups' of internal quarantines. Active programs such as trapping for fruit flies and screwworm flies operate in far North Queensland and Torres Strait Islands, but a national crop surveillance scheme similar to the Victoria Crop Information Service is required.

Containment In devising unified 'pest' control programs within Australia, quarantine should be considered as one of the methods available for use in combination with others. Quarantine, for example, had the potential to contain annual ryegrass toxicity in South Australia in the 1950s but unfortunately was not considered.

Eradication Since the establishment of the eradication program in 1977, achievements include the eradication of Giant African Snail and Potato Spindle Tuber Viroid and containment of Black Sigatoka disease of bananas to the Cape York area.

Table 10.1: Plant quarantine - some examples of important pathogens not present in Australia

Common name of disease	Pathogen species	Hosts
Fire blight disease	Erwinia amylovora	Apples, pears some ornamental shrubs
Dutch elm disease	Ceratocystis ulmi	Elm trees
Avocado sunblotch viroid	Avocado sunblotch viroid	Avocados
Plumpox virus	Plumpox virus	Plums
Soybean cyst nematode	Heterodera glycines	Soybeans

Case Study 2: CLIMEX and DYMEX simulations of the potential occurrence of the rice blast disease in south-eastern Australia. (Lanoiselet *et al*., 2002)

Australian rice is relatively free of diseases with only a small number of minor diseases being recorded (Lanoiselet *et al*., 2001, Cother and Nichol, 1999). Rice blast caused by *Magnaporthe grisea*, the most significant disease of rice worldwide, does not occur in Australia, although it has been recorded in 85 other countries (CMI, 1981). The pathogen has never been reported from the rice growing regions of Australia although it has been reported from weeds from several states. Lanoiselet *et al*. (2002) used available information on the epidemiology of rice blast (e.g. Hashioka, 1965; Suzuki, 1975; Hemmi and Imura, 1939; Ou, 1985), to construct models of the pathogen to predict its likely establishment if it were to be introduced into Australia.

The model was validated using independent data from California. The results from the models indicated that rice blast represents a significant threat to the Australian rice industry, if it were introduced, as it would increase and reproduce readily under Australian conditions. Australian rice varieties therefore should be screened for resistance to blast overseas.

Physical or mechanical control

Mechanical sieving and grading of seed and separation by flotation are important means by which seed for sowing can be freed of various pathogen propagules such as galls and sclerotes. Tillage can also help in the control of diseases. Cultural practices such as deep ploughing of diseased crop residue and burning of crop stubble are important physical means used to suppress disease caused by *Sclerotinia* spp. and other organisms which survive in stubble, but which produce aerial spores to infect subsequent and adjacent crops.

The physical agents most commonly used in controlling plant diseases are temperature (high or low) and various types of radiation. These methods have greater application in intensive, high value horticultural and vegetable crop situations. Heat treatments are used for soil sterilisation (live steam, electric, hot water), controlling infections in propagative organs (hot water, virus-free apical growth at high temperatures). Post-harvest heat therapy has been used to give control of brown rot (*Monilinia fructicola*); peaches are heat treated before full maturity for 24 hours and then ripened at 24°C to reach full ripeness without brown rot development and with minimum losses from *Rhizopus* and other rot fungi. Refrigeration is probably the most widely used method of controlling post-harvest diseases of fleshy products (Agrios, 1997). The reduced temperatures do not kill the pest or pathogen, but reduce their growth or activity. Gamma irradiation has also been found to be a satisfactory method of destroying pests in fruits such as tomatoes and strawberries (Agrios, 1997)

Cultural control

Cultural control is defined as the tactical use of regular operations to reduce the activity of pests. It specifically excludes the use of introduced biological agents and pesticides. Cultural control is important because it generally involves minimum cost and avoids or reduces adverse environmental effects. The techniques involve practices that modify the environment to favour crop growth but discourage or avoid conditions that favour pest development. The likelihood of pest problems as well as considerations such as fertility status of the soil needs to be taken into account in any crop production system. In all instances, a detailed knowledge of the biology of the pest and the key factors that influence its main activity are required to take full advantage of cultural practices.

Cultural methods, which can be manipulated to control disease, include mineral nutrition, time of sowing, special cultivation, water management, crop rotation, sanitation and clean seed.

Mineral nutrition The addition of well-balanced artificial fertilisers promotes vigorous growth of crops which are then generally better able to withstand the deleterious effects of many pathogens. Mineral nutrition is also an important factor affecting the predisposition of a plant to pests. Water stress, fertilisers and growth regulants may all affect the suitability of the host crop for growth, survival and reproduction of insect pests (Coaker, 1987). Different types, levels and balance of nutrients influence the severity of pests amongst the various host/pathogen combinations. This results from an interaction between modified host susceptibility and pathogen virulence. In some cases, even different forms of the same nutrient are known to affect pathogen virulence differently. For example, high levels of nitrogen have been shown to increase the levels of some wheat diseases (Darwinkel, 1980; Leitch and Jenkins, 1995) which can translate to increased relative yield losses (Ash and Brown, 1991). Ash and Brown (1991) attributed this to a reduction in translocation of nitrogen from diseased leaves. Fertiliser application may also increase aphid reproduction (Gash *et al.,* 1996).

Time of sowing by Adjustments to time of sowing can make it possible to avoid the main period of pathogen activity. For example, early sown cereal crops frequently suffer more severe attacks by a number of important fungal leaf pathogens than do later sown crops. With increased knowledge of pathogen behaviour and a better understanding of the genetics of controlling phasic development of cereal plants, new cultivars could be bred allowing a greater degree of flexibility in sowing time specifically to avoid severe damage certain pathogens. This means of control is usually not very easy to use because of crop constraints.

Special cultivation Deep burial of crop residue is known to have beneficial effects by reducing the seasonal carry-over of inoculum associated with diseases such as eye-spot lodging of cereals and bacterial blight of cotton. The type of cultivation can affect soil insects by changing the soil environment, exposing the insects to natural enemies or by direct damage from the implements used (Stinner and Howe, 1990),

Water management Where crops are irrigated, both the amount, timing and method of watering can be altered to suppress the effects of disease. Overwatering, for example, is conducive to the activity of damping-off organisms like *Pythium* spp. and *Phytophthora* spp. Spread of diseases such as *Septoria* spots and blotches and bacterial blights, which are normally spread by raindrop splash, are enhanced by overhead watering. Flooding, where practicable (*e.g*. rice paddies), reduces the level of disease-causing organisms such as plant-parasitic nematodes by creating anaerobic conditions.

Crop rotation Crop rotation is one of the oldest approaches to disease control in cultivated plants and is just as important today; even more so where more intensive cropping systems have evolved. Its principal function is to eliminate pests or to sufficiently reduce their levels to allow worthwhile crop yields. This is usually achieved by replacing readily available sources of preferred or susceptible hosts

with non-preferred or resistant hosts in the cropping sequence, thereby interrupting the cyclic activities of the pest. Crop rotation is most effective against pest species that have a narrow host range and limited dispersal.

Although uncommon, it is possible for rotations to completely eliminate certain pathogens. Examples include certain obligate parasites that can survive only on living host tissue and whose survival depends on resting propagules in soil which are relatively short-lived. The chances of reinfestation or reinfection from outside sources, however, are often high, either from windblown propagules (fungal spores) or from various introduced sources of contamination (hay, seed, soil on stock or implements).

Sanitation assumes particular significance when allied to the effective use of rotations. For example, weeds infesting neighbouring uncultivated land, such as fence lines and gullies, may constitute a ready source of re-infestation of either the weed itself or some pathogen or insect pest for which it is a host. Crop rotation is not particularly effective against diseases that have exceptional powers of dispersal.

Although rotation plays a significant role in modifying the effects of many pathogens which possess a soil-borne phase, it is ineffective against organisms such as *Rhizoctonia solani* (bare-patch disease), the root-rotting fungus, which possesses a wide host range and a strongly competitive, saprophytic ability. The practice of rotation nevertheless is an essential feature for the commercially viable production of many field crops including peas, cotton and canola that would otherwise succumb to the build-up of diseases. Crops such as canola have been shown to release glucosinolates during decomposiition which contribute to their so-called biofumigation effect (Warton *et al.,* 2001; Kirkegaard *et al.,* 2000).

Sanitation Sanitation includes all activities aimed at eliminating or reducing pathogen levels in association with plants, paddocks, machinery and storage facilities, i.e. from any refuge acting as a source of spread to other healthy plants or plant products and into clean areas. In many instances, sanitation forms an important part of an overall disease control program and in some cases, control of certain diseases relies largely or solely on proper sanitary measures.

Practices vary in scale of operation - from decontamination of stock and farm machinery, spot spraying of weeds or roguing weeds or diseased plants, to broad-acre disposal of diseased crop residue either by deep burial or by burning. Normal farm hygiene (weed-free areas along fencelines and around buildings, clean grain storage areas) and the purchase of clean seed are examples of sanitation practices not necessarily directed against any specific pest. On the other hand, many practices are so directed. These include the control of a weed known to be an alternative host harbouring some serious disease or insect pest, or the use of legislation to enforce proper sanitation.

Use of clean seed One important aspect of sanitation which bears special mention because of its application to all producers is the use of clean seed. Crop seed, like soil, is a natural repository for a wide spectrum of pests (insects, weeds and diseases) and hence is an ideal vehicle for the introduction and spread of new pests and diseases into 'clean' areas or for their re-establishment in already infested areas. Seed-borne diseases are especially important, as representatives of all major groups of disease-causing organisms (viruses, fungi, bacteria, nematodes) can be disseminated by seed.

In horticulture, seed may be taken to include propagative material such as cuttings, rhizomes and corms. Such material can be infected by a wide array of diseases which may be passed on directly to the vegetative growth of new progeny. All the individuals in a crop thus propagated may become diseased, and it is therefore particularly important in this industry to ensure that planting is carried out with disease-free stock.

