

Horizontal or Generalized Resistance to Pathogens in Plants

P.J. Keane

*Department of Botany, La Trobe University, Victoria
Australia*

1. Introduction

The threat to world wheat production and the panic among the agricultural science community caused by the emergence of the 'super virulent' wheat stem rust (*Puccinia graminis tritici*) race Ug99 in East Africa (Singh et al., 2011) is a reminder that the name and ideas of the South African J.E. van der Plank should not be forgotten. Based on his long experience with resistance to *Phytophthora infestans* in potatoes, he developed in his seminal books, *Plant Diseases: Epidemics and Control* (1963) and *Disease Resistance in Plants* (1968), the quantitative study of disease epidemics and the associated concepts of 'vertical' and 'horizontal' resistance to emphasize the two contrasting types of resistance to disease in crops. He contended that our preoccupation through the 20th Century with the more scientifically fascinating and precise vertical resistance, controlled by identifiable genes with a major effect, had resulted in the unfortunate neglect of the more mundane and nebulous horizontal resistance, mostly inherited quantitatively, even though it is evident that the former is unstable in the field while the latter is more stable and consistently useful. The saga of the scientific study of disease resistance shows our human tendency to dig where the light shines brightest, not where we know the potatoes are buried. This tendency continues unabated with the preoccupation of molecular biologists with vertical resistance, often discussed currently as if it is the only form of resistance.

Before their domestication, plants co-evolved with their parasites and underwent natural selection for resistance to them. Since the dawn of agriculture, plants with a degree of resistance to pathogens and insect pests have been selected by farmers, either consciously or unconsciously. In the genetically diverse crops of early agriculture, when plants of particular species began to be crowded together and so made more vulnerable to pest and disease attack, plants with great susceptibility would have been selected against in competition with more resistant types. They would have contributed fewer offspring to the next generation. Traditional farmers would have learned very early that it was better to select seed or vegetative propagating material from the healthiest plants and they still do so today. It is highly likely that they were selecting for partial or quantitative (van der Plank's 'horizontal') resistance. They weren't selecting for resistance to particular pests or diseases but rather for general plant health. They selected for pest and disease resistance as they selected for higher yield and other quantitatively inherited traits such as size and quality of the harvested product and adaptation to the environment. This process has been replicated

by Simmonds (1964, 1966) who re-created the domesticated potato common in Europe (*Solanum tuberosum* ssp. *tuberosum*) by repeated mass selection of true seed of the best types from the highly variable wild Andian potato (*S. tuberosum* ssp. *andigena*). The potatoes were exposed to late blight epidemics during the selection process, and it was shown that the selected materials were more resistant to the disease than the standard domesticated potato (Thurston, 1971).

Through the 1800s there were reports of wheat farmers noticing in their fields occasional 'off-types' with complete resistance or immunity (probably 'vertical resistance' in van der Plank's terms) to a prevalent disease, although little practical use was made of this until it was understood that it was inherited (Biffen, 1905, p.40). It would have been possible to spot these completely resistant types and to distinguish them from 'escapes' only when disease levels were very high and practically all plants in a crop were heavily diseased. In the late 1800s, plants that appeared to be resistant in the field were selected, multiplied and promoted for use on a wider scale. A farmer in South Australia, James Ward, planted a South African variety called De Toit and noticed that it was generally "as rusty as a horse nail" except for a few plants that were rust-free (Callaghan & Millington, 1956) and were later thought to have arisen from a contaminant (Farrer, 1898). Ward saved the seed of these plants and increased it to produce a commercial variety named "Ward's Prolific". Because of its rust resistance and other characters, this became the most widely grown wheat in South Australia at the time. Rees et al. (1979) showed that in the 1970s this variety had little horizontal resistance and so it is likely that Ward had selected a type of extreme resistance that has since been overcome by the pathogen. Another South Australian farmer, Daniel Leak, noticed an occasional rust-free plant in a crop of heavily rusted Tuscan wheat, and selected and multiplied this as a commercial variety called 'Leak's Rust-proof' (Williams, 1991). There were many similar attempts at selecting rust resistant wheat, resulting in other varieties like 'Anderson's Rust-proof' and 'Kalm's Rust-proof' for which rust resistance was claimed (Cobb 1890). Farmers were developing a general appreciation of inherited variation in disease resistance. The Australian wheat breeder, William Farrer (1898), was one of the first to declare that resistance to wheat stem rust was inherited (Biffen, 1905). With the discovery of Mendel's work on the genetics of particular traits of the garden pea, his methods were soon applied to the trait of extreme disease resistance that had been observed in certain crops. R.H. Biffen, working at Cambridge University with resistance to stripe rust (*Puccinia striiformis*) in wheat, was the first to take up these studies, beginning in 1902 and summarizing his findings in papers entitled 'Mendel's laws of inheritance and wheat breeding' (Biffen, 1905) and 'Studies in the inheritance of disease resistance' (Biffen, 1907). He showed that a high level of disease resistance or immunity was inherited as a simple Mendelian character, and so could be crossed readily into well adapted varieties. Thus began our enchantment with the use of 'resistance genes' to protect our crops from disease and the search for them in crop varieties and in the centres of evolution and diversification of the crops (Vavilov, 1951).

Potato late blight caused by *Ph. infestans* has been central to modern plant pathology since the catastrophic epidemics in 1845-47 in Western Europe and Ireland that triggered the terrible Irish famine of the period and led to our understanding that a fungus could invade and cause disease in a healthy plant. While no completely resistant 'off-types' were noticed amongst the heavily blighted potato crops of the 1800s, a wild *Solanum* species collected

from Mexico was shown to be immune to late blight (Salaman, 1910). In breeding experiments, individuals of this species stood out as completely healthy amongst genotypes of the cultivated potato that were severely diseased. This stimulated collecting expeditions to Central America to discover sources of resistance in wild relatives of the cultivated potato. *Solanum demissum*, a wild potato species from Mexico, was shown to have several resistance (R) genes that conferred immunity to late blight. This immunity was evident as a hypersensitive necrotic response in the leaf tissues invaded by the pathogen. At the time, it was considered to be the solution to the late blight problem in Europe and North America (Reddick, 1934). These R genes were cross-bred into the domesticated potato (*S. tuberosum* ssp. *tuberosum*) that formerly had no identifiable genes for immunity to late blight. However, with their widespread use in the field these varieties soon succumbed completely to the disease (Thurston, 1971). They were immune to some isolates (races) of the fungus but were completely susceptible to other races that increased in the pathogen population in response to the selective pressure resulting from the widespread use of a particular resistant variety. It was said that their resistance “broke down” with the selection of ‘virulent’ races of the pathogen that could invade the varieties with R genes. Van der Plank (1963) said that these varieties showed ‘vertical resistance’, named after the extreme vertical differences evident in the graphic plot of the degree of resistance (or, conversely, the amount of disease) on the Y-axis against a series of races of the pathogen along the X-axis (Figure 1). The sharp contrast between varieties with high levels of disease and those with very low levels and often no disease at all (immunity), shown by the abrupt vertical jumps in van der Plank’s bar graphs, allowed identification of Mendelian genes with strong effects on resistance; hence the genes are often referred to as ‘major resistance genes’ or just ‘resistance genes’. Varieties with different resistance genes gave completely different plots of resistance (or amount of disease) against races (Figure 1).

Varieties lacking R genes, or whose R genes were matched by the virulence of the prevailing pathogen races, gave more-or-less similar amounts of disease when inoculated with different races. The amount of disease could be high or low. Van der Plank said that varieties showing low levels of disease had ‘horizontal resistance’, the plot of degree of resistance or amount of disease against a series of races being more-or-less horizontal, or at least not showing extreme variation from complete resistance to great susceptibility evident with vertical resistance (Figure 1; van der Plank, 1963, Figs. 14.1, 14.2). The plot for two varieties may be displaced up or down, but the more resistant variety is more resistant to all races. The graph may not be completely horizontal; it may show some up and down displacements depending on the relative ‘aggressiveness’ of the races, but the displacements are the same for different varieties. The fundamental difference between the two types of resistance is that vertical resistance in the host varieties shows a sharp differential interaction (a strong statistical interaction) with the pathogen races; i.e. the amount of vertical resistance is specific for a particular race (very high for one race in Figure 1, very low for the other). It is ‘race-specific’. Horizontal resistance is not race-specific to the extreme degree evident in vertical resistance (Figure 1). Because it is race-specific, the effect of vertical resistance is prone to being lost rapidly due to selection of virulent races in the pathogen population. Lacking this sharp interaction, horizontal resistance tends to be more stable, more ‘durable’. That is its big advantage. Researchers working with late blight resistance in potato concluded that “R-gene hypersensitivity cannot be relied upon as a permanent protection against *Phytophthora infestans* and so the necessity of providing a

degree of field protection in new cultivars is generally recognised by potato breeders.”(Malcolmson, 1976). The ‘field protection’ referred to here is horizontal resistance.

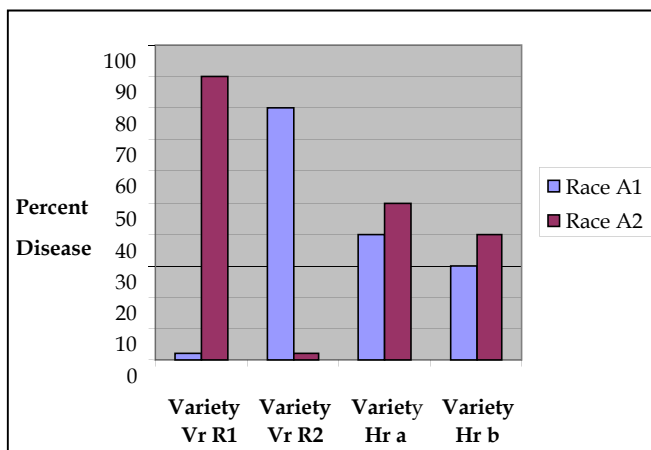


Fig. 1. Plot of percent disease in two varieties with vertical resistance (Vr R1 with resistance gene R1; Vr R2 with resistance gene R2) and two varieties lacking vertical resistance (R genes) but expressing some horizontal resistance (Hr a, Hr b) infected with two pathogen races (A1 with avirulence against R1; A2 with avirulence against R2). Vertical resistance shows a strong interaction with the races (i.e. is ‘race-specific’). Horizontal resistance does not (i.e. is ‘non-race-specific’) although it shows significant main effects of Variety (variety Hr a is more susceptible to both races than Hr b) and Race (race A2 is more aggressive than A1 on both varieties, and this is also evident in the vertically resistant varieties where the R gene is ineffective).