A farmer or grower should endeavour to establish a crop with 'clean' seed. Unfortunately, it is usually impossible to detect contaminated seed by superficial examination. He must therefore treat his own

seed as appropriate (seed dressings) or rely on other agencies and schemes such as seed certification and regulatory measures (Agricultural Seeds Act) where they exist, or on seed producers or cooperatives to supply him with clean seed. Such seed has either been produced from a parent crop inspected and found free of designated disease and/or has been tested for freedom from disease or treated in some way either chemically (seed dressings), physically (heat therapy - hot water) or mechanically (various mechanical graders and liquid separators) to exclude, destroy or remove specific seed-borne diseases.

Breeding for resistance

The use of varieties of plants resistant to particular diseases is one of the main methods of disease control. Host plant resistance is the inherent ability of crop plants to restrict, reduce or overcome a pest infestation (Kumar, 1984). This definition includes the concept of tolerance, in which a plant variety will be able to tolerate a level of pest infection without a reduction in yield. Many authors consider that tolerance should be treated separately from true resistance (Keane and Brown, 1997; Beck, 1965).

The main advantages of using resistant cultivars are the saving in cost to the landholder, the absence of poisonous residues which pollute the environment, independence from high energy sources based on fossil fuels and easy integration with chemical control methods if warranted. Additionally, for some pests, there are no other economic or effective controls available.

Biffen (1905) was the first to demonstrate Medelian inheritance of major genes for resistance using stripe rust of wheat. In Australia, breeding for resistance in field crops has been mainly concerned with the control of pathogen organisms - virus, bacterium, nematode or fungus. The development of bunt-resistant wheat cultivars by W.J. Farrer after 1899 is one of the earliest examples of successful breeding for disease resistance. This was followed by cultivars resistant to flagsmut (*Urocystis agropyri*) which, unlike the resistance to stem rust (*Puccinia graminis* f.sp. *tritici*), has proven extremely stable. Breeders in some states incorporate this resistance into all new wheat cultivars.

Different forms of physical and biochemical resistance occur between and within host-pathogen combinations, but all are under genetic control (Whitney, 1976). Although the aim of the breeder is to use the most suitable form of resistance, basic information on the mode of resistance is not essential for selection in applied programs. In the conventional sense, plant breeders rely on sexual recombination occurring for the transfer of resistance genes. In more recent times they have genetically engineered inter-specific transfer of genes and chromosome segments to confer resistance to several diseases. Irradiation and several chemicals have been used to transgress these characters (McIntosh, 1976; Brock, 1977). These processes made possible the transfer of rust resistance from *Aegilops* and *Agropyron* species to cultivated wheat (Sears, 1956; 1972; Knott, 1961).

Sources of resistance are commonly obtained from regions where plants have developed a natural resistance to specific pests through co-evolution. The germplasm from such wild species often provides satisfactory resistance after hybridisation with cultivated species. Another common approach to identifying sources of resistance is to screen cultivated species and lines collected from a wide geographic area following either artifical inoculation or exposure to natural infection in the field. In some cases it is even possible to breed a resistant variety from a resistant individual.

In the last ten years it has become possible to introduce 'anti-pest' genes from a range of sources using molecular biology. Genes for increasing resistance to insects are either plant derived genes or genes for *Bacillus thuringiensis* (*Bt*) endotoxins or other non-plant toxins (Dent, 2000). Plant genes that code for inhibitors of digestive enzymes or secondary metabolites have been investigated (Gatehouse *et al.*, 1998; Hallahan *et al.*, 1992; Llewellyn and Higgins, 1998). *Bt* is a soil bacterium that produces a range of toxic protein crystals. The bacterium and the toxin have been used extensively as biocontrols. The

production of the endotoxin by transformed tobacco was first reported by Vaeck *et al.,* (1987). *Bt* has been transformed into tomato, cotton, maize and potato (Delannay *et al.*, 1989; Perlack *et al*., 1990; Koziel *et al.*, 1993; Merritt, 1998). Cotton expressing the *Bt* genes has been grown commercially in Australia since the 1994/95 season (Constable, 1998).

Transgenic plants have been produced in experimental systems that express varying levels of resistance to fungi, bacteria and viruses. Resistance to fungi has concentrated on the use of constitutive expression of chitinase and glucanase enzymes, expression of pathogenicity proteins, stilbene synthesis and the use of avirulence genes (Punja, 2001). In viruses, resistance has centred on the use of virus coat protein, virus replicase, antisense RNA and antiviral proteins (Huisman *et al.,* 1992; Fitchen and Beachy, 1993).

Variability of pathogen virulence and the effect of environmental parameters on the host response to attack must be considered. The environment, for example, largely determines the expression of resistance (Park *et al.,* 1992; Ash and Rees 1994). Apparent host resistance, due to escape from infection, is possible, in which event exposure to the pathogen under different cultural or environmental conditions may lead to apparent resistance breakdown. A further problem is that selecting for resistance to a single pathogen may result in selection for susceptibility to another. Varying degrees of specialisation exist amongst host-pathogen combinations and several physiologic races of the pathogen may be widely distributed in the field. This is an important consideration when screening for sources of resistance, which are effective against all known races.

Underlying any understanding of resistance of plants to pests and, specifically, diseases, is the gene-for-gene theory proposed by Flor (1956; 1971). This theory states 'for each gene conditioning rust reaction in the host, there is a specific gene conditioning pathogenicity in the parasites'. This theory partially explains the cycles often associated with resistance breakdown in crops where resistance is equated with immunity.

Resistance to plant diseases has been classified by Van der Plank (1963; 1968) as either horizontal (non-pathotype specific) or vertical resistance (race/pathotype specific resistance). In essence vertical resistance operates against pathotypes offering immunity to these pathotypes whereas horizontal resistance acts across all pathotypes of the pathogen. Other differences between these types of resistance are given in Table 10.2 from Keane and Brown (1997).

Breeding for resistance, while generally considered to be the ideal means of controlling plant disease is not without its limitations and shortcomings. The relatively high cost of maintaining stem rust resistance in wheat cultivars, for example, has been queried and attempts to develop worthwhile resistance against troublesome soil-borne pathogens have been frustrated; the difficulties in discovery of suitable sources of resistance to the take-all fungus and the complexity of the genetics of *Rhizoctonia solani*, (the cause of bare-patch of wheat), are specific examples. The cost of plant breeding may be reduced in some cases through the use of molecular markers for desirable traits such as disease resistance.

Table 10.2 Characters of specific and non-specific resistance (from Keane and Brown, 1997)

Character	Vertical or pathotype specific resistance	Horizontal or non-pathotype specific resistance
Definition	Resistance that is effective against some pathotypes but not others	Resistance that operates to a similar extent against all pathotypes
Alternative terminology	Vertical resistance. Major gene resistance Seedling resistance Monogenic resistance Qualitative resistance	Horizontal resistance Generalised resistance Field resistance Adult plant resistance Quantitative resistance
Expression	Provides a high level of protection but is prone to 'breakdown' as new virulent pathotypes evolve after which plants are fully susceptible	Provides lower levels of resistance and does not break down. Resistance often increases as plants mature.
Inheritance	Inherited as a single gene with identifiable effect. Equated with the gene-for gene concept	Usually inherited additively. Many genes involved (polygenic)
Mechanism	Operates after pathogen has penetrated host, often being associated with a hypersensitive response in the host	Reduced rate and degree of infection, colonisation and sporulation results in a reduced rate of spore production.
Epidemiology	Reduces the amount of initial inoculum, delaying the start of an epidemic. Once epidemic starts, the disease progress curve is similar to that of a fully susceptible cultivar	Slows down the rate of epidemic development due to its quantitative nature.
Effectiveness	Most effective when a diversity of specific resistance genes occurs within and between crops. Usually used by breeders because it is easy to detect and manipulate	Most effective when there is genetic uniformity within and between crops. Often avoided by breeders because it is difficult to detect and manipulate.

Biological control

Biological control can be considered to include control based on the management of some aspect of the biology of the pathogen species - genetic manipulation, breeding for resistance in crop plants, crop rotation, grazing management - aspects which are considered separately. In the more specialised sense, it refers to the manipulation by man, of introduced and indigenous natural enemies of the disease organism/pest in order to suppress it. Plant protectionists, in particular, adopt a more general interpretation which has been modified from Garrett (1965) as 'any condition under which, or practice whereby, survival or activity of a pest is reduced through the agency of any other living organism (except man)'.

Case Study 3: Molecular markers for Cereal Cyst Nematode resistance in wheat

Cereal cyst nematode (CCN) (*Heterodena avanae Woll.*) is widely distributed in south eastern Australia. In Victoria and South Australia an average yield loss of 8.0% is attributed to CCN (Eastood *et al.*, 1991). Breeding for resistance to CCN has centred on the use of resistance from two wheat varieties, AUS10894 (O'Brien & Fischer 1974) and Festiguay (McLeod, 1976) and resistance from *Triticum tauschii* (Eastwood *et al.,* 1991). These loci have been named *Cre1, Cre2* and *Cre3* respectively. Breeding has been hampered by slow and inaccurate screening methods and inappropriate breeding strategies (Rathjen *et al.,* 1998). This has been overcome by the use of molecular markers for nematode resistance. DNA-based markers derived from sequence information from the *Cre3* locus have been used to identify wheat lines carrying resistance alleles at the *Cre1* and/or *Cre3* (Ogbonnaya *et al.*, 2001b). Furthermore, these markers have been used to locate homologous sequences introgressed from *Aegilops vestrecosa* (Ogbonnaya *et al.*, 2001a). These markers are now routinely used in breeding programs in South Australia and Victoria to select for CCN resistance in progeny.

The suppression of insects and weeds by the use of natural enemies has seen more success than the use of these types of agents against plant diseases. Two examples of successful biological control in Australia are the *classical* control of prickly pear (*Opuntia inermis* and *O. stricta*) and later, control of green vegetable bug (*Nezara viridula*). Caterpillars of the moth *Catoblastis cactorum* successfully controlled both prickly pear species, then covering 25 million hectares of agricultural and pastoral land in eastern Australia, within a few years of its introduction in 1925. In the 1950s the introduction of two strains of the wasp *Asolcus basalis* brought a dramatic decline in the numbers of the green vegetable bug, then a serious pest of a range of field crops. In these cases, the target was introduced and is referred to as classical biological control. This type of biological control is appealing, as it has a number of advantages including:

- minimal cost to the landholder;
- self-perpetuating populations of the controlling agent, responsive to changes in the pest host population density;
- non-polluting;
- no off-target effects;
- independent of fossil fuels; and
- the ability to integrate with other methods.

Alternatively, biocontrol agents of an introduced organism may be selected from within the target country, screened for pathogenicity, formulated and used in a similar way as a biocide for control. This is termed the *inundative* approach. This approach lends from both biological control and biocide application and the agent is termed a biopesticide. This type of control shares many of the advantages listed above but may lead to profit for the commercialising entity and carries fewer quarantine risks.

A biopesticide is a type of augmentative biological control agent in which an inundative application of a living organism is used to kill the target pest. In this type of strategy, massive amounts of inoculum of the organisms (usually fungi, nematodes or bacteria) are applied in an effort to manage the target. This is a general term applied to a range of pests and control organisms. Furthermore, this term does not include the use of toxins or secondary metabolites alone applied as pesticides. These chemicals, although derived from microorganisms, are simply analogous to synthetic pesticides and do not contain a living organism as an active ingredient. A parallel term to biopesticide, used to describe the suppression of the pest, is biopestistat (Crump *et al.,* 1999). These types of biological control agents, when applied to a pest, suppress the population to below an economic threshold or injury level. There is a growing interest in the use of these types of organisms in conjunction with competitive crops to

control weeds. However, they do not kill the target organism *per se*, and so are excluded from the remaining discussion of the term biopesticide.

Biopesticides have been used to control a range of pests including insects, weeds and diseases. In all of these cases, the biopesticide is packaged, handled, stored and applied in a fashion similar to that of traditional pesticides (Ash, 2002). The success of this type of control revolves around the cost of production, the quality of the inoculum and the field efficacy of the organism. In comparison to classical control, in which the cost of research and development is borne by the community, biopesticides are usually developed by commercial companies in an expectation that they will recoup their costs by sale of the product. This type of strategy can be used against both native and introduced pests. Biopesticides may be further subdivided based on the type of target pest. For example, a bioherbicide is a biopesticide developed to kill weeds. Further sub-division based on the type of agent used is also common. The term mycoherbicide is used widely to describe the formulation of a fungal agent in a bioherbicide.

Phenomena such as fungistasis and antagonism, the role of antibiotics in soil, soils suppressive or conducive to pathogens, saprophytic competitiveness, trap crops and breeding for features favourable to biological control are all potentially useful as control measures. Of particular interest is the natural field suppression of *Gaeumannomyces graminis* f.sp. *tritici*, the 'take-all' fungus, following consecutive crops of either wheat or barley, commonly referred to as 'take-all' decline. This specific suppression is thought to be caused by the antagonistic activity of one or a few organisms. Non-specific, or general suppression, thought to involve many soil organisms, has also been recognised. If these mechanisms can be elucidated then they may be able to be harnessed for effective bio-control commercially.

Case Study 4: Biological control using rhizosphere bacteria

The rhizosphere is a biologically diverse area of soil influenced by the proximity of plant roots. The bacteria that inhabit this region are often of the genera *Pseudomonas, Xanthomonas* and *Phyllobacterium*. Non-pathogenic rhizobacteria may have an effect on plant growth by producing toxins or on pathogens by a range of mechanisms including competition, antibiosis or siderophores production. These bacteria may also produce chemicals, such as salicylic acid, lipopolysaccharides and siderophore iron-regulated factors, which induce a systemic resistance to plant pathogens on plants, making these plants resistant to virulent pathogens (Alstrom, 1991; Van Peer *et al.,* 1991; Wei *et al.,* 1991). Induced systemic resistance (ISR) mediated by rhizobacteria has been reported in *Arabidopsis*, bean, carnation, cucumber, radish, tobacco and tomato using either species of *Pseudomonas* or *Serratia* (van Loon, *et al.,* 1998). ISR may be expressed as delayed symptom development, reduced disease severity and decreased incidence of disease. This type of resistance is thought to be mediated through jasmonic acid and ethylene. Furthermore, it has been hypothesised that jasmonic acid could also be involved in systemic resistance to herbivory. The interaction between the salicylic and jasmonic acid mediated pathways is unclear. In some cases suppression of one pathway by the other has been recorded. Once activated, induced resistance may remain active even under declining numbers of rhizobacteria. These bacteria may also enhance plant growth through the production of phytohormones. In fact under field conditions it has been demonstrated that plant growth promoting rhizobacteria (PGPR) can reduce disease levels and increase yield (Kloepper *et al.,* 1993; Wei *et al.,* 1996). For ISR to be effective it has been estimated that a minimum concentration of 10^5 colony forming units per gram of root need to be present (Raaijmakers *et al.,* 1995). This can be achieved by the application of the bacteria as a soil drench or as a seed coating.

Cross protection, whereby plants become resistant to pathogens following prior exposure to an avirulent strain usually of the same species, may also be useful in the control of disease. Traditionally it has been associated with the use of mild virus strains to confer immunity against subsequent attack by more virulent strains of the same virus. More recently, similar reactions have been recorded amongst other main groups of pathogen micro-organisms. Active resistance mechanisms in plants include the hypersensitive response, increases in active oxygen and oxidative enzyme activity, phytoalexin production, cell wall modification and the production of pathogenesis-related proteins (Hammerschmidt and Nicholson, 1999). All of these mechanisms can be elicited by contact with avirulent or lowly virulent pathogens. This induced resistance is often systemic, inducing a resistant phenotype remote from the site of challenge. In addition to induction by necrotrophic pathogens, certain chemicals and avirulent pathogens can induce systemic acquired resistance (SAR). SAR is characterised by the accumulation of salicylic acid and pathogenesis-related (PR) proteins within the plant. SAR has been induced by the exogenous application of salicylic acid indicating that it is primarily a signalling molecule.

The crown gall disease caused by *Agrobacterium radiobacter* p.v. *tumefasciens* can be controlled by inoculating the host first with the closely related non-pathogenic bacterium *A. radiobacter* p.v. *radiobacter*. The underlying mechanism involves the production of bacteriocin by the non-pathogen that is toxic to the pathogen and some other bacteria (Penalver *et al*., 1994). Commercial advantage has been gained from this discovery by the protection of almond seedlings against soil-borne crown gall following inoculation of almond seedlings with the non-pathogen before transplanting from the nursery into the field.

Chemical control

Pesticides and their judicial use are essential to modern agriculture. After their introduction in the 1950s, insecticides were viewed as the universal panacea for all pest problems. However, following the revelations in Rachael Carson's book and the development of resistance to many pesticides, a more rational approach to the use of pesticides has developed. This relies on an understanding of pest population dynamics, the mode of action of the pesticide, application technology, safe use and toxicology (Dent, 2000).

The simplest approach to decision-making in pesticide application is the prophylactic or scheduled use of pesticides. This relies on the tenet that the infestation is always sufficient to cause economic injury. This risk-averse strategy may be justified in the short term when crop value is high or damage thresholds are low. In the longer term, the benefits are outweighed by factors such as pesticide resistance, secondary pest resurgence, destruction of natural enemy populations and over-exposure of farmers to poisonous chemicals. More sophisticated approaches to decision making, lower dose and application rates, better targeting and application technology and greater specificity have all reduced the risks associated with pesticide use. The discovery and development of new agrichemicals follows a series of well-defined steps from discovery through to product registration and launch. Estimates of one in 10,000 to one in 20,000 chemicals are successfully developed for commercialisation.

Insecticides

Insecticides are generally divided into four major classes; organochlorines, organophosphates, carbamates and pyrethroids. There are also a small group of miscellaneous insecticides.

DDT is the best known example of an organochlorine insecticide. Other organochlorines such as aldrin, dieldrin and endosulfan share charcteristics of DDT in that they have a broad spectrum of activity, are persistent and accumulate in the body fat of mammals. Due to the last two factors, the use of DDT like insecticides is considered inappropriate and their use has been banned in most developed countries.

Organophosphate chemicals, which are active against cholinesterase (a respiratory enzyme), were developed as a by-product of nerve gas research during World War II. As a group, they are highly toxic but have a short persistence in the environment. This means that the timing of their application is critical. The systemic insecticides in this group are particularly effective against phloem-feeding insects.

The carbamate insecticides are derivatives of carbamic acid and like the organophosphates have an anticholinesterase activity. They have a broad range of activity and act as contact, stomach and systemic insecticides.

Pyrethrins and pyrethroids are amongst the safest insecticides known due to their low mammalian toxicity. However, they are highly toxic to fish and non-target invertebrates. They have low persistence and have both high contact activity and some repellency (Hammond, 1996).

Insect growth regulators affect metamorphosis, reproduction and larval and nymphal development. As such, they are highly specific to insects. However, as their action is considered slow they are not appropriate as insecticides in all situations. Other unique insecticides are being developed through modification of existing insecticides or the screening of natural products from pathogens of insects.

Fungicides The first fungicides were discovered in the mid- to late-1800s. The range and importance of fungicides available has changed dramatically since this time. To be successful, fungicides must possess certain characteristics. Mainly they should not cause injury to the plant to which they are applied, and must be environmentally safe. Foliar fungicides are exposed to rain and dew and this means that they must be nearly insoluble in water but still soluble enough to function as a fungicide. Tenacity or adhesiveness is another important factor in the efficiency of a fungicide as it must cover the surface well, in order to protect the entire exposed area. Tenacity and spreading ability are often enhanced by the addition of spreaders and stickers. Fungicides fall into two basic groups, protectant fungicides and systemic fungicides.

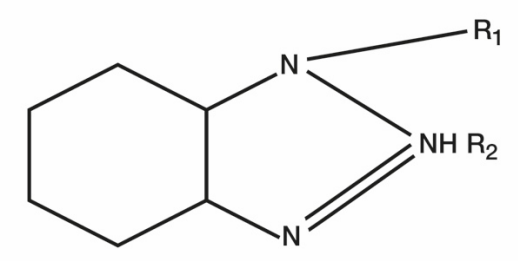
Protectant fungicides form a barrier to infection on the surface of the plant, either preventing germination of spores or killing pathogens on the surface, thus preventing their entry. These fungicides have little effect on pathogens that have gained entry to the host.