Horizontal resistance has had a much longer history in human knowledge than vertical resistance and has had a greater profusion of names. Once, probably all observed resistance was of this type. It is probably the resistance that keeps ‘minor pathogens’ consistently ‘minor’ and consequently is not much studied for these pathogens because they are of minor importance. For the ‘major’ pathogens, farmers would often recognize that certain varieties ‘got less disease’ than other varieties. For particular diseases, ‘slow-rusting’, ‘slow-mildewing’ (for powdery mildews), ‘slow-blighting’ (for potato late blight) or ‘slow-blasting’ (for rice blast) are older terms that accurately describe horizontal resistance, an essential feature of which is the slowing down of epidemics. In natural plant communities or crops with a diversity of resistance genes (e.g. traditional mixed crops), vertical resistance will also slow down epidemics, but in current monocultures vertical resistance tends to prevent epidemics until such time as it is matched by virulence in a large proportion of the pathogen population. Several terms have been used to distinguish this general resistance from the race-specific resistance discovered in the early 1900s. These have included ‘partial resistance’, ‘quantitative resistance’, ‘generalized resistance’, ‘field resistance’, ‘adult-plant resistance’, ‘durable resistance’, and ‘tolerance’. All have their problems. ‘Race-non-specific resistance’, usefully abbreviated to ‘non-specific resistance’, best captures the essential

nature of this resistance as highlighted by van der Plank, although at a fine level a degree of race-specificity has been shown to apply to it (Parlevliet, 1995). Field resistance was commonly used for potato varieties lacking R genes, but is not a good general term as the resistance of a crop variety in the field could be due to a combination of vertical and horizontal resistance. 'General resistance' can refer to the resistance of a variety to several pests and diseases and again could be the result of both vertical and horizontal resistance. Sometimes 'tolerance' is used for 'horizontal resistance', but this is certainly incorrect. Tolerance has a special meaning; it refers to plant varieties that suffer less damage for a given degree of infection compared with a disease sensitive variety (Caldwell et al., 1958; Schafer, 1971). This has the same meaning as 'rust-enduring' referred to by N.A. Cobb (1894), one of the earliest students of rust resistance. Certainly, horizontal resistance is usually 'partial' and 'quantitative' and can be expressed in a gradient from very little disease to quite a lot, depending on the host genotype, the aggressiveness of the pathogen and the environment. But, for some diseases it can be complete. Thus, the non-prescriptive term, 'horizontal resistance', is perhaps the best. 'Generalized resistance' as applied to potatoes is also apt. Robinson (1976) favoured the terms 'vertical' and 'horizontal' resistance because they were somewhat abstract, and in fact had application beyond disease resistance; e.g. to fungicide use whereby copper-based fungicides could be regarded as 'horizontal' in effect because they knocked out several enzyme systems and the fungi could not adapt to them, while the new, highly specific fungicides like benomyl are 'vertical' because they knock out only one narrow function and the fungi can adapt to them (they 'break down' in the same way that vertical resistance breaks down).

Until it is matched by a virulent race, vertical resistance tends to be complete or to reduce reproduction of the pathogen to a tiny amount. That is its great attraction and that is how it was first noticed (Callaghan & Millington, 1956; Biffen, 1907; Salaman, 1910). In the 1800s it was observed by farmers as stark 'off-types' in crops of rusted wheat. The domesticated wheat species probably had vertical resistance genes against stem rust, while the domesticated potato in Europe did not have vertical resistance genes against late blight until they were bred into it from a wild relative. Horizontal resistance is harder to detect and measure, although it is likely to be selected unconsciously by observant farmers who collect their seed from their healthiest looking plants. It tends to be partial or quantitative in its expression (i.e. there is some disease and some sporulation of the pathogen) but the rate of development of the disease epidemic is reduced compared with that on a susceptible variety under similar environmental conditions. Van der Plank's interest in disease epidemiology (the quantitative study of populations of pathogens and crops) led to his understanding of the importance of partial resistance. There has been a recent shift in thinking about pests and diseases - we no longer talk about their 'control', which implies their elimination from a crop; rather we now talk about pest and disease 'management' which implies acceptance of some level of their presence as long as they cause little economic loss. Under the modern concept of Integrated Pest Management (IPM), moderate levels of pest/disease resistance in a crop may be sufficient if applied synergistically with cultural control methods and minimal, targeted use of pesticides. Under the IPM approach, resistance does not have to be complete; partial resistance may be all that is required, and horizontal resistance becomes important. Modern plant breeders and pathologists talk about "avoiding high degrees of susceptibility". The idea of IPM was promoted initially by entomologists in response to the phenomenon of 'breakdown of insecticide efficacy' that is analogous to the 'breakdown of

disease resistance'. Entomologists have realized the futility of the 'scorched earth' approach to the use of insecticides in an attempt to eliminate pests from crops. They now accept that there is a threshold level of pest infestation below which little economic damage is caused. Entomologists are now emphasizing crop resistance (mostly of a horizontal nature) to insect pests after a long period of total reliance on insecticides (and consequent neglect and decline of resistance to insect pests in crops), while the repeated problem of the breakdown of disease resistance in many crops, exemplified by the emergence of wheat stem rust race Ug99, has led to a re-awakening of plant pathologists to the merits of horizontal resistance after a long period of pre-occupation with vertical resistance. It appears that both entomologists and plant pathologists are emerging from a period of bedazzlement by scientific and technical 'revolutions' (the 'insecticide revolution' and the 'resistance gene revolution') that showed great promise in the laboratory but lost their effectiveness following their widespread use as 'stand-alone' control measures in the farmers' fields. It is now realized that these brilliant technical developments, that offered an illusion of a simple and complete 'revolution' in control of pests and diseases, have to be incorporated into the ecological complexity of crop growth and production in the field. Rather than 'revolutions', a steady evolution of stable IPM methods, based on a foundation of steady evolution of horizontal resistance, is required. Of course, in addition to cultural methods, IPM methods may include targeted use of pesticides and vertical resistance, which, in IPM, are supported by the other methods that prolong and enhance their value. This approach is far from new. In the 1890s, Australia and North America produced vast quantities of very cheap wheat that flooded the European markets. It is obvious that the production was highly successful in most years. The potentially devastating wheat stem rust was managed effectively for long periods by a combination of use of early maturing varieties (that avoided the worst of the epidemics late in the growing season and allowed disease escape), drought adapted varieties (that allowed wheat to be grown in drier climates not conducive to the rust), the use of varieties that had probably been selected unconsciously over the years for a degree of horizontal resistance, and later, the addition of vertical resistance (e.g. in a variety like Thatcher in North America) and the deliberate incorporation of horizontal resistance by cross-breeding with tetraploid wheats.

2. Vertical or specific resistance

Researchers working to develop and deploy stem rust resistance genes (Sr genes) in wheat varieties in North America (Stakman & Levine, 1922) and Australia (Waterhouse, 1936, 1952; Watson, 1958; Watson and Luig, 1963) had exactly the same experience of 'breakdown of resistance' controlled by single resistance genes (Sr genes) as the researchers working with R genes in potato. This is well documented for several countries by Person (1967). The situation was starkest in the spring wheat crops in Australia (Table 1). Eureka, the first commercial wheat variety bred with a vertical resistance gene (Sr6) against stem rust in Australia, was released in 1938 and because of its resistance and other qualities was very popular, increasing to constitute nearly 20% of the wheat area in northern New South Wales by 1945 (Watson, 1958). However, races of stem rust able to attack the variety (i.e. virulent on Sr6) increased from practically nil in 1938 to make up 72% of the rust isolates collected in the area in 1945. The resistance of Eureka was seen to have 'broken down', and the variety rapidly lost popularity. The wheat variety Gabo and some others with resistance gene Sr11 were released in 1942 and by 1950 made up 62% of the same wheat area. But races of stem

rust with virulence on Sr11 increased from nil to 91% by 1950 and the resistance of Gabo was broken. The period of the 'boom and bust' cycle of resistance breeding had begun and the fascinating phenomenon of pathogen-resistance specificity was revealed.

Year	Variety (Resistance gene)	Dominant stem rust race	Disease/Resistance situation in the field
1938	Eureka (Sr6) released	126-Avirulent on Sr6	No disease/Resistance effective
1942		126-Virulent on Sr6	Disease widespread/Resistance broken down
1942	Gabo types (Sr11) released	222-Avirulent on Sr11	No disease/Resistance effective
1948		222-Virulent on Sr11	Disease widespread/Resistance broken down
1950	Festival (Sr9b) released	21-Avirulent on Sr9b	No disease/Resistance effective
1959		21-Virulent on Sr9b	Disease widespread/Resistance broken down
1958	Mengavi (Sr36) released	34-Avirulent on Sr34	No disease/Resistance effective
1960		34-Virulent on Sr34	Disease widespread/Resistance broken down

Table 1. The interaction between the wheat cultivars Eureka, Gabo, Festival and Mengavi and wheat stem rust (*Puccinia graminis tritici*) races in Australia, showing the repeated breakdown of vertical resistance as new races evolved (Watson and Luig, 1963; after Knott, 1989).

Many researchers prefer the term 'race-specific resistance', which accurately describes the statistical interaction which is the essential feature van der Plank sought to highlight in his definition of 'vertical resistance' and is the basis of resistance breakdown. Races that can specifically invade (i.e. are virulent on) varieties with certain resistance genes cause the breakdown of that resistance when they increase to a high proportion of the total rust population in the field. 'Specific resistance' is a neat abbreviation as long as it is understood as 'race-specificity' not 'species-specificity'. The use of the term 'virulence' in plant pathology, where it is used to describe the ability of a pathogen race to invade a plant with a particular resistance gene, is different from its use in medicine, where it is used to describe what in plant pathology would be called the aggressiveness of a pathogen. In plant pathology, the pathogenicity of an organism consists of its virulence (ability to infect varieties with particular vertical resistance genes) and its aggressiveness (the amount of disease it causes on varieties it is able to infect). 'Virulence' is the ability of a pathogen to overcome vertical resistance, while 'aggressiveness' is the ability to overcome horizontal resistance. In much current plant pathology writing, there is a tendency to revert to the medical meaning of 'virulence', which requires the development of another term for the ability of a pathogen to match vertical resistance.

Another set of terms is used to describe the interaction of a specialized pathogen and its host. If a pathogen can infect and sporulate more-or-less normally on a plant, the interaction

is said to be 'compatible'; if the interaction results in hypersensitive necrosis that largely excludes the pathogen, it is said to be 'incompatible', a term used very early in describing the effect of vertical resistance (see Hayes et al., 1925)(Table 2). These terms are particularly apt for biotrophic pathogens such as rusts and powdery mildews, where there is an intimate parasitic symbiosis between the pathogen and its host. In the same sense, the normal interactions of a plant and its mycorrhiza and endophyte symbionts would be said to be 'compatible'. The importance of the 'basic compatibility' required for a symbiont to live in its host has been well explored by Heath (1981). In fact, the copious molecular investigations of incompatibility would be better directed to trying to understand the mechanisms of compatibility - how does a biotrophic symbiont like a rust or a powdery mildew obtain nutrients from its host plant and why can't this feeding relationship be replicated in a Petri dish?

Pathogen	Plant host	
	Resistance gene RR or Rr	Double mutant rr (Susceptible regardless of A/a)
Avirulence gene AA or Aa	Incompatible interaction Hypersensitive necrosis; pathogen does not complete its life-cycle	Compatible interaction Pathogen infects and completes its life cycle
	Horizontal resistance is masked	Horizontal resistance evident
Double mutant aa (Gives virulence against R)	Compatible interaction Pathogen infects and completes its life cycle	Compatible interaction Pathogen infects and completes its life cycle
	Horizontal resistance evident	Horizontal resistance evident

Table 2. Summary of the gene-for-gene interaction involved in vertical resistance. Resistance is usually dominant to susceptibility in the plant. Avirulence is usually dominant to virulence in the pathogen. The only starkly unique interaction occurs when an A gene matches an R gene. Arrow 1 indicates the change induced by breeding an R gene into a plant variety; arrow 2 indicates the change associated with the breakdown of resistance. Note that for wheat stem rust, the R gene is given the symbol Sr, for wheat stripe (yellow) rust it is given the symbol Yr etc.

Our knowledge of vertical resistance has a short history, beginning with the discovery of Mendel's genetics and the subsequent work of R.H. Biffen at Cambridge University in 1902. It has been shown to be expressed almost universally as a hypersensitive necrosis of host cells contacted by the pathogen during the early stages of infection, in some cases occurring rapidly in the first few cells contacted and so evident only under a microscope (infection type 0 in the scheme of Stakman and Levine, 1922, for cereal rusts), and in others occurring only after the pathogen has invaded a large patch of cells which dies and so is evident to the naked eye as a necrotic fleck (infection types 0; and 1 in cereal rusts). In some cases, the pathogen may develop to the extent of a small amount of sporulation before the lesion becomes necrotic (infection type 2). It has been shown to be controlled by a 'gene-for-gene' interaction between the pathogen and the host (Flor, 1956). Flor worked with flax rust (*Melampsora lini*), which completes its sexual reproductive cycle on flax (*Linum usitatissimum*), rather than with wheat stem rust which requires two hosts (wheat and barberry) to complete its sexual cycle. In a brilliant study, he showed that for every rust

resistance gene in flax there is a corresponding gene for virulence (actually, avirulence if genes are named after their dominant allele) in flax rust (Table 2). Resistance is usually dominant to susceptibility and resistance genes occur as multiple alleles at a restricted number of loci in the plant while avirulence is usually dominant to virulence and avirulence genes occur at separate loci in the rust. That is, the number of resistance genes that can be expressed in the plant is restricted while there is no such restriction on the number of possible virulences that can be expressed in the rust. This ensures that the pathogen will always be able to overcome the resistance expressed in the host. While the meticulous and exhaustive work of Flor with flax rust has been repeated for very few other diseases, there is evidence that the relationship occurs in many highly specialized parasitic relationships (Sidhu, 1975), including several insect-plant relationships (Broekgaarden et al., 2011). It is usually expressed after the formation of haustoria in the highly specialized biotrophic parasites such as the rusts and powdery mildews and so appears to require intimate molecular contact between the pathogen and host. Rust resistance genes have been cloned from four of the five resistance loci in flax and they all appear to code for similar proteins, in the Nucleotide Binding Site-Leucine Rich Repeat (NBS-LRR) class (Ellis et al., 2007). Many resistance genes for a wide range of pathogens (including *Phytophthora*, rusts, powdery mildew, downy mildew, viruses, nematodes and bacteria) in a wide range of hosts (including potato, lettuce, tomato, barley, maize and *Arabidopsis*) have been found to fit into the same or similar class (Martin et al., 2003; Nimchuk et al., 2003). The resistance genes against the highly specialized phloem-feeding insect parasites of plants also appear to fall into this class (Broekgaarden et al., 2011). Thus, the recent molecular studies appear to confirm that vertical resistance involves a particular molecular system for recognition and rapid response to an invading parasite. On the other hand, the avirulence genes cloned from flax rust code for small secreted proteins ('effector proteins') that show no similarity between loci, providing evidence that their main function is probably something to do with the normal metabolism of the fungus and not to make them 'avirulent' on their host. Their 'avirulence' arises from the fact that they happen to be recognized by the resistance-coded proteins, leading to inhibition of the fungus. If avirulence genes code for proteins with a function in the normal life of the pathogen, this would explain the commonly observed phenomenon of stabilizing selection (Flor, 1956; van der Plank, 1968), whereby races expressing virulence (recessive mutants of A genes, often assumed to be non-functional) at one or more loci tend to be less fit than avirulent races on hosts that have no resistance genes. If avirulence genes code for a variety of normal functions in a pathogen, they could vary in the likelihood of their virulent (double recessive) mutant rising to prominence in the pathogen population, via mutation and selection for fitness, and so overcoming the particular resistance in the plant population (Luig, 1983). For example, if an avirulence gene (AvrX) coded for an essential function in the pathogen, then loss of this function in the homozygous virulent mutant (avrX avrX) may mitigate against the selection and buildup of this mutant, even if it matches the resistance RX in the host population. This could help explain the phenomenon of 'weak' and 'strong' vertical resistance genes as proposed by van der Plank (1968) and observed commonly in the field - i.e. some vertical resistance genes break down much more rapidly than others. The genes that are overcome rapidly are matched by races in which the mutation to virulence has little cost in fitness of the pathogen. If the mutation to virulence against a particular gene imposes a high cost in general fitness of the pathogen, the virulent race will not build up rapidly and the resistance gene will not be rapidly overcome; it will be seen to be 'strong'.

The ecological significance of the gene-for-gene relationship is that the rust is always able to match the resistance in the host – it can have unrestricted expression of virulence genes able to match any resistance genes that may occur in the plant population. The evolutionary significance is that the rust and the flax can co-exist and co-evolve. The evidence for this is that the host and the pathogen still exist: the plant has not driven the rust to extinction and the rust has not driven the plant to extinction. Mathematical studies have shown that the gene-for-gene system as described by Flor and elucidated further by Person et al. (1962) can be the basis of co-evolution when both the plant and the parasite are genetically variable and adaptive over time (i.e. are outbreeding) and genetically diverse in space (i.e. occur in populations of the species consisting of several different genotypes)(Mode, 1958). Geneticists call this ‘balanced polymorphism’ (Person, 1967). The gene-for-gene relationship (vertical resistance) probably evolved as a system that protected natural, genetically diverse, outbreeding and adaptive plant populations from excessive disease on the basis of the well documented ‘mixture’ or ‘multiline’ effect; pathogen races sporulating on a particular plant would not have been able to attack the immediate neighbors which had other resistance genes. This controls the pathogen population so that it doesn’t overly reduce the fitness of the host population (otherwise the host could be outcompeted by other species and become extinct) and the parasite is also able to survive. This system functions as long as the pathogen does not build up a ‘super race’ with virulence against all the genes in the plant population. The stabilizing selection first observed by Flor (1956) and referred to above would tend to reduce the chance of such a race developing.

Thus, we can hypothesize that vertical resistance evolved in the outbreeding, genetically diverse, wild ancestors of crop species before their domestication, and that, as Mode (1958) said, the systems of vertical resistance we see in crop species today “are the relics of ancient systems of polymorphism, stemming from the time when wheat, barley and flax reproduced by outbreeding.” (i.e. before their complete domestication). Evidence for this is the fact that the regions of evolution and diversification of crops (the Vavilov Regions) are the repositories of the vertical resistance genes in those crops (Leppik, 1970); that is where plant breeders, inspired by Vavilov (1951), have gone to find new resistance genes. It is possible that vertical resistance continued to play a role in stabilization of disease in traditional agriculture, where crop diversity was maintained. The rapid breakdown of vertical resistance in modern agriculture is due to the fact that we are now using the genes in planting systems that lack the genetic diversity in space and time of the ancestral wild and the early domesticated plant communities (Browning, 1974; Simmonds, 1979). It is a fascinating fact of agricultural botany that domestication has transformed many of our most important crop species from outbreeders in the wild to inbreeders in agriculture, and we are increasingly transforming our agricultural systems from polycultures to monocultures. The constant trend in modern industrial agriculture, driven, against all ecological wisdom, by global economics and centralized, powerful agricultural institutions, has been the steady elimination of this diversity in crop populations, including the critically important repositories of diversity in the regions of crop evolution (the Vavilov Centres). Thus, in modern agriculture the use of vertical resistance has coincided with a tendency to remove the genetic diversity that probably underpinned it in the wild pathosystems and in traditional mixed agricultural systems. In the deployment of resistance genes, crops are in fact becoming global monocultures, hence the problem and the panic created by Ug99.