Systemic fungicides are absorbed by the plant and so act by preventing growth of pathogens within the host. They are the translocated through the plant, largely in the xylem. As such, their predominant direction of movement is upwards through the plant. The advantages and need for systemic fungicides for plant disease control have been widely recognised. For instance, a systemic fungicide would not only be less subject to weathering but might also translocate to new leaves and shoots, thus requiring less frequent and possibly less precise application. A systemic fungicide should be more effective against internal pathogens. Almost all systemic fungicides bind to specific active sites on enzymes so are prone to resistance build-up in the fungi through mutations which alter the active site of the enzyme.

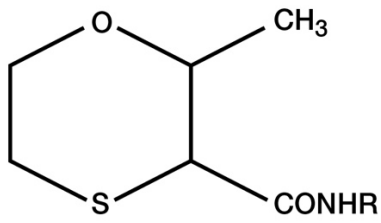
(i) Inorganic fungicides - the most widely used inorganic fungicides contain copper. Bordeaux mixture (copper sulfate and calcium hydroxide) was the first fungicide used against grapevine diseases in France. The copper is the fungitoxic component and calcium hydroxide is used to reduce the phytotoxicity of the chemical. Copper oxychloride is preferred as a fungicide as it can be stored more easily and is less corrosive. Inorganic sulphur compounds, including elemental sulphur and lime-sulphur, are effective fungicides primarily against powdery mildews.

(ii) Organic fungicides - the organic sulphur compounds (dithiocarbamates) are used extensively and are largely protectants. This group includes thiram, maneb and zinib. These fungicides inactivate amino acids and enzymes in the pathogen after being hydrolysed to the isothiocyanate radical.

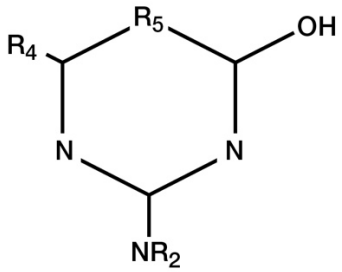
The remainder of the fungicides are systemic in their movement within the plant. Acylanines are a group of fungicides that include metalaxyl and are effective against a range of oomycte fungi. It is often sold under the trade names Ridomil or Apron as a seed dressing. Resistance to this class of fungicide is widely reported (Mukalazi *et al*., 2001; Parra and Ristaino, 2001). The benzimidazole class of fungicides (including benomyl) has the following structure.



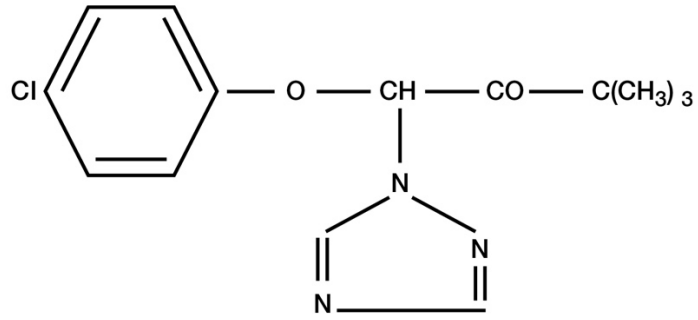
Benomyl is hydrolyzed to methybenzimidazol-2-ylcarbonate in the plant and inhibits fungal mitosis by disassembling microtubules of the mitotic spindle. These fungicides are effective against a range of fungi. Oxathins (including carboxin) have the formula



and interfere with mitochondrial polypeptides. These were the first systemic fungicides discovered (1966). Fungicides containing this active ingredient are effective against damping-off diseases caused by *Rhizoctonia* and some rust and smut fungi. Hydroxypyrimidines inhibit adenosine deaminase in powdery mildews and include fungicides such as ethirimol (Milstem) and diamethirimol (Milcurb). These fungicides have the general formula



Steroid inhibiting fungicides disrupt the biosynthesis of ergosterol. These include prochloraz (an imidazole), fenarimal (a pyramide) and triadimefon (a triazole). Triazoles (Bayleton, Baytan and Tilt) have a protective and curative action against a range of foliar, root and seedling diseases. The formula of triadimefon is shown below.



Fosetyl Al (aluminium tris (ethylphosphorate)) is an unusual fungicide in that it is thought to invoke phytoalexins (natural antifungal compounds) in the host as well as having some phytotoxic action of its own. It is effective against oomycete fungi such as *Phytophthora* spp. and downy mildew fungi. It is also unusual in that it is phloem mobile and so, unlike most fungicides, it can be applied to the foliage and it will be translocated to the roots. It may be applied as a foliar spray, soil drench, root dip or postharvest dip.

Other organic, protectant fungicides include quinones, aromatic compounds and heterocyclic compounds. This last class includes fungicides such as captan, iprodine and vinclozolin. These fungicides may be applied in a foliar formulation or as a seed dressing. In addition to these broad categories of fungicides there are a number of miscellaneous fungicides that are important in specific situations. These include Chloroneb (used as a seed treatment), Triforine (effective against a range of ascomycete fungi) and Propamocarb (effective oomycete fungi and some rusts). Stobulurins are a group of fungicides developed as analogues of compounds produced by wood-rotting fungi and are now widely available. Another group is the so-called defence activators, such as Acibenzolar-S-methyl (CGA 245704 or Actigard 50WG) that induce a systemic resistance in plants in a similar fashion to necrotising plant pathogens (Louws *et al*., 2001).

Antibiotics Antibiotics are sometimes used to control bacterial diseases and diseases caused by phytoplasmas. Antibiotics are absorbed by the plant and translocated. Commonly used antibiotics include streptomycin, tetracyclines and cycloheximide. Drawbacks with the use of antibiotics are their phytotoxic effects and the rapid development of resistance in the disease causing organisms.

Nematicides Most nematicides are volatile soil fumigants. They are injected into the soil where they are effective against a range of organisms including insects, fungi, bacteria and weeds. The four main groups of nematicides are halogenated hydrocarbons, organophosphates, isothiocyanates and carbamates.

Halogenated hydrocarbons are effective nematicides and include methyl bromide. They act by disrupting membranes and the nervous system. These are highly toxic compounds. Methyl bromide is scheduled for worldwide withdrawal from routine use as a fumigant by 2015 under the directive of the Montreal Protocol on ozone-depleting substances. Organophosphates, as well as being insecticides also act as nematicides. These include provate (thimet) and fenamiphos (nemacur). They act by inhibiting cholinesterase resulting in paralysis and death of the nematode. Carbamates also inhibit cholinesterase and may be used to control nematodes (e.g. aldicarb (Temik)). Finally the isothiocyanates (e.g. metam-sodium (vapura)) are also registered as nematicides which release methylisothiocyanate. This compound acts on enzymes.

Resistance to biocides

Within a pest population, a small number of resistant individuals occur. When pesticides are repetitively applied these individuals have a competitive advantage. Through reduced competition,

this group becomes the dominant one in the population. Resistance to biocides has become a common problem especially to the systemic compounds. This occurs because they often have a specific active site on an enzyme for their action. Commonly, resistance arises through:

- decreased permeability of the pest cell membrane;
- detoxification of the chemical;
- decreased conversion to the toxic compound;
- decreased affinity at the reactive site of the enzyme;
- by-passing the blocked reaction; or
- compensation for the effect of inhibition.

Resistance can be managed however, through the rotation of chemical groups, alternation of protectant and systemic biocides, the tank mixing of protectants and systemics and the adoption of pesticide resistance management strategies. Pesticide resistance has usually been monitored through the use of bioassays (Moorman *et al.,* 1994). Due to drawbacks in the interpretation and speed of some of these assays (Vaughan *et al.,* 2001), techniques such as DNA and enzyme assays are becoming more widely used (Luck *et al.,* 1994). The most difficult part of the implementation of any resistance management strategy remains the adoption by the growers (Dent, 2000).

Case Study 5: Insecticide resistance in *Aphis gossypii* Glover (Hemitera : Aphididae), a serious threat to Australian cotton (Herron *et al*., 2001)

Insecticide resistance in cotton aphids (*A.gossypii*) has been reported from Hawaii (Hollingsworth *et al*., 1994), the southern USA (Kerns and Gaylor, 1992; O'Brien *et al.,* 1992), China (Guilin *et al.,* 1997) and Australia (Herron *et al.,* 2000). In Australian cotton production, aphid control occurs late in the season through specifically targeted insecticide applications or by coincidental control arising from insecticide applications to control other insects. The introduction and adoption of transgenic cotton expressing the *Coy a Ac* gene has reduced the need for early season insecticide applications and so has contributed to late season aphid population increases.

Aphids collected Australia-wide, were subjected to bio-assays using seven commonly applied insecticides. Those data were used to calculate the LC50 (lethal concentration to kill 50% of the test population) and LC99.9.

Herron *et al.* (2001) found negligible levels of endosulfan resistance in *A.gossypii*. This could be attributed to the *Helicoverpa amigera* resistance management strategy (Shaw 1999). However, in the longer term, this strategy may contribute to insecticide resistance as more targeted sprays are applied. Highly resistant aphids from Emerald, Queensland, could only be controlled using drafinthiuron and field failures using perimicarb were reported from the same region. This led to the introduction of an *A.gossypii* resistance management strategy.

The main threats to the resistance management strategy are posed by resistant aphids moving from nearby crops, cross resistance due to insensitive anticholinesterase and negligible fitness costs associated with resistance to organophosphates and carbamates (Herron *et al.,* 2001)

Control strategies and forecasting services

A thorough knowledge of the ecology of pathogens is vital when devising strategies for control of diseases. Case Study 6 is an example of utilising knowledge of the ecology of a pathogen in disease control. Forecasting, when a disease is likely to develop, enables growers to apply fungicides at appropriate times.