3. Horizontal or non-specific resistance

Many words have been written and much heated argument generated in trying to define horizontal resistance precisely. The diversity of terminology applied to disease resistance has been summarized by Robinson (1969, 1976). Much has been said above about vertical resistance. This is necessary in a chapter about horizontal resistance if we define horizontal resistance as any resistance that is not vertical, as originally proposed by Black and Gallegly (1957) who defined field resistance (i.e. horizontal resistance) in the potato as “all forms of inherited resistance that plants possess with the exception of hypersensitivity as controlled by R-genes”. Black restated this view in 1970 – “Field resistance to blight may be defined as the degree of resistance exhibited by a plant to all races of the fungus to which it is not hypersensitive.” Such a definition is clear when ‘specific resistance’ and ‘non-specific resistance’ are substituted for ‘vertical resistance’ and ‘horizontal resistance’, respectively, as many like to do. Race-non-specific (horizontal) resistance is any resistance that is not race-specific, i.e. that does not operate on the gene-for-gene recognition system of Flor (1956) involving a hypersensitive necrosis response in the plants, and, on initial evidence, a particular molecular interaction as described by Ellis et al. (2007). It is the resistance expressed when there are no genes for vertical resistance in the plant or when the resistance has been overcome. Hayes et al. (1925) and Stakman and Levine (1922), in describing the infection types in wheat stem rust, considered that the presence or absence of hypersensitive necrosis marked the divide between resistance and susceptibility; it was then recognized that there are “different levels of susceptibility” (Parlevliet, 1995). Such a definition opens up a Pandora’s box of possible phenotypes, genotypes and mechanisms of horizontal resistance, which is why its definition has been so difficult and contentious. Just about any attempt at precision in defining it raises exceptions that defy the particular definition (Robinson, 1976).

Vertical resistance determines the basic compatibility or incompatibility of an interaction between a plant and its parasite (Table 2). Disease develops normally or it does not. However, once a parasite establishes basic compatibility with its host (i.e. is able to invade and reproduce normally) it is logical to suppose that there are very many points in the subsequent compatible symbiotic infection process that may determine whether the invasion and reproduction is fast and prolific or slow and limited. This is especially so in the highly specialized biotrophic pathogens that depend for their nutrition on an intimate physical and physiological association with live host cells, usually occurring through highly specialized haustoria formed within the cells. Its spores have to germinate on the leaf surface, germ tubes have to locate stomata and form appressoria and penetration pegs through stomatal pores (in rusts) or grow over the surface and directly penetrate the cuticle (in powdery mildews), then the infection hyphae have to penetrate cell walls, form haustoria and establish the active metabolic process of deriving nutrients from the host and becoming a sink for nutrients within the plant. It then has to invade further, forming many more haustoria, and eventually sporulate on the surface of the plant (for powdery mildews) or break through the epidermis to form a pustule of spores (for rusts). During all of these interactions there are opportunities for the physiological processes or morphological structures of the plant to hinder or slow down the interaction, and this could depend on very many genes that play a part in normal plant metabolism and structure. The pathogen will invade fast and sporulate prolifically, allowing it to create a destructive epidemic in the

host population, if it encounters no great physical or physiological obstructions during the process of obtaining nutrients and colonizing the plant tissue and sporulating on the surface of the plant. It will invade more slowly if it encounters any physical or physiological obstructions during the parasitism. These obstructions are likely to be fortuitous, related to normal functions in the plant that, primarily, have nothing to do with resistance; they will exist whether or not the pathogen is present. For example, the proportion of peduncle tissue occupied by sclerenchyma in a wheat variety may restrict invasion and sporulation by stem rust (Hursh, 1924; Hart, 1931). A plant may just have tougher structures that are not damaged by the invading pathogen. This is especially important in stem pathogens, where invasion of a weak stem may result in the collapse of the whole plant. This is clearly evident in all damping-off diseases caused by *Pythium* species. Pythiums can only invade soft, immature hypocotyls, causing collapse (damping-off) of the plants. Once the hypocotyls become lignified they are resistant. This is horizontal resistance. A variety in which lignification is delayed could have less resistance than a variety that is lignified early. It is now possible to alter or reduce the lignification of pasture grasses in order to improve their digestibility to livestock; it has been observed that plants altered in this way become highly susceptible to rusts and insect attack, indicating that lignification of cell walls may be linked to the horizontal or generalized resistance of plants to parasites and herbivores (P. Dracatos, pers. comm.). It is important to note that impediments apart from the basic determination of compatibility may occur also in the pre-penetration and penetration phases of infection, as noted below in potato varieties expressing horizontal resistance prior to the incorporation of R genes. For example, the waxiness of the leaf surface may determine the proportion of spores landing on a leaf that are able to locate stomata and penetrate the leaf. Partial resistance to *Puccinia hordei* in barley has been shown to act before haustoria are formed (Niks, 1988). The cuticle thickness and the rate of vacuolization of epidermal cells may determine the proportion of powdery mildew spores that can establish infections (Schlosser, 1980). These are all expressions of horizontal resistance. It is to this multitude of processes that molecular biologists might profitably look for ways to enhance the resistance in plants, rather than perpetuating the preoccupation with vertical resistance genes whose effects are always likely to be overcome by mutations and selection in the pathogen.

The inheritance of horizontal resistance is best discussed in contrast to the inheritance of vertical resistance. Vertical resistance is invariably controlled by easily identifiable Mendelian genes with strong effects and is very well understood, now even down to the molecular expression of some of the genes involved (Ellis et al., 2007). In most cases, the inheritance of horizontal resistance is complex, different in different diseases and poorly understood, except to say that it is mostly additive and quantitative; it is about as well understood as the genetics of any other quantitative character such as 'yield'. This is not surprising given that horizontal resistance is likely to consist of any aspect of a plant's biology that slows down the growth and sporulation of a pathogen invading the plant in a basically compatible interaction. The obstructions the parasite may encounter are numerous and varied, and so their modes of inheritance will be numerous and varied. A single mechanism may be of great importance in the inhibition, and so the resistance may be dominated by the single gene that controls that mechanism (which may be called a 'resistance gene', e.g. the gene *Rpg1* for durable resistance to stem rust in barley; Steffenson, 1992). Or it may be due to many aspects of the interaction, in which case it would be recognized as having 'quantitative' or 'additive' or 'complex' inheritance, and said to be

controlled by 'polygenes' or inherited 'polygenically'. It is entirely possible that factors in the host that fortuitously inhibit the pathogen may be overcome by adaptation in the pathogen population (Parlevliet, 1995). That is, there may be variants of the pathogen that are not inhibited as much as other variants, and these variants may have a selective advantage because of their slightly greater fitness. They may sporulate more than other isolates and so contribute more offspring to the next generation. However, this adaptive process is not expected to be as rapid as that involved in the breakdown of vertical resistance. When it occurs, it is more likely to be expressed as a slow 'erosion' rather than a rapid 'breakdown' of resistance, as noted by Toxopeus (1956) and Niederhauser (1962) for late blight in potatoes. The difference between the more inhibited and less inhibited pathogen phenotypes is likely to be a matter of degree, not the extreme differences evident in vertical resistance, and so the selective pressures changing the pathogen population are likely to be far less. There are commonly several points of inhibition; being part of the normal functioning of the plant, they are likely to function independently in resistance, and so the adaptation of a pathogen variant at one point of inhibition is not likely to affect other points of inhibition.

Horizontal resistance can be accumulated by continually crossing and selecting varieties with resistance with little detailed understanding of the genes involved, in the same way that yield and environmental adaptation of a crop have been built up steadily from generation to generation with little understanding of the many genes involved. This is shown in some of the examples given below. Resistance to vascular streak dieback in cocoa was selected in Papua New Guinea even before the cause of the disease was known. Because the genes controlling horizontal resistance are not primarily 'resistance genes' but just the genes involved in the normal processes of the plant, van der Plank (1968) has suggested that 'there may be large untapped reserves of horizontal resistance in many crops'. Parlevliet (1995) concludes that the search for quantitative (horizontal) resistance in alien species is unlikely to be fruitful and advises that "Fortunately, there is in most crop-pathogen systems no need for these procedures, as quantitative resistance appears to be present sufficiently within the crop species whenever scientists look for it." There are many examples where crossing of susceptible or resistant varieties results in transgressive segregation of resistance, whereby some progeny are more resistant than either parent (and some are more susceptible) (Skovmand et al., 1978). Robinson (1979) suggests that 'good sources' of resistance are not necessary for breeding for horizontal resistance, which, because it is not based on R genes but rather on the normal processes of a plant, can be built up from the normal range of genetic resources in a species. In fact, the preoccupation with genes and gene-transfer (by crossing and back-crossing) in conventional breeding (and now in genetic engineering using recombinant DNA methods) is inimical to the development of horizontal resistance, which usually requires the accumulation of many unknown genes, better served by recurrent selection methods (Robinson, 1979).

The early students of vertical resistance were well aware of horizontal resistance and greatly valued it, probably because they were the first witnesses of the catastrophic breakdown of vertical resistance. Hayes et al. (1925), based on observations of wheat stem rust, described the difference between vertical and horizontal resistance very early in the development of our understanding of disease resistance - "It is known definitely that there are two types of resistance: (1) a true protoplasmic resistance which varies very little, and (2) a morphological resistance which varies with the age of the host and the conditions under

which it is grown." They considered that the former was due to a "real physiological incompatibility between the resistant plants and the invading fungus" and that "the struggle between host and parasite was short and decisive and involved only a few cells in the most resistant plants ---- In susceptible varieties, however, the fungus apparently does not injure the host cells immediately but actually seems to stimulate them to increase physiological activity." Stakman and Harrar (1957) in their important textbook *Principles of Plant Pathology* recognized that "There are various types of resistance in plants. The more kinds a variety has, the more likely it is to be generally resistant. The high degree of specificity between certain physiological races of pathogens and certain varieties of plants has been emphasized repeatedly. A variety may be immune from one race but completely susceptible to another. If more can be learned about the kinds of resistance that are effective against all physiological races, however, it might be possible to breed varieties that have at least some resistance to all races. --- For example, some varieties of wheat have physiological resistance to many races of the stem rust fungus. If it is possible to add resistance to entrance because of stomatal characters, to extension in the tissues because of tough cells and barriers of sclerenchyma, and to the rupture of the epidermis by the sporulating mycelium, the variety should be much more resistant than those which have only one or a few of the many characters that can contribute to resistance. Even though the specific contribution of each character might be relatively slight, the combination of all of them might be effective under a wide range of conditions." The insights of these early students of disease resistance have often been forgotten.