Case Study 6: Forecasting sclerotinia stem rot of canola

Stem rot of canola is caused by the fungal pathogen *Sclerotinia sclerotorium.* This pathogen has a very wide host range of 408 species in 278 genera (Boland and Hall 1994). The disease is an increasing threat to canola in south-eastern Australia. Until recently, sclerotinia epidemics were thought to occur once in a five to eight year period. However, Australian research has seen epidemics of sclerotinia on canola in New South Wales on an annual basis. This reflects an increase in inoculum pressure from greater reliance on canola in the rotation. In surveys conducted in 1998, 1999 and 2000, levels of stem rot in some crops were found to exceed 30% in all years. This correlates to 15 to 30% yield losses. Estimates of losses due to sclerotinia in 1999 in New South Wales alone exceeded $170 million. Unlike blackleg (caused by *Leptosphaeria maculans*), there are no available sources of resistance to sclerotinia. Therefore control of sclerotinia in Canada and Europe relies on the use of forecasting systems and strategic fungicide applications.

Fungicides applied to flowers of canola have been shown to reduce the level of sclerotinia stem rot in Canada. The level of *S. sclerotorium* present on petals and the risk of favourable weather during petal fall are considered the key determinants in Sclerotinia epidemics. The level of infection in petals can be determined by using a petal testing kit that relies on isolation and identification of the pathogen or immunological tests. Both of these tests may be unreliable if there are spores of other non-pathogenic species of *Sclerotinia* on the petals (Hind *et al.,* 2001). The results from these tests are then combined with meteorological information to produce risk maps that are generated using surface soil moisture content over several days. The models may also incorporate relative humidity values and temperature thresholds conducive to the germination of the sclerotinia spores. The forecast maps show regions where the environmental conditions are favourable for the development of sclerotinia Maps are also available which show growing day degrees for canola and rainfall patterns. When this information on petal infection, risk calculated using weather data and the economics of control are integrated, a decision on fungicide application can be made.

Integrated control

It was largely from ecological considerations that the concept of integrated control was first conceived. Initially the concept combined the use of pesticides and natural enemies in a compatible manner. The modern approach advocates the integration of several control measures in a unified program referred to as integrated control or integrated systems of pest/disease management and control. The concept is defined by the Food and Agriculture Organisation as 'a pest management system that in the context of the associated environment and the population dynamics of the pest species, utilises all suitable techniques and methods in as compatible a manner as possible and maintains the population at levels below those causing economic injury'. The determination of levels of tolerable pest or disease damage is thus an essential prerequisite to the development of integrated control programs. These threshold levels should be determined both in terms of foreseeable crop loss and the economics of crop production and marketing.

In its broadest context integrated control embraces the pest/ disease complex of a specific crop and not just a single organism. The approach demands a thorough understanding of the ecology and dynamics of the 'pest' complex that is usually made up of weeds, insects and diseases. In brief, an understanding of the agro-ecosystem is required. This knowledge is lacking, however, and more research is required before the practice of integrated control can be elevated beyond its present empirical level. The recognition of key pests (those against which control measures are essential if economic production is to be maintained) however, reduces the number of pests of immediate concern. In the meantime, integrated control programs can be based on existing knowledge with the aim of minimising chemical applications where used to control pests/diseases.

PRINCIPLES

- Pests and diseases have plagued crops since they have been established, but we have only recognised the causes in the last few centuries.
- The first step in managing diseases and pests is the correct identification.
- After determining the identity of the pest their numbers must be determined. This can be done through direct counts (e.g. insects or fungal spores) or indirectly through the damage they cause.
- Pest numbers vary in both space and time and so the estimation of their numbers is dependent on the sampling strategy used.
- Pests may reduce yield in three primary ways: by reducing the plant population, diverting nutrients from the produce or destroying the marketable product.
- The relationship between yield loss and pest intensity or severity can be modelled in a number of ways.
- The environment is one of the key driving variables in pest populations. The host acts as an integrator of the effect of the environment and the pest.
- Pest and disease control can be classified as either curative of preventative. Both methods involve the application of the principles of exclusion, avoidance, protection and eradication, applied either as regulatory, physical, cultural, biological or chemical methods of control.

References

Agrios GN (1997) *Plant Pathology.* 4th edition. Academic Press Ltd., San Diego, California

Alstrom S (1991) Induction of disease resistance in common bean susceptible to halo blight bacterial pathogen after seed bacterization with rhizosphere pseudomonads. *Journal of General and Applied Microbiology* **37,** 495-501

Analytis A and Kranz J (1972) Űber die korrelation zwischer befullshäufigkeit and beffassfårke bei pflanzen krankheiten. *Phytopatholgie Zeitschrift.* **73,** 201-207

Anon. (1948) The measurement of potato blight. *Transactions of the British Mycological Society* **31,** 140-141

Arnold, JR (1977) *Rust Epidemic Analysis, Yield and Quality.* M.Sc. Thesis, University of New England, Armidale, Australia. 217 pp

Ash GJ (1990) *Stripe Rust in Northern New South Wales, Australia.* PhD Thesis, University of New England, Australia. 299pp

Ash GJ (2002) Biopesticides. In *Encyclopedia of Pest Management.* D. Pimentel (ed.) Marcel Dekker, NY pp85-88

Ash GJ and Brown JF (1990) Yield losses in wheat caused by stripe rust (*Puccinia striiformis* West) in northern New South Wales. *Australian Journal of Experimental Agriculture* **30,** 103-108

Ash GJ and Brown JF (1991) Effect of nitrogen nutrition of the host on the epidemiology of *Puccinia striiformis* f.sp. tritici and crop yield in wheat. *Australasian Plant Pathology* **20,** 108-114

Ash GJ and Lanoiselet VM (2001a) First report of *Colletotrichum acutatum* causing a leaf spot and hull rot of pistachio. *Australasian Plant Pathology* **30,** 365-366

Ash GJ and Lanoiselet VM (2001b) First report of *Alternaria alternata* causing late blight of pistachio (*Pistacia vera*) in Australia. *Plant Pathology* **50,** 803

Ash GJ and Rees RG (1994) Effect of post-inoculation temperature and light intensity on expression of resistance to stripe rust in some Australian wheat cultivars. *Australian Journal of Agricultural Research* **45,** 1379-1386

Ash GJ, Brown JS and Rees RG (1991) Prediction of severity of stripe rust on wheat in Australia using regional weather data. *Plant Protection Quarterly* **6,** 43-44

Attaran A, Roberts DR, Curtis CF and Kilama WL (2000) Balancing risks on the backs of the poor. *Nature Medicine* **6**, 729

Bailey KL, Johnston AM, Kutcher HR, Gossen, BD and Morrall RAA (2000) Managing crop losses from foliar diseases with fungicides, rotation, and tillage in the Saskatchewan Parkland. *Canadian Journal of Plant Science* **80***,* 169-175

Baker R (1971) Analysis involving inoculum density of soil-borne pathogens in epidemiology. *Phytopathology* **61,** 1280-1292

Bateman GL, Ward E, Hornby D and Gutteridge, R.J. (1997). Comparisons of isolates of the take-all fungus, *Gaeumannomyces graminis* var. *tritici* from different cereal sequences using DNA probes and non-molecular methods. *Soil, Biology and Biochemistry* **29,** 1225-1232.

Beck SD (1965) Resistance of plants to insects. *Annual Review of Entomology.* **10**, 207-232

Biffen RH (1905) Mendel's laws of inheritance and wheat breeding. *Journal of Agricultural Science* **1,** 4-48

Bishop AL, McKenzie HJ, Barchia IM and Spohr LJ (2000) Moon phase and other factors affecting light-trap catches of *Culicoides brevitaris* Kieffer (Diptera : Ceratopogonidae). *Australian Journal of Entomology* **39,** 29-32

Black SA (1965) Resistance of plants to insects. *Annual Review of Entomology* **10**, 207-232

Boland GJ and Hall R (1994) Index of plant hosts of *Sclerotinia sclerotorium*. *Canadian Journal of Plant Pathology-Revue Canadienne de Phytopathologie* **16**, 93-108

Brock RD (1977) Genetic engineering and plant improvement. *Journal of the Australian Institute of Agricultural Science* **43**, 14-21

Brown JF (1975) Diseases of wheat - their incidence and control. In Lazenby, A. and Matheson, E. (eds) *Australian Field Crops, Vol.1. Wheat and Other Temperate Cereals,* Angus and Robertson, Sydney

Brown JF (1997) Airborne inoculum. In J.F. Brown and H.J. Ogle (eds.) *Plant Pathogens and Plant Diseases*. Rockvale Publications, Australia

Brown JS (1988) Relationship between stripe rust severity and grain yield loss in wheat in Dooen, Victoria. *Australian Journal of Agricultural Research* **39,** 563-567

Brown JS and Holmes RJ (1983) Guidelines for use of foliar sprays to control stripe rust of wheat in Australia. *Plant Disease* **67**, 485-487

Brown JS, Smith E, Bell CJ and Price TV (1986) Duration of control of stripe rust of wheat with foliar sprays. *Australasian Plant Pathology* **15**, 9-10

Buchenau GW (1975) Relationship between yield loss and area under the wheat stem rust and leaf rust progress curves. *Phytopathology* **65**, 1317-1318

Burleigh JR, Eversmeyer MG and Roelfs AP (1972) Development of linear equations for predicting wheat leaf rust. *Phytopathology* **62**, 947-953

Calpouzos L, Roelfs AP, Madson ME, Martin FB, Welsh JR and Wilcoxson RD (1976) A new model to measure yield losses caused by stem rust in spring wheat. *University of Minnesota Agricultural Experimental Station Technical Bulletin*

Campbell CL and Noe JP (1985) The spatial analysis of soil borne pathogens and root diseases. *Annual Review of Phytopathology* **23**, 127-148

Campbell JB (1996) *Introduction to remote sensing.* 2nd edn. Guildford: New York, USA

Carlson GA and Main CE (1976) Economics of disease-loss management. *Annual Review of Phytopathology* **14**, 381-403

Carter GA and Miller RL (1994) Early detection of plant stress by digital imaging within narrow stress-sensitive wavebands. *Remote Sensing Environment* **50,** 295-302

Cavelier M and Couvreur L (1995) Effect of eyespot control on the yield of winter wheat and eyespot damage in Belgium. *Agronomie* **15***,* 139-151