It is worth discussing resistance to late blight in potato in more detail as this clearly shows the contrast between vertical and horizontal resistance, as thoroughly reviewed by Thurston (1971). The potato now widely grown throughout the world, *S. tuberosum* ssp. *tuberosum*, but thought to have originated in the Andian region of South America, had no vertical resistance to *Ph. infestans* until resistance genes were bred into it in Europe by crossing with a wild relative, *S. demissum*, which is native to Central America and clearly had a co-evolved vertical resistance pathosystem with *Ph. infestans* (controlled by R genes, eleven of which have been transferred into the potato in the 20th Century; Malcolmson & Black, 1966). It appears that *Ph. infestans* evolved as a parasite on *S. demissum* and other wild species in Central America and not on *S. tuberosum* ssp. *tuberosum* and *S. tuberosum* ssp. *andigena* in the Andes, and is in fact a 'new-encounter' pathogen on *S. tuberosum* ssp. *tuberosum* (Leppik, 1970). Varieties like Maritta and Kennebec that have the R1 resistance gene transferred from *S. demissum* show complete resistance expressed as hypersensitive necrosis to pathogen races with avirulence on R1, but are susceptible to races that have virulence against R1 (Table 2); however, Kennebec is more susceptible than Maritta to these virulent races (van der Plank 1963). Maritta has more background horizontal resistance than Kennebec to races that can infect both varieties. Many potato varieties lack R genes, and these vary in horizontal resistance to *Ph. infestans*. The variety Capella has a very high degree of horizontal resistance: it can become infected (i.e. it can be said to be 'compatible' with *Ph. infestans* - the fungus can grow and reproduce in the variety), but the fungus takes longer to produce lesions, the lesions are smaller, the fungus sporulates sparsely on the lesions, the plants remain green overall, the epidemic develops slowly, and the variety still yields well (van der Plank, 1963). This is in sharp contrast to the very susceptible varieties being grown in Ireland at the time of the great Irish Potato Famine in 1845-47, where the disease developed very fast, spread throughout western Europe in a matter of months, killed plants

rapidly, decimated yield and rotted even the few tubers that were formed. Beginning in 1912, many observers documented the occurrence of degrees of resistance to late blight in the field before the time when R genes were bred into the potato (Thurston, 1971). This was often referred to as 'general resistance'. It was noted that inhibition of disease in these early resistant varieties could be due to inhibition at several stages of the process of pathogenesis, most notably resulting in (i) a reduced number of infections for a given inoculum dose (i.e. inhibition acting prior to penetration of the leaf by the fungus), (ii) a reduced rate of growth of mycelium in the plant tissues, (iii) a delay in sporulation, and (iv) a reduced number of sporangia produced per unit area of lesion. While vertical resistance is invariably associated with the sudden collapse and death of host cells during the initial establishment of parasitism (especially establishment of haustoria), horizontal resistance can be associated with death of invaded host tissue much later in the parasitic process. Van der Plank (1968, p.185) described how necrosis often occurs in the centres of developing lesions on potato varieties with horizontal resistance to *Ph. infestans*, resulting in a narrower zone of sporulation on the lesion than in a susceptible variety; he considered that it was possible to judge the degree of horizontal resistance of a variety by the amount of necrosis evident in sporulating lesions. Necrosis that appears to reduce the amount of sporulation on lesions is also evident in wheat varieties with horizontal (adult-plant) resistance to stripe rust (*Puccinia striiformis*) and Robusta coffee with horizontal resistance to leaf rust (*Hemileia vastatrix*).

Recent molecular studies have found that factors controlling general (horizontal) resistance to potato late blight occur on almost every potato chromosome and have confirmed that this resistance is, indeed, polygenic (Gebhardt & Valkonen, 2001). Several Quantitative Trait Loci (QTL) for use in marker assisted breeding for horizontal resistance have been located. The degree of general resistance observed in the early varieties was often seen to be affected by environmental factors and the developmental stage of the plant. The well documented history of general resistance in potatoes allows the conclusion that this resistance has, indeed, been durable. For example, Thurston (1971) documents the history of the variety Champion, which was first widely grown in Ireland in 1877 and was clearly popular because of its resistance to late blight, constituting 70% of the potato plantings in 1898. In 1953 Muller & Haigh reported that Champion still had a very high level of resistance. However, following the discovery of the R genes in *S. demissum* and their transfer into *S. tuberosum* ssp. *tuberosum*, as Thurston (1971) commented, "For several decades, almost all potato breeders dropped their work on general resistance and concentrated on obtaining commercial potato varieties with R-genes." Following the failure of R genes to provide long-term resistance, potato breeders turned back to horizontal resistance (Toxopeus, 1964). Van der Plank (1971) documented the fact that six potato varieties released without R genes in the 1920s and 1930s maintained their resistance rating of 6-9 (on a scale of 3=very susceptible to 10=very resistant) over a 30-year period from 1938 to 1968. Black (1970) showed that such resistance could be accumulated rapidly through crossing and selecting appropriate resistant material. In fact, Black turned back to *S. demissum* as a source of horizontal resistance, maintaining that it was mainly horizontal resistance that protected the wild potato species from *Ph. infestans* in Mexico. He showed that by crossing and selecting agronomically useful potato varieties in the presence of late blight, high levels of horizontal resistance could be accumulated. He established that the crossing of two moderately resistant varieties could result in some highly resistant progeny (due to transgressive

segregation). As he said, "it is possible for two resisters --- to possess different resistance factors, and thereby to produce on hybridization a proportion of seedlings of greater resistance than either parent." In fact, Black's 1970 paper is a compact manual for breeding for horizontal resistance. It shows how easy breeding for a quantitative character can be, involving steady accumulation of resistance rather than the game of snakes and ladders associated with breeding for vertical resistance. It avoids the bewildering work of collecting, identifying and naming new pathogen races, and involves working on races only to the extent that inoculation of test plots must be done with pathogen races that have virulence on all the vertical resistance genes that occur in the parent plants.

4. Examples of disease management with horizontal resistance

4.1 Rusts of maize

In the Americas, the two co-evolved rust pathogens of maize, common rust (*Puccinia sorghi*) and tropical rust (*P. polysora*), are regarded as minor diseases. At least one of them is found infecting nearly every maize plant throughout its natural range in Central America, but there is no report of serious rust epidemics on maize in the region (Borlaug, 1972). Certainly, the maize rusts "have been much less important in limiting corn production in the tropics and subtropics than has *Puccinia graminis tritici* on wheat under similar conditions." (Borlaug, 1965). In the extensive and productive corn belt of the United States, the common species, *P. sorghi*, has caused little damage even though the conditions of vast areas of intensive cultivation, continuous presence of the pathogen and environmental conditions conducive to the rust are ideal for epidemic development (Hooker, 1967). There are vertical resistance genes against the maize rusts but these have been unimportant, and the minor status of the rusts has been maintained by horizontal resistance. The fact that maize is outbreeding has facilitated the continuous bulk selection in maize for horizontal resistance, whereas the inbreeding small grain cereals have not allowed this process except following conscious cross-breeding to create the genetic diversity required for selection of improved types. Experience with the maize rusts is evidence that the long list of 'minor diseases' observed for each crop species and listed in the various compendia are kept to their 'minor' status by horizontal resistance (Hooker, 1967; Simmonds, 1991).

Van der Plank (1968, p. 155) has described the local, on-farm and highly effective selection for horizontal resistance to tropical rust (*P. polysora*) in maize in Africa. Maize was probably first introduced to Africa soon after Columbus crossed the Atlantic in 1492 and began the introduction to the Old World of American crops. Given the amount of shipping contact between Africa and America over the centuries, there were undoubtedly numerous introductions, resulting in great genetic diversity of the crop in Africa. The fact that maize is outbreeding would also have ensured its genetic diversity. *Puccinia sorghi* was introduced very early and remained of no importance, as in its centre of evolution (Harlan, 1976). Maize became a staple crop and thrived in Africa for at least four centuries in the absence of the tropical rust with which it had co-evolved in America. When *P. polysora* eventually found its way to Africa in 1949 (Schieber, 1971), it caused devastating epidemics, killed plants and massively reduced maize yields, and swept across the continent in a way that suggested a grand epidemic and great susceptibility in the maize populations, the like of which has never been reported in America (Borlaug, 1972). Van der Plank (1968) presented evidence that horizontal resistance to tropical rust had declined greatly in maize in Africa during its

400-year separation from the rust, resulting in a destructive epidemic when the pathogen was eventually introduced. In fact, *P. polysora* was barely mentioned in the plant pathology literature before it became destructive on maize in Africa. The pathogen spread eastward across Africa and into Southeast Asia and Melanesia. However, the destructiveness of the disease declined after the initial epidemic (Cammack, 1960), and now throughout this extended range it is regarded as of little importance although it can be found on most maize plants. It is evident that selection by farmers of resistant types from genetically variable populations, as has been done since time immemorial in America, resulted in rapid accumulation of horizontal resistance to *P. polysora* in African and Asian maize populations. Farmers would have selected seed preferentially from the resistant survivors of the epidemic; often they would have had no choice since highly susceptible genotypes were killed (Harlan, 1976) or would not have produced much seed. Van der Plank (1968) and Robinson (1976) argued that this experience showed how rapidly and effectively adequate levels of horizontal resistance could be accumulated by bulk selection from genetically diverse crop populations. In contrast to what happened on the farms, researchers conducting seedling tests concluded that there was no resistance in African maize. They were looking for vertical resistance. They were looking for 'genes for resistance' (Stanton & Cammack, 1953).

4.2 Leaf rust (*Hemileia vastatrix*) of coffee

The contrast between vertical and horizontal resistance has been evident in the quest to control leaf rust (*Hemileia vastatrix*) on Arabica coffee (*Coffea arabica*), the species grown in the highlands of many tropical countries to produce high-flavor coffee. Leaf rust has long been a devastating disease on Arabica coffee. It destroyed the plantations in Ceylon (Sri Lanka) in the period 1870 - 1890, reducing the industry from the world's major supplier of coffee to nil (Large 1962), and has caused serious problems since its spread to all coffee-producing countries, including the Americas following its introduction from Africa in 1970. Much of the damage results from the premature defoliation of leaves with even moderate amounts of infection. Severe defoliation eventually kills the coffee bushes. Beginning in 1911 with Kent's selection in India, a succession of resistance genes (S_H genes 1 to 6) was used in an attempt to control the disease, but with their widespread use in the field these all succumbed rapidly to selection of virulent rust races (having virulence genes 1 to 6) (Rodrigues, 1984). This was the typical expression of vertical resistance.