Chester KS (1950) Plant disease losses: Their appraisal and interpretation. *Plant Disease Reporter Supplement* **193**, 189-362

Coaker TH (1987) Cultural methods: the crop. *In* Burn, A.J., Coaker, T.H. and Jepson, P.C. (eds) *Integrated Pest Management*. Academic Press, London, pp 69-88

Cobb NA (1892) Contribution to an economic knowledge of the Australian rusts (Urediniae). *Agricultural Gazette of New South Wales* **3**, 60-65

Cole MJ and Gaunt RE (1984) A sequential sampling plan for stripe rust management in winter wheat. *Proceedings of 37th New Zealand Weed and Pest Control Conference*, 294-299

Constable G (1998) Biotechnology risks and benefits: the Ingard cotton example. *Proceedings 9th Australian Agronomy Conference*, Wagga Wagga, 92-98

Cook RJ (1994) Problems and progress in biological control of wheat take-all. *Plant Pathology* **43**, 429-437

Cother EJ and Nichol H (1999) Susceptibility of Australian rice cultivars to the stem rot fungus, (*Sclerotium oryzai*). *Australasian Plant Pathology* **28**, 85-91

Crump NS, Cother EJ and Ash GJ (1999) Clarifying the nomenclature in microbial weed control. *Biocontrol Science and Technology* **9,** 89-97

Darwinkel A (1980) Grain production of winter wheat in relation to nitrogen and diseases. 1. Relationship between nitrogen dressing and yellow rust infection. *Zeitschrift für Acker-und Pflanzenbau* **149**, 229-308

Delanney X, Lavallee BJ, Prokscl, RK, Fuchs RL, Sims SR, Greenplate JT, Marrone PG, Dodson RB, Augustine JJ, Layton JG and Fischhoff DA (1989) Field performance of transgenic tomato plants expressing the *Bacillus thuringiensis* var. *kurstaki* insect control protein. *Bio/Technology* **7,** 1265-1269

Dent, D (2000) *Insect Pest Management.* 2nd ed. CABI Publishing, Cambridge

Doling DA and Doodson JK (1968) The effect of yellow rust on the yield of spring and winter wheat. *Transactions of the British Mycological Society* **51**, 427-434

Eastwood RF, Lagudah ES, Appels R, Hannah M and Kollmorgen JF (1991) *Triticum tauschii*: a novel source of resistance to cereal cyst nematode (*Heterodera avenae*). *Australian Journal of Agricultural Research* **42**, 69-77

Everitt JH and Nixon PR (1986) Canopy reflectance of two drought-stress shrubs. *Photogrammetric Engineering and Remote Sensing* **52**, 1189-1192

Eyal Z and Ziv O (1974) The relationship between epidemics of septoria leaf blotch and yield losses in spring wheat *Phytopathology* **64**, 1385-1389

Fitchen JH and Beachy RN (1993). Genetically engineered protection against viruses in transgenic plants. *Annual Review of Microbiology* **47**, 739-763

Flor HH (1956) The complementary genetic systems in flax and flax rust. *Advances in Genetic.* **8**, 29-54

Flor HH (1971) Current status of the gene-for-gene concept. *Annual Review of Phytopathology* **9**, 275-296

Foley DH (1981) Pupal development rate of *Heliothus armigera* under constant and fluctuating temperatures. *Journal of Australian Entomological Society* **20**, 13-20

Fouché PS and Booysen N (1996) Estimating stress conditions in fruit crops by low altitude infrared remote sensing. *Second International Airborne Remote Sensing Conference and Exhibition, San Francisco, California,* III. 489-491

Garrett SD (1965) Toward biological control of soil-borne plant pathogens. In Baker, K.F. and Synder WC (eds) *Ecology of Soil-Borne Plant Pathogens: Prelude to Biological Control*. University of California Press, Berkeley, 4-17

Gash AF, Carter N and Bale JS (1996) The influence of nitrogen fertiliser applications on the cereal aphids *Metopolophium dirhodum* and *Sitobion avenae. Brighton Crop Protection Conference: Pests and Diseases.* Volume 1. British Crop Protection Council, Farnham, UK, pp 209-214

Gatehouse AMR, Brown DP, Wilkinson HS, Down RE, Ford L, Gatehouse JA, Bell HA and Edwards JP (1998) The use of transgenic plants for the control of insect pests. *In* Kerry BR (ed) *British Crop Protection Council Symposium Proceedings No. 71. Biotechnology in Crop Protection: Facts and Fallacies.* British Crop Protection Council, Farnham, U.K. pp. 25-33

Goodwin PH, Hsiang T, Xue BG and Lui HW (1995) Differentiation of *Gaemannomyces graminis* from other turf-grass fungi by amplification with primers from ribosomal internal transcribed spacers. *Plant Pathology* **44**, 384-391

Guilin C, Runzxi L, Mingjiang H, Kefui X and Shiju J (1997) Comparison of the cotton aphid resistance level between Xinjiang and Shandong populations. *Resistance Pest Management Newsletter* **9,** 10-12

Hallahan DL, Pickett JA, Wadham LJ, Wallsgrove RM and Woodcock CM (1992) Potential of secondary metabolites in genetic engineering of crops for resistance. In Gatehouse AMR, Hilder VA and Boulter D (eds.) *Plant Genetic Engineering for Crop Protection.* CABI, Wallingford, U.K. pp. 215-247

Hammerschmidt R and Nicholson RL (1999) A survey of plant defense responses to pathogens. In *Induced Plant Defenses Against Pathogens and Herbivores: Biochemistry, Ecology and Agriculture.* Agrawal AA, Tuzun S and Bent E (eds) APS Press St Paul Minnesota

Hammond RB (1996) Residual activity of Lambda-cyhalothrin against bean leaf beetle (Coleoptera:Chrysomelidae) in soybeans. *Crop Protection* **13**, 365-373

Hashioka Y (1965) Effects of environmental factors on development of causal fungus, infection, disease development, and epidemiology in rice blast disease. In *The Rice Blast Disease*, 153-161. (J. Hopkins Press: Baltimore, USA)

Hemmi T and Imura J (1939) On the relation of air humidity to conidial formation in the rice blast fungus, *Piricularia orzae,* and characteristics in the germination of conidia produced by the strains showing different pathogenicity. *Annals of the Phytopathological Society of Japan* **9**, 147-156

Herdina and Roget DK (2000) Prediction of take-all disease risk in field soils using a rapid and quantitative DNA soil assay. *Plant and Soil* **227***,* 87-98

Herdina, Yang HA and Ophelkeller K (1997) Correlation of take-all disease severity and inoculum level of *Gaeumannomyces graminis* var. *Tritici* using a slot-blot hybridization assay. *Mycological Research* **101**, 1311-1317

Herron G, Powis K and Rophail J (2000) Baseline studies and preliminary resistance survey of Australian cotton aphid *Aphis gossypii* Glover (Hemiptera: Aphidadae). *Australian Journal of Entomology* **39**, 33-38

Herron GA, Powis K and Rophail J (2001) Insecticide resistance in *Aphis gossypii* Glover (Hemiptera: Aphidadae) a serious threat to Australian cotton. *Australian Journal of Entomology* **40**, 85-91

Hind TL, Ash GJ and Murray GM (2001) *Sclerotinia minor* on canola petals in New South Wales – a possible airborne mode of infection by ascospores? *Australasian Plant Pathology* **30**, 289-290

Hollingsworth RG, Tabashnik BE, Ullman DE, Johnson MW and Messing R (1994) Resistance of *Aphis gossypii* (Hemoptera: Aphidadae) to insecticides in Hawaii: Spatial patterns and relation to insecticide use. *Journal of Economic Entomology* **87**, 293-300

Horsfall JG and Barratt RW (1945) An improved grading system for measuring plant disease. *Phytopathology* **35**, 655

Hugh-Jones M, Barre N and Nelson G (1992) Landsat-TM identification of *Amblyomma variegatum* (Acari: Ixodidae) habitats in Guadeloupe*. Remote Sensing of Environment* **40**, 43-55

Huisman MJ, Cornelissen BJC and Jongedijk E (1992) Transgenic potato plants resistant to viruses. *Euphytica* **63,** 87-197

Irwin ME (1999) Implications of movement in developing and deploying integrated pest management strategies. *Agricultural and Forest Meteorology* **97**, 235-248

James WC (1971) An illustrated series of assessment keys for plant disease, their preparation and usage. *Canadian Plant Disease Survey* **51**, 39-65

James WC (1974) Assessment of plant disease and losses. *Annual Review of Phytopathology* **12**, 27-48

James WC and Shih CS (1973) Relationship between incidence and severity of powdery mildew and leaf rust on winter wheat. *Phytopathology* **63,** 183-187

James WC, Jenkins JEE and Jemmett JL (1968) The relationship between leaf blotch caused by *Rhynchosporium secalis* and losses in grain yield of spring barley. *Annals of Applied Biology* **62**, 273-288

James WC, Shih CS, Hodgson WA and Callbeck LC (1972) The quantitative relationship between late blight of potato and loss in tuber yield. *Phytopathology* **62***, 92-96*

Jeger MJ, Vandenbosch F, Madden LV and Holt J (1998) A model for analysing plant-virus transmission characteristics and epidemic development. *Journal of Mathematics Applied in Medicine and Biology* **15**, 1-18

Jensen JR (1996) *Introductory digital image processing - A remote sensing perspective,* 2nd edn. Prentice-Hall: Upper Saddle River, USA

Johnson KB, Johnson SB and Teng PS (1986) Development of a simple potato growth model for use in crop-pest management. *Agricultural Systems* **19**, 189-209

Kapp G (2000) WHO wins reprieve for DDT against malaria. *Lancet* 356, 2076

Keane P and Brown J (1997) Disease management: resistant cultivars. In JF Brown and HJ Ogle (eds.) *Plant Pathogens and Plant Diseases*. Rockvale Publications, Australia

Kerns DL and Gaylor MJ (1992) Insecticide resistance in field populations of cotton aphid (Hemiptera: Aphidadae). *Journal of Economic Entromology* **85**, 1-8