Quantitative resistance to leaf rust has been found in Arabica coffee, for example in Ethiopia and in particular material from eastern Sudan and Kenya (van der Graaff, 1986). This was expressed as differences in latent period, number of lesions, and period of leaf retention after infection. Transgressive segregation for resistance was observed in some crosses and there was no doubt that the resistance was inherited quantitatively. However, most interest has centred on the resistance of a less important commercial species of coffee, *Coffea canephora* (especially the varieties known as 'Kouillou' and 'Robusta'). This species is adapted to the tropical lowlands, where it has become commercially important (e.g. in Brazil and Indonesia) although it is regarded as having inferior flavor to Arabica coffee. Leaf rust commonly infects Kouillou coffee in Brazil but it is not regarded as a serious problem (Eskes 1983), despite the fact that the warm, humid lowland environment appears ideal for the activity of the rust, which is more damaging on Arabica coffee at lower than at higher

altitudes. Following the introduction of *C. canephora* to Java in 1900 after the devastation of Arabica coffee by rust, the Robusta variety showed high levels of resistance, which has been maintained and even increased by selection and breeding to the present time (Kushalappa & Eskes, 1989). On trees in Indonesia, older leaves commonly have some lesions, but these never cover the leaves and they never appear to cause premature defoliation. No one seems concerned about the disease. The resistance is partial, is quantitatively inherited, and has been stable over a very long time; there are no reports of a sudden destructive upsurge of rust on the lowland species. It is horizontal resistance. Like the horizontal resistance of potato to *P. infestans*, it is associated with necrosis of large areas of tissue which appears to limit sporulation. This resistance has played a big part in the management of rust in Arabica coffee in recent decades (Kushalappa & Eskes, 1989). The horizontal resistance of Robusta coffee was incorporated into Arabica coffee in a rare hybridization between the tetraploid, self-compatible *C. arabica* and the diploid, cross-pollinated *C. canephora*, discovered in 1927 in East Timor (Rodrigues, 1984) where plantings of highlands Arabica coffee overlapped with plantings of the lowlands Robusta coffee. Tetraploid progeny of this hybridization were selected as 'Hibrido de Timor' and planted widely as a rust resistant Arabica flavor type in East Timor. Later in Brazil, the compact high quality variety Caturra was crossed with the Timor hybrid to produce the agronomically acceptable Catimor lines of Arabica coffee with the flavor of Arabica and the rust resistance of Robusta (Rodrigues, 1984). Catimor lines are now grown widely around the world to manage rust, apparently without any catastrophic loss of resistance. Similar types of full flavoured, rust-resistant coffee, known as 'Arabusta coffee', presumably of a similar origin, are now grown commercially in Indonesia (e.g. in the Toraja region of South Sulawesi). Moreno-Ruiz & Castillo-Zapata (1990) have described in detail the development of the rust resistant, compact variety 'Colombia' from 'Hibrido de Timor' in Colombia.

Arabica coffee can still be found growing wild and semi-domesticated in the highlands of Ethiopia, where it and the rust co-evolved. Here we can see the ecology of a crop species and its co-evolved pathogen in wild, ancestral communities of the species. It is evident that the wild coffee forests consist of a mixture of genotypes with different resistance genes, and probably types with a moderate degree of horizontal resistance, such that coffee rust is not seen as being epidemic there, certainly not to the extent seen in commercial Arabica coffee plantations in various countries since the 1870s (van der Graaff, 1986).

4.3 Blackleg disease of canola caused by *Leptosphaeria maculans*

The value of horizontal resistance was shown by its straightforward selection in the oil-seed crop canola (*Brassica napus*, bred for seed with low levels of toxic erucic acid and glucosinolates) to control blackleg disease (caused by the ascomycete *Leptosphaeria maculans*) that had practically destroyed the crop in southern Australia in the 1970s (Salisbury et al., 1995). This brilliant work enabled the establishment of a highly productive canola industry and added a crucial crop to the wheat-legume rotation that has sustained dryland cropping in Australia. The fungus invades the laminae of cotyledons and initial leaves as a biotroph but tissues behind the hyphal front die and the fungus eventually sporulates on the dead tissue. The fungus grows down the petiole and into the stem where it invades and eventually kills tissues of the stem cortex. Stem cankering ('blackleg') is the main cause of damage to the plants; in the most susceptible types stems may be completely girdled and

the plant tops may collapse and die before maturity. There is variation in the pathogenicity of isolates of the pathogen; some weakly pathogenic types can form lesions on cotyledons and leaves but not stem cankers while highly pathogenic types progress to form damaging stem cankers. The latter predominate in Australia and selection of disease resistance was essential for the survival of the crop.

Salisbury and co-workers selected horizontal resistance to blackleg by exposing a wide range of canola genotypes to the pathogen in nursery plots heavily contaminated with infested crop residues. The more resistant types survived the blackleg epidemics that developed and were selected for further breeding work. These had mature plant resistance which was evidently inherited polygenically. The resistance was partial: the pathogen invaded and colonized the cotyledons and leaves of resistant types but stem cankering was reduced or eliminated, although under heavy disease pressure, the resistant types could still suffer significant amounts of disease. Continued improvement in resistance was achieved by crossing of partially resistant types and further selection using the same method of field testing (Salisbury, 1988), with the result that the canola varieties produced had "the highest levels of blackleg resistance of any spring canola varieties in the world" and when these were grown with appropriate cultural control measures, losses were negligible. In this initial work little attention appears to have been paid to pathogen races even though it was known that the fungus reproduced sexually and was highly variable. It was not necessary to do so. In a disease such as blackleg, it is possible to imagine that any plant characters associated with strengthening of the stem base may well contribute to resistance or tolerance to the disease; a stronger stem may be less liable to invasion by the fungus, and, if invaded, may be less liable to collapse leading to the death of the plant. This is the sort of resistance seen in many plant species to weak pathogens such as *Pythium*: as tissues of the hypocotyl and lower stem mature, they become completely resistant simply by dint of their increased mechanical strength.

There are many lessons to be learned from this work. Firstly, inoculations of seedlings in a glasshouse gave different results than field tests. Later Salisbury & Ballinger (1996) showed that the resistance of seedlings tested in a glasshouse and of developing plants tested in the nursery plots were under different genetic control. The resistance evident in the blackleg nurseries was effective in the field. The basis for the success of this work was the field testing of resistance against the prevailing races of the highly variable fungus. There is evidence that this resistance can be eroded over time under severe disease pressure (Salisbury et al., 1995), but it has been relatively stable and subject to steady improvement in breeding programs. There has been no spectacular breakdown. In stark contrast, when in a separate program several varieties with vertical resistance (immunity expressed as hypersensitive necrosis) to the disease, controlled by a single dominant resistance gene bred into canola from a related species, *Brassica rapa* ssp. *sylvestris*, were released, the resistance broke down within three years (Sprague et al., 2006). Races of the pathogen virulent on these resistant varieties were selected from among the highly variable *L. maculans* population. Researchers then had to worry about the races of the pathogen and its high variability.

It appears that these vertically resistant varieties had been developed without a background of the horizontal resistance selected by Salisbury. When the resistance broke down, disease severity (measured as percent of the stem cross section blackened) was very high in the varieties with vertical resistance compared with nearby older varieties with only horizontal

resistance. The availability of varieties with immunity gave the farmers a false sense of security and encouraged them to plant the crop more intensively than previously, placing immense selective pressure on the pathogen. It is clear that the management of this disease in the future should rely on the horizontal resistance selected in the 1970s and since built up by regular crossing and selection among resistant varieties, combined with cultural control measures such as crop rotation and separation of new plantings from the previous crops (Marcroft et al., 2004). If vertical resistance is used, it must be added to a background of horizontal resistance. If there is evidence of erosion of horizontal resistance, this can be addressed by a program of steady improvement in resistance as practiced during the 1980s.

4.4 Vascular streak dieback of cocoa caused by *Oncobasidium theobromae* in Southeast Asia and Melanesia

It is now rare to see complete susceptibility to a pathogen in the field. Historical records sometimes give an indication of it (as in the Irish Potato Famine of 1845-47, or the coffee leaf rust epidemics in Southeast Asia in the 1890s), or we can glimpse it when a very susceptible variety is inoculated in a glasshouse, but in general we grow up seeing only crops that have been selected for a relatively high degree of horizontal resistance. These are the survivors of epidemics past. In 1969 in Papua New Guinea the author witnessed the extreme susceptibility of cocoa (*Theobroma cacao*) to a dieback disease later shown to be caused by the indigenous basidiomycete, *Oncobasidium* (*Ceratobasidium*) *theobromae*, which invades only the xylem and causes vascular streaking after which the disease was named (Keane & Prior, 1991). This new-encounter pathogen killed a large proportion of the genetically diverse cocoa plantings established in Papua New Guinea in the 1950s and 60s, leaving only the types with a degree of resistance that enabled them to survive the destructive epidemic. Farmers in the field and agronomists at the Lowlands Agricultural Experiment Station, Keravat, East New Britain Province, had no choice but to propagate from the survivors and, in so doing, selected types with disease resistance that has ever since sustained the industry in Papua New Guinea and throughout Southeast Asia where cocoa has become a major crop despite the presence of the disease (Indonesia is now the third largest producer of cocoa in the world). In fact, this resistance was selected by farmers and agronomists even before the cause of the disease was known, following the fundamental process of natural selection that has undoubtedly sustained wild plant species through evolutionary time and domesticated species since the dawn of agriculture. Some cocoa clones being tested on the Experiment Station were highly susceptible and became extinct - the fungus grew through their xylem so rapidly that it penetrated into the lower stems and roots, completely blocked the xylem, and killed the trees. Others were only slightly affected. Resistant genotypes become infected but the disease progresses more slowly in the xylem, doing less damage to the trees, and the fungus sporulates less. Resistance is quantitatively inherited and has high heritability (Tan & Tan, 1988). It has been relatively easy to select for in breeding programs. The epidemics are much reduced compared with those seen in the 1960s and the resistance is adequate to control the disease as part of an IPM program that includes heavy pruning of cocoa and shade trees to remove infected branches and maintain an open, drier canopy. It has been durable for over 50 years and is still important wherever cocoa is grown throughout the region. Vertical resistance has not been found for this disease, and it is postulated that it is unlikely to occur in this new-encounter pathogen that has had, at most, a history of 300 years of contact with cocoa since the first introduction of the crop to Southeast Asia from Central America.

4.5 Foliar disease of *Eucalyptus* in Australia

Eucalyptus globulus (blue gum) is undergoing a rapid process of domestication. It is fast becoming one of the few indigenous Australian species to be added to the pantheon of the world's domesticated plants and is now one of the most widely planted tree species in the temperate zones. While foliar diseases are of little concern in native forests, they can be destructive in plantations of a single species such as blue gum (Park et al., 2000). One of the most serious diseases has been *Mycosphaerella* leaf blight caused by species of the ascomycete *Mycosphaerella*, which have co-evolved with *Eucalyptus*. The fungi initially invade the leaf tissues biotrophically and then cause sudden death of the invaded area to produce a necrotic blight on which the ascocarps are formed. Young, soft, expanding leaves are much more susceptible to infection than older, fully expanded, harder leaves. When collections of blue gum provenances from throughout its natural range were compared at one location favourable for *Mycosphaerella* leaf blight, significantly different degrees of disease incidence and severity occurred on the different provenances (Carnegie et al., 1994). Provenances from cold locations where the disease was likely to be less active tended to be very susceptible, while those from warmer areas with more summer rainfall where the disease was likely to be active were much more resistant. There had apparently been greater selection for disease resistance in locations where it was of more benefit to the host. This is horizontal resistance. It is partial, being assessed on a continuous scale from low to high percent leaf area affected, and is quantitatively inherited (Dungey et al., 1997). It has not been seen to be associated with hypersensitive necrosis of leaf tissue. It can be readily selected for in breeding programs and will be important in the development of improved varieties of blue gum for places where the disease is serious.

5. Resistance to stem rust and stripe rust in wheat

Some history of the early selection of 'off-types' of wheat with apparent high levels of resistance to stem rust during severe epidemics in Australia is referred to above. In 1894, a farmer, H.J. Gluyas, from the northern wheat belt of South Australia selected from Ward's Prolific an 'off-type' which he called "Early Gluyas"; from 1910 to about 1940 this was an important variety in the drier areas of Australia and became an important parent in the wheat breeding programs that developed from the turn of the 20th Century (Callaghan & Millington, 1956). An early contribution to rust control in Australia was the selection of early maturing varieties of wheat by William Farrer (Callaghan & Millington, 1956). These tended to escape stem rust, which built up and did most of its damage late in the growing season, and could be grown in drier areas where the disease was less of a problem. As well as aiming to produce early maturing varieties, Farrer (1898) also aimed to produce rust resistant varieties and this was one of his selection criteria. It has since been shown that some of his varieties did indeed have resistance to some of the rust races common up until 1926 (Waterhouse, 1936). Farrer's most famous variety was Federation, derived from crosses between Indian varieties, Canadian Fife wheats, and a high yielding commercial variety of the time, Purple Straw. This had stiff, short straw, was a good yielder, and matured early. It was first released in 1901, and from 1910 to 1925 was the most widely grown wheat in Australia. Dundee, a variety derived from Federation with similar characteristics, was in the top two or three most popular varieties in New South Wales and Victoria in 1938, on the dawn of the era of breeding and deployment of varieties with

identified vertical resistance genes. The relatively long-lived popularity of the Federation-type wheats may indicate that, although they were regarded as being susceptible to stem rust, they may have had a degree of horizontal resistance that enabled them to continue to yield well and remain popular with farmers. The fact that they had short, stiff straw could have contributed to this. Farrer had been aware that varieties with erect, stiffer leaves tended to suffer less rust infection. He attempted to combine the (partial) rust resistance of late maturing varieties with earliness (Guthrie, 1922). His methods showed an awareness of quantitative genetics and what is now called 'transgressive segregation'. While it is often stated that he did not develop rust-resistant varieties, this assessment is usually made through the lens of vertical resistance that came to dominate breeding for stem rust resistance in Australia after his death. One of his varieties, Bomen, was still regarded as a valuable variety in the rust-prone northern districts of New South Wales in the early 1920s, and in fact won a Royal Agricultural Society prize for the best crop in 1921 when stem rust was a serious problem (Guthrie, 1922). It is possible that, given his breeding intentions and his methods of selecting for quantitative characters, Farrer did select a degree of horizontal resistance which underpinned the evident longevity of some of his varieties. After his death, his methods which favored the selection of horizontal resistance were replaced by the selection of the Mendelian genes for vertical resistance which has continued to dominate wheat breeding to the present time.

As discussed above for Australia (Watson and Luig, 1963; Table 1) and summarized for North America and Kenya by Person (1967), the use of vertical resistance to control wheat stem rust up until about 1960 resulted in the rapid breakdown of resistance as new races in the rust population adapted to the successive deployment of one or two resistance genes in particular varieties of wheat. This resistance broke down rapidly in Australia and Kenya (within about 5 years), and was also lost in North America, although there it was longer-lived (being effective for more than 10 years in some of the most important varieties). The breeding and deployment of vertical resistance involved a massive effort in surveying the races in the rust populations as the researchers attempted to keep track of the adaptation of the rust to the new varieties, producing bewildering lists of races. In fact, trying to review the race changes in the rust populations is truly confusing, as noted by Waterhouse (1952). The race names bear no relationship to the resistance genes they are matching, but rather are named after the pattern of virulence shown on sets of 'differential' varieties with different resistance genes (McIntosh et al., 1995). Only the fully initiated can easily keep track of the virulence genes that are being expressed in the rust populations. Van der Plank (1983) criticized the current concept of a pathogen race as stretching the bounds of taxonomic practice. If a new resistance gene is found in a host, the number of possible pathogen races increases exponentially (following Flor's gene-for-gene hypothesis, the potential number of races is 2 to the power of the number of resistance genes) and the previously described races have to be re-described to include their virulence on the new gene. Each race has a unique set of characteristics which consists of its virulence, its aggressiveness, and its overall fitness to survive in the environment (Luig, 1983); together these characteristics contribute to its ability to cause epidemics and so each race has to have a taxonomic identifier of some sort. For example, the most common races in Canada during the 1920s had a longer uredinial period on standard varieties than the less common races (Newton et al., 1932), and this trait would have contributed to their survival in the rust population. The two major races of stem rust in North America from the 1930s to the 1960s, race 56 and race 15B, differed greatly in

their aggressiveness as well as their virulence (Katsuya & Green, 1967). On varieties on which both races were virulent, race 56 gave a higher number of infections per unit amount of inoculum, especially at higher temperatures (20-25°C), and had a 2-day shorter latent period than race 15B. Uredinia of race 56 expanded faster although race 15B ultimately had larger uredinia, and race 56 produced more spores per uredinium than race 15B. These differences are important characteristics of the races. They indicate variation in the ability of the races to invade a plant after basic compatibility has been established, equivalent to variation in horizontal resistance in the plants.

In Australia, relatively stable control of the rust in the most rust-prone areas of northern New South Wales and southern Queensland was achieved after about 1960 by assembling combinations of several resistance genes (up to five) in particular varieties so that mutants virulent for one or two genes were still blocked by other unmatched resistance genes (Watson, 1970; McIntosh, 1976; Park, 2007). It was also considered that races with multiple virulences were likely to be less fit than races with simple virulence (Flor, 1956; van der Plank, 1968; Leonard, 1969), and so were unlikely to build up rapidly in the rust population (Watson, 1970). The release of varieties with just one or two resistance genes was avoided so that the rust was denied possible stepping stones for developing full virulence on the varieties with several resistance genes. However, even some of the multiple resistances broke down - e.g. Sr7a, Sr11, Sr17, Sr36 in the variety Mendos (Luig, 1983; Park, 2007), and Sr5, Sr6, Sr8, Sr12 in the variety Oxley (Luig, 1983). However, the strategy was largely successful and McIntosh (1976) was able to conclude that "The sacrifice for almost 35 years of rust resistance has been a regular turnover of cultivars and the loss of effectiveness of a number of resistance genes." Park (2007) considered that particular combinations of genes such as Sr2, Sr24 and Sr26 had been particularly effective. The success was built on a constant effort in surveying the occurrence of rust races and breeding of new varieties with appropriate resistance genes.

There was a general view among wheat pathologists and breeders that horizontal resistance to stem rust was "uncommon in bread wheats" (Watson, 1974) or "yet to be clearly demonstrated" (Knott, 1971), and that it was less likely to have been accumulated over time in the inbreeding crops like wheat than in an outbreeding crop like maize (van der Plank, 1968; Knott 1971). However, Knott (1968) acknowledged the importance of the resistance of Hope and H-44 which can be considered to show horizontal resistance. Scraps of evidence of horizontal resistance can be seen in the early preoccupation with vertical resistance. For example, of the group of varieties with Sr11 involved in the second breakdown of resistance noted in Australia (Table 1), Waterhouse (1952) ranked Gabo as being more resistant than Yalta, both of which had Sr11. After the breakdown of the Sr11 resistance, Yalta was rapidly eliminated from the rust liable areas of New South Wales while Gabo, "being somewhat less susceptible" was still grown successfully (Watson, 1958). It is likely that Gabo had a greater degree of horizontal resistance than Yalta. It was generally known that there were potentially useful forms of resistance other than the vertical resistance expressed as hypersensitivity. Watson (1958) recognized the potential of what he called the "morphological resistance" in Webster that had been transferred into the variety Fedweb and had remained effective against all local races of stem rust. In fact, the resistance of Fedweb lasted from 1938 to 1964 (Park, 2007). Watson (1974) recognized that there were two known types of non-specific resistance that had been transferred into bread wheat (*Triticum aestivum*) from other wheat species. Resistance from *T. turgidum* var. *dicoccum* (Yaroslav

Emmer) had been transferred into the varieties Hope and Renown, and from *T. turgidum* var. *durum* (Iumillo) into the famous variety Thatcher. These resistances appeared to be controlled mainly by single genes, but Watson thought there were other unidentified genes involved. Watson (1974) thought non-specific resistance (presumably from the above sources) had performed well in the cultivars Warigo and Selkirk in the 1973-74 wheat stem rust epidemic in Australia. Warigo had been an exceptional variety during the period of repeated release and breakdown of varieties with single resistance genes (1938 - 1960; Table 1). Released in 1943 and known to have Sr17 (a recessive resistance; McIntosh et al., 1995), its resistance lasted an exceptional 16 years until 1959 (Park, 2007). It had Yaroslav Emmer in its parentage and Watson (1974) assumed that this was part of its success. Now it is known also to contain Sr2, another recessive gene from Yaroslav Emmer that has conferred durable resistance on many varieties (McIntosh et al., 1995). Rees et al. (1979) documented a wide range of horizontal resistance in wheat varieties including some of the older ones used in Australia.