Kerr A and Keane PJ (1997) Prediction of disease outbreaks. In J.F. Brown and H.J. Ogle (eds.) *Plant Pathogens and Plant Diseases*. Rockvale Publications, Australia

Khan TN (1987) Relationship between net blotch (*Drechslera teres*) and losses in grain yield of barley in Western Australia. *Australian Journal of Agricultural Research* **38**, 671-679

Khangura RK and Barbetti MJ (2001) Prevalence of blackleg (*Leptosphaeria maculans*) on canola (*Brassica napus*) in Western Australia. *Australian Journal of Experimental Agriculture* **41***,* 71-80

King JE (1976) Relationship between yield loss and severity of yellow rust recorded on a large number of single stems of winter wheat. *Plant Pathology* **25**, 172-177

Kingsolver CH, Barksdale TH and Marchetti MA (1984) Rice blast epidemiology. *The Pennsylvania State University Agricultural Experiment Bulletin. 853.* 33 pp

Kirkegaard JA, Rebetzke GJ and Richards RA (2001) Inheritance of root glucosinolate content in canola. *Australian Journal of Agricultural Research* **52**, 745-753

Kirkegaard JA, Sarwar M, Wong PTW, Mead A, Howe G and Newell M (2000) Field studies on the biofumigation of take-all by *Brassica* break crops. *Australian Journal of Agricultural Research* **51**, 445-456

Klassen GR, Balzerzak M and DeCock WAM (1996) 55 ribosomal RNA gene spacers as specie-specific probes for eight species of *Pythium. Phytopathology* **86**, 581-587

Kloepper JW, Tuzun S, Lui L and Wei G (1993) Plant growth-promoting rhizobacteria as inducers of systemic disease resistance. In *Pest Management: Biologically Based Technologies,* R.D. Lumsden, and J.L. Vaughn (eds), pp 156-165 Washington DC. American Chemical Society

Knott DR (1961) The inheritance of rust resistance: VI. The transfer of stem rust resistance from *Agropyron elongatum* to common wheat. *Canadian Journal of Plant Science* **41**, 109

Koziel MG, Beland GL, Bowman C, Carozzi NB, Crenshaw R, Crossland L, Dawson J, Desai N, Hill M, Kadwell S, Lauris K, Lewis K, Maddox D, McPherson K, Meghji MR, Merlin E, Rhodes R, Warren GW, Wrights M and Erola ST (1993) Field performance of elite transgenic maize plants expressing an insecticidal protein derived from *Bacillus thuringiensis. Bio/Technology* **11**, 194-200

Kumar R (1984) *Insect Pest Control with Special Reference to African Agriculture.* Edward Arnold, London

Lanoiselet VM, Ash GJ, Cother EJ, Priest MJ and Watson A (2001) First report of *Waitea circinata* causing sheath spot and *Rhizoctonia oryzae-sativa* causing aggregate sheath spot on rice in south-eastern Australia. *Australasian Plant Pathology* **30**, 369-370

Lanoiselet VM, Cother EJ and Ash GJ (2002) CLIMEX and DYMEX simulations of the potential occurrence of the rice blast disease in south eastern Australia. *Australian Plant Pathology* **31**, 1-7

Large EC (1954) Growth stages in cereals. Illustration of the Feekes scale. *Plant Pathology* **3,** 128-129

Large EC (1996) Measuring plant disease. *Annual Review of Phytopathology* **4**, 9-28

Leitch MH and Jenkins PD (1995) Influence of nitrogen on the development of septoria epidemics in winter. *Journal of Agricultural Science* **124***,* 361-368

Llewellyn DJ and Higgins TJ (1998) Biotechnical approaches to crop protection: Novel sources of insect tolerance genes other than *Bt* toxins. Pest Management, Future Challenges, 6th *Australasian Applied Entomological Research Conference,* Univ. of Queensland, Australia pp 223-229

Lorenzen B and Jensen A (1989) Changes in leaf spectral properties induced in barley by cereal powdery mildew. *Remote Sensing of the Environment* **27**, 201-209

Louws FJ, Wilson M, Campbell HL, Cuppels, DA, Jones JB, Shoemaker PB, Sahin F and Miller SA (2001) Field control of bacterial spot and bacterial speck of tomato using a plant activator. *Plant Disease* **85**, 481-488

Luck JE, Gillings MR and Steel CC (1994) Amplification and cloning of a beta-tubulin gene fragment from strains of *Botrytis-cinerea* resistant and sensitive to benzimidazole fungicides. *New Zealand Journal of Crop and Horticultural Science* **22**, 173-179

Madden LV and Hughes G (1999) Sampling for plant disease incidence. *Phytopathology* **89**, 1008-1103

Madden LV (1980) Quantification of disease progression. *Protection Ecology* **2**, 159-176

Main CE (1977) Crop destruction - the raison d'etre of plant pathology. In Horsfall, J.G. and Cowling, E.B. (eds) *Plant Disease an Advanced Treatise. Volume I. How Disease is Managed.* Academic Press Inc., NY pp 55-78

Manners JG (1993) *Principles of Plant Pathology.* Cambridge University Press. Great Britain

McIntosh RA (1976) Genetics of wheat and wheat rusts since Farrer (Farrer Memorial Oration). *Journal of the Australian Institute of Agricultural Science* **42**, 203-216

McLeod RW (1976) Sources of resistance to *Heterodera avenae* Woll. in New South Wales. *Proceedings of the Linnean Society of New South Wales* **100**, 195-201

Melchers LE and Parker JH (1922) Rust resistance in winter wheat varieties. *United States Department of Agriculture Bulletin.* No. 1046, pp 32

Merritt CR (1998) The commercialisation of transgenic crops - the *Bt* experience. In Kerry, BR (ed) *British Crop Protection Council Symposium Proceedings No. 71. Biotechnology in Crop Protection: Facts and Fallacies.* British Crop Protection Council, Farnham, UK pp 79-86

Miller SA, Madden LV and Shmitthenner AF (1997) Distribution of *Phytophthora* spp. in fields determined by immunoassay. *Phytopathology* **87**, 101-107

Moorman GW, Lease RJ and Vali RJ (1994) Bioassay for dicarboximide resistance in *Botrytis cinerea*. *Plant Disease* **78**, 890-891

Moran MS, Inoue Y and Barnes EM (1997) Opportunities and limitations for image-based remote sensing in precision crop management. *Remote Sensing of Environment* **61**, 319-346

Mukalazi J, Adipala E, Sengooba T, Hakiza JJ, Olanya M, and Kidanemariam HM (2001) Metalaxyl resistance, mating type and pathogenicity of *Phytophthora infestans* in Uganda. *Crop Protection* **20**, 379-388

Mundy EJ (1973) The effect of yellow rust and its control on the yield of Joss Cambier winter wheat. *Plant Pathology* **22**, 171-176

Nicol JM, Davies KA, Hancock, TW and Fisher JM (1999) Yield loss caused by *Pratylenchus thornei* on wheat in South Australia. *Journal of Nematology* **31***,* 367-376

Nobbs JM, Liu Q, Hartley D, Handoo Z, Williamson VM, Taylor S, Walker G and Curran J (2001) First record of *Meloidogyne fallax* in Australia. *Australasian Plant Pathology* **30**, 373

O'Brien DJ, Abdel-Aal YA, Ottea JA and Graves JB (1992) Relationship of insecticide resistance to carboxylesterases in *Aphis gossypii* (Hemiptera: Aphidadae) from mid-south cotton. *Journal of Economic Entomology* **85**, 651-657

O'Brien PC and Fischer JM (1974) Behaviour of *Heterodera avenae* in two wheat cultivars. *Proceedings of 1st National Plant Pathology C*onference, Sydney

O'Dell M, Flavell RB and Hollins TW (1992) The classification of isolates of *Gaevmannomyces graminis* from wheat, rye and oats using restriction length polymorphisms in families of repeated DNA sequences. *Plant Pathology* **41**, 554-562

Ogbannaya FC, Seah S, Delibes Al, Jahier J, Lopez-Brana I, Eastwood RF and Lagudah ES (2001a) Molecular-genetic characterisation of a new nematode resistance gene in wheat. *Theoretical and Applied Genetics* **102**, 623-629

Ogbonnaya FC, Subrahamanyan NC, Moullet O, de Majnik J, Eagles HA, Brown JS, Eastwood RF, Kallmorgen J, Appels R and Lagudah ES (2001b) Diagnostic DNA markers for cereal cyst nematode resistance in bread wheat. *Australian Journal of Agricultural Research* **52**, 1367-1374

Ophel-Keller K, Engel B and Heinrich K (1995) Specific detection of *Gaevmannomyces graminis* in soil using polymerase chain reaction. *Mycological Research* **99**, 1385-1390

Ou SH (1985) Blast. In Rice Diseases, 2nd ed. (CAB International: Oxon, UK)

Park RF, Ash GJ and Rees RG (1992) Studies on the effects of temperature on the response of some Australian wheat cultivars to *Puccinia striiformis* f.s.p. *tritici. Mycological Research* **3**, 166-170

Parra G and Ristaino JB (2001) Resistance to mefenoxam and metalaxyl among field isolates of *Phytophthora capsici* causing Phytophthora blight of bell pepper. *Plant Disease* **85**, 1069-1075

Penalver R, Vicedo B, Salcedo CI and Lopez MM (1994) *Agrobacterium radiobacter* strains K84, K1026 and K84 agr(-) produce an antibiotic-like substance, active *in vitro* against A-*tumefaciens* and phytopathogenic *Erwinia* and *Pseudomonas* spp. *Biocontrol Science and Technology* **4***,* 259-267

Peñuelas J, Filella I, Lloret P, Muñoz F and Vilajeliu M (1995) Reflectance assessment of mite effects on apple trees. *International Journal of Remote Sensing* **16**, 2727-2733

Perlack FJ, Deaton RW, Armstrong TA, Fuchs RL, Sims SR, Greenplate JT and Fischhoff DA (1990) Insect resistant cotton plants. *Bio/Technology* **8**, 939-943