In North America there were two spectacular resistance breakdowns leading to major epidemics in 1935 (associated with the coming to dominance in the rust population of race 56) and 1954 (associated with the dominance of race 15B) (Person, 1967), but it appears that stem rust was generally controlled, except for these two major epidemics, through the use of multiple resistance genes (e.g. Thatcher had Sr5, Sr9g, Sr12 and Sr16; Kolmer et al., 2011; Luig, 1983), and incorporation into the background of many varieties of the horizontal resistance (referred to as 'adult plant resistance') derived from tetraploid wheats (Hare & McIntosh, 1979). The resistance of Hope (with resistance from Yaroslav Emmer) and Thatcher (with resistance from Iumillo) may have helped to partly protect the spring wheat crops in North America during the severe epidemic of 1954. Selkirk, which became the leading spring wheat variety after the 1954 epidemic, had six identified resistance genes (Sr2, Sr6, Sr7b, Sr9d, Sr17, Sr23; Luig, 1983), including Sr2 and Sr17, the two identified recessive genes from Yaroslav Emmer. The evidence that the main North American varieties may have had a degree of horizontal resistance could also account for the longevity of the vertical resistance of these varieties from 1935 through to the 1960s. Certainly, Stakman & Christensen (1960) recognized the occurrence of important levels of resistance in wheat varieties that were susceptible at the seedling stage. It was noted during the stem rust epidemic of 1954 that the amount of damage on the durum wheat variety Stewart was three times that on the bread wheat variety Lee, and this was attributed to the earliness and non-specific resistance of Lee (Loegering et al., 1967).

The ability of cereals to slow down the development of rusts, even though the plants were considered basically susceptible, was recognized by pre-eminent breeders and pathologists many years ago (Farrer, 1898; Stakman & Harrar, 1957). A famous early example was the oat species *Avena byzantina* known as Red Rustproof, which was introduced to the southern United States and recognized as being resistant to crown rust (*Puccinia coronata*) in the 1860s (Luke et al., 1972). It was partially resistant, not immune, and has remained so for over 100 years. It is 'late-rusting'. It is also 'slow-rusting', expressed as a low percent of leaf area infected during the growing season. The degree of resistance varies between varieties of the species. Luke et al. (1972) had no hesitation in recognizing this as 'horizontal resistance'. They were also inclined to call it 'generalized resistance'. Wilcoxson (1981) comprehensively reviewed the biology of slow rusting in cereals and discussed the evidence for long-lived, slow-rusting against stem rust in some well known wheat varieties such as Lee,

McMurachy, Kenya 58, Thatcher and Idaed 59. In crosses between fast- and slow-rusting varieties, Skovmand et al. (1978) showed that transgressive segregation occurred in all crosses, and that slow-rusting was quantitatively inherited with a narrow-sense heritability of 80%. In Europe, farmers and plant breeders over many years used a satisfactory level of horizontal (partial) resistance to protect spring barley against leaf rust (*Puccinia hordei*) (Parlevliet, 1981). Slow rusting in cereals generally involved decreased frequency of penetration, slower invasion of host tissue, longer latent period, smaller pustules, lower sporulation rate, and shorter period of sporulation, singly or, more commonly, in combination (Kuhn et al., 1978; Parlevliet, 1979). The detailed physiology of these effects is not understood, but early workers determined that some physical features of cereals could affect rust development. For example, Hursh (1924) found evidence that the proportion of sclerenchyma to collenchyma in the upper peduncle was correlated with the resistance to stem rust of Sonem Emmer and Kota wheat compared with Little Club. Horizontal resistance, being partial, is strongly affected by environmental factors. It has long been known that excessive nitrogenous fertilization makes cereals more susceptible to rusts (Hursh, 1924). Hart (1931) showed that Webster had several morphological features that increased its resistance to stem rust compared with very susceptible varieties. These included a higher proportion of sclerenchyma in the peduncle and the degree of lignification and relative toughness of the epidermis which often prevented uredinia breaking through to the surface. The resistance in Webster has since been attributed to the gene Sr30, which has been overcome in the Australian variety Festiguay (Knott & McIntosh, 1978). However, it is unlikely that the set of morphological features that inhibit rust infection in Webster (Hart, 1931) is controlled by only one gene.

The known sources of horizontal resistance (often referred to as 'adult plant resistance' or 'durable resistance') against stem rust in wheat are very narrow, consisting mainly of the two tetraploid wheats, *T. turgidum* var. *dicoccum* (cv. Yaroslav Emmer) and *T. turgidum* var. *durum* (cv. Iumillo) and the bread wheat variety Webster (McIntosh et al., 1995). Leppik (1970) lists a range of wild wheat species discovered through the activities of the Russian collecting expeditions that are possible sources of resistance. An unfortunate downside of the spread of dwarf wheat varieties around the world from the 1960s has been the loss of the genetic diversity in the local land races that had probably undergone selection for horizontal resistance. But building up horizontal resistance in a crop does not necessarily involve just searching for sources of resistance, but rather crossing and selection of existing varieties in such a way that genes involved in the normal functioning of the plant that happen to interfere with pathogen growth and development are accumulated and resistance is built up.

Stripe (yellow) rust (*Puccinia striiformis*) was first detected in Australia in 1979 and its history of control by resistance has been very different from that of stem rust. The first response to the incursion, which caused heavy losses in some of the most widely grown varieties such as Zenith, was to deploy vertical resistance genes (YrA and Yr6). However, the effectiveness of these genes was lost very rapidly (Wellings & McIntosh, 1990). It was observed that some wheat varieties such as Condor, Egret and Olympic, although clearly susceptible at the seedling stage, showed varying degrees of adult-plant resistance and suffered much lower losses than the very susceptible varieties such as Zenith (McIntosh & Wellings, 1986). These had similar temperature-sensitive, partial, adult-plant resistance to that observed in some prominent varieties such as Cappelle-Desprez in Europe (Johnson, 1978), and Gaines,

Nugaines and Luke in the Pacific Northwest of the United States (Milus & Line, 1986). The degree of resistance increased as the plants matured, and was greater at higher than lower temperatures (Qayoum & Line, 1985). The resistance was quantitatively inherited, and some crosses showed transgressive segregation (Milus & Line, 1986). This resistance has been long-lived. In Australia, it has been incorporated into many varieties (e.g. Meering and its successors, developed from Condor) and has proved to give long-lasting resistance to stripe rust (Park & Rees, 1989). While it has been identified with Yr18 (McIntosh et al., 1995), additive genes have also been found (Park, 2008) and it has been attributed to a 'Y18 complex' (Ma & Singh, 1996). It is therefore evident that control of stripe rust has relied largely on horizontal resistance.

With the emergence of race Ug99 and its derivatives, virulent on several important Sr genes (Sr24, Sr36, Sr21, Sr31, Sr38) that have been widely distributed around the world from the CIMMYT program in Mexico, wheat breeders are considering turning back to horizontal resistance to control stem rust (Schumann & Leonard, 2011). The emergence of this race has shown the dangers of relying on vertical resistance while steadily eroding the diversity of the global genetic resources of a crop. Before central agencies distributed and promoted particular wheat genotypes around the world, there would have been much greater genetic diversity in the crops. From country to country, from valley to valley and from farmer to farmer there would have been variation in the planting material, including variation in the deployment of resistance genes. An epidemic in one area would not necessarily have threatened another area. With the centralization of breeding for rust resistance in crops such as wheat and the influence of central agencies like CIMMYT in distributing resistance genes, there has been a global narrowing in the base of vertical resistance, such that new races originating in East Africa can threaten the wheat crops of many countries. In fact, it is likely that the global varieties have steadily replaced the local landraces that, in the absence of vertical resistance, probably relied on horizontal resistance to survive and yield in the face of rust infection. Further, the destructive nature of race Ug99 in East Africa suggests that the varieties it is attacking have little horizontal resistance. From the emphasis placed on vertical resistance in wheat breeding (the perpetual search for resistance genes), it is highly likely that horizontal resistance has not been maintained at a high level in the major wheat varieties, in stark contrast to the situation in maize.

6. Resistance of plants to insects

Resistance of plants to insect attack has many of the same characteristics as resistance to microbial pathogens, except that the plant-insect interaction is complicated by the behavioral biology of insects that is lacking in pathogens. Thus, in many reviews, resistance to insects is divided into three components: (i) non-preference, (ii) antibiosis, and (iii) tolerance (Painter, 1958). Tolerance is used here in precisely the same sense as in plant pathology - it is the ability of a plant to survive a certain level of insect attack without suffering significant loss of yield. Entomologists have developed the concept of a threshold level of infestation, below which the insect does not cause significant loss of yield and is not worth worrying about. Non-preference involves the avoidance by insects of particular plants and attraction to others for oviposition or feeding. This involves inherited traits of the plants (e.g. chemical stimuli, colours, morphologies) that can be just

as important in breeding for resistance as mechanisms of antiobiosis. However, it is based on the behavior of the insects and has no equivalent in pathogens. More recently, an indirect mechanism of protection of plants from insects has been recognized (Broekgaarden et al., 2011). This involves the attraction of predator and parasitoid insects to plants releasing volatile chemicals as a result of attack by herbivorous insects. The predation and parasitism then reduces the populations of the pest, effectively protecting the plant. Again, there is no equivalent in plant pathology. Some plants like *Acacia* species produce extra-floral nectaries that attract ants that in turn protect the plant from insect herbivores. These indirect mechanisms are amenable to selection in order to improve the protection of plants from insect pests.

Antiobiosis refers to properties of plants that directly reduce the amount of insect infestation on the plant (equivalent to reducing the amount of pathogen infection). This is similar