Peterson RF, Cambell AB and Hannah AE (1948) A diagrammatic scale for estimating rust intensity on leaves and stems of cereals. *Canadian Journal of Research Section C* **26**, 496-500

Pielou EC (1977) *Mathematical Ecology.* John Wiley and Sons, New York, p 385

Pinkerton JN and Alderman SC (1994) Epidemiology of *Anguina-agrostis* on highland colonial bentgrass. *Journal of Nematology* **26***,* 315-323

Pruess KP (1983) Day-degree methods for pest management. *Environmental Entomology* **12**, 613-619

Punja ZK (2001) Genetic engineering of plants to enhance resistance to fungal pathogens - a review of progress and future prospects *Canadian Journal of Plant Pathology-Revue Canadienne de Phytopathologie* **23***,* 216-23

Raaijmakers JM, Leeman M, Van Oorschot MMP, Van der Sluis I and Schippers B (1995) Dose-response relationships in biological control of fusarium wilt of radish by *Psuedomonas* spp. *Phytopathology* **85**, 1075-1081

Rabbinge R and Rijsdijk FH (1983) EPIPRE: a disease and pest management system for winter wheat, taking account of micrometeorological factors. *European Plant Protection Organisation Bulletin* **13**, 297-305

Rathjen AJ, Eastwood RF, Lewis JG and Dube AJ (1998) Breeding wheat for resistance to *Heterodera avenae* in Southeastern Australia. *Euphytica* **100**, 55-62

Rayner RW (1961) Measurement of fungicidal effects in field trials. *Nature* **90**, 328-330

Roget DK and Rovira AD (1991) The relationship between incidence of infection by take-all fungus (*Gaeumannomyces graminis* var. *tritici*), rainfall and yield of wheat in South Australia. *Australian Journal of Experimental Agriculture* **31**, 509-513

Romig RW and Calpouzos L (1970) The relationship between stem rust and loss in yield of spring wheat. *Phytopathology* **60**, 1801-1805

Romig RW, Calpouzos L and Madson ME (1969) The effect of stem rust on yield-loss in spring wheat. *Phytopathology* **59**, 1047

Rowell JB (1973). Effect of dose, time of application, and variety of wheat on control of leaf rust by 4-n-butyl-2,2,4 triazole. *Plant Disease Reporter* **57**, 653-657

Rowell JB (1985) Evaluation of chemicals for rust control. In Roelfs AP and Bushnell WR (eds) *The Cereal Rusts. Volume II. Diseases, Distribution, Epidemiology and Control.* Academic Press, Inv., Orlando, USA pp 561-589

Schneider RW, Williams RJ and Sinclair JB (1976) Cercospora leaf spot of cowpea. Models for estimating yield losses. *Phytopathology* **66**, 384-388

Schumann GL (1991) *Plant Diseases: Their Biology and Social Impact*. APS Press, St. Paul, Minnesota

Sears EK (1956) The transfer of leaf-rust resistance from *Aegilops umbellata* to wheat. *Brookhaven Symposium of Biology* **9**, 1

Sears ER (1972) Chromosome engineering in wheat. *Stadler Symposia* **4,** 23. University of Missouri, Columbia

Seck M, Roelfs AP and Teng PS (1988) Effect of leaf rust (*Puccinia recondita tritici*) on yield of four isogenic wheat lines. *Crop Protection* **7**, 39-42

Seem RC (1984) Disease incidence and severity relationships. *Annual Review of Phytopathology* **22**, 133-150

Shaw AJ (1999) *Cotton Pest Management Guide,* 99/00, Agdex 151/68. NSW Agriculture, Orange

Slootmaker LAJ and van Essen A (1969) Yield losses in barley caused by mildew attack. *Netherlands Journal of Agricultural Science* **17**, 279-282

Southwood TRE (1966) *Ecological Methods.* Chapman and Hall, London p 524

Southwood TRE (1978) *Ecological Methods, with Particular Reference to the Study of Insect Populations*. 2nd ed. Chapman & Hall, London

Stern VM, Smith RF, van den Bosch R and Hagen KS (1959) The integrated control concept. *Hilgardia* **29**, 81-101

Stinner BR and Howe GJ (1990) Arthropods and other invertebrates in conservation-tillage agriculture. *Annual Review of Entomology* **35**, 299-318

Stynes BA and Veitch LG (1981) A synoptic approach for crop loss assessment used to study wheat. IV. The description of cultural practices and the combination of cultural data with soil data. *Australian Journal of Agricultural Research* **32**, 1-8

Stynes BA Wallace HR and Veitch LG (1979) A synoptic approach for crop loss assessment used to study wheat. I. An appraisal of the physical and chemical soil properties in the study area. *Australian Journal of Soil Research* **17**, 217-225

Stynes BA, Veitch LG and Wallace HR (1981) A synoptic approach for crop loss assessment used to study wheat. V. Crop growth and yield. *Australian Journal of Agricultural Research* **32**, 9-19

Suzuki H (1975) Meteorological factors in the epidemiology of rice blast. *Annual Review of Phytopathology* **13**, 239-256

Taylor LR (1961) Aggregation, variance and the mean. *Nature* **189**, 732-735

Teng, PS (1979) Modeling Disease - Loss Systems in Cereals. *Proceedings of Symposia IX International Congress of Plant Protection Washington, DC, USA* pp 122-127

Teng PS and Gaunt RE (1980) Modelling systems of disease and yield loss in cereals. *Agricultural Systems* **6**, 131-154

Teng PS, Close RC and Blackie, MJ (1980) A comparison of models for estimating yield loss caused by leaf rust (*Puccinia hordei*) on Zephyr barley in New Zealand. *Phytopathology* **69**, 1239-1244

Thorton PK and Dent, JB (1984) An information system for the control of *Puccinia hordei. Agricultural Systems* **15**, 225-243

Toler, R.W., Smith, B.D. and Harlan, J.C. (1981) Use of aerial colour infrared photography to evaluate crop disease. *Plant Disease* **65,** 24-31

Turechek WW, Ellis MA and Madden LV (2001) Sequential sampling for incidence of Phomopsis leaf blight of strawberry. *Phytopathology* **91**, 336-347

Vaeck M, Reynaerts A, Hofte H, Jansens S, DeBeuckeleer M, Dean C, Zabeau M, Montagu M, van and Leemans J (1987). Transgenic plants protected from insect attack. *Nature* **328**, 33-37

Van der Plank, JE (1963) *Plant Diseases: Epidemics and Control.* Academic Press, London

Van der Plank, JE (1968) *Disease Resistance in Plants.* Academic Press, London

van Loon LC, Bakker PAHM and Pieterse CMJ (1998) Systemic resistance induced by rhizosphere bacteria. *Annual Review of Phytopathology* **36**, 453-483

Van Peer R, Niemann GJ and Schippers B (1991) Induced resistance and phytoalexin accumulation in biological control of Fusarium wilt of carnation by *Psuedomonas* sp. strain WCS417r. *Phytopathology* **81**, 728-734

Vaughan SJ, Steel CC and Ash GJ (2001) Apparent degradation of pyrimethanil by *Botrytis cinerea* and other fungi on agar plates is caused by migration of the fungicide within the agar medium. *Australasian Plant Pathology* **30**, 367-368

Veitch LG and Stynes BA (1979) A synoptic approach for crop loss assessment used to study wheat. II. Relationships between soil properties and traditional soil classifications. *Australian Journal of Soil Research* **17**, 227-236

Veitch LG and Stynes BA (1981) A synoptic approach for crop loss assessment used to study wheat. III. Variables derived from soil properties and traditional soil classifications suitable for regression studies. *Australian Journal of Soil Research* **19**, 13-21

Vogelmann JE and Rock BN (1989) Use of thematic mapper data for the detection of forest damage caused by the pear thrips. *Remote Sensing of Environment* **30**, 217-225

Warton B, Matthiessen JN and Shackleton MA (2001) Glucosinolate content and isothiocyanate evolution - Two measures of the biofumigation potential of plants. *Journal of Agricultural and Food Chemistry* **49,** 5244-5250

Washington WS and Pascoe I (2000) First record of *Monilinia fructicola* on strawberry fruit in Victoria, Australia. *Australasian Plant Pathology* **29***, 70*

Webb DH and Nutter FW (1997) Effects of leaf wetness duration and temperature on infection efficiency, latent period, and rate of pustule appearance of rust in alfalfa. *Phytopathology* **87***,* 946-950

Wei G, Kloepper JW and Tuzun S (1996) Induced systemic resistance to cucumber diseases and increased plant growth by plant-growth promoting rhizobacteria under field conditions. *Phytopathology* **86**, 221-224

Wei G, Kloeppper JW and Tuzun S (1991) Induction of systemic resistance of cucumber to *Colletotrichum orbiculare* by select strains of plant growth-promoting rhizobacteria. *Phytopathology* **81,** 1508-1512

Wellings CR, Wong PTW and Murray GM (1985) Use of multiple regression to examine the effect of leaf rust and yellow spot on yield of wheat in northern New South Wales. *Australasian Plant Pathology* **14**, 62-64

West JS, Kharbanda PD, Barbetti MJ and Fitt BDL (2001) Epidemiology and management of *Leptosphaeria maculans* (phoma stem canker) on oilseed rape in Australia, Canada and Europe. *Plant Pathology* **50***,* 10-27

Whitney PJ (1976) *Microbial Plant Pathology.* Hutchinson, London

Williams DG and McDonald G (1982) The duration and number of immature stages of codling moth, *Cydia pomonella*. (L) (Tortricidae: Lepidoptera). *Journal of Australian Entomology* **21**, 1-4

Wilson, AGL, Hughes, RD and Gilbert H (1972 Response of cotton to pest attack. *Bulletin of Entomological Research* **61**, 405-414

Wratten SD, Lee G and Stevens DJ (1979) Duration of cereal aphid populations and their effects on wheat yield and quality. *Proceedings 1979 British Crop Protection Conference - Pests and Disease.* British Crop Protection Council, Farnham, UK, pp 1-8

Zadoks JC, Chang TT and Konzak CF (1974) A decimal code for the growth stages of cereals. *Weed Research* **14**, 415-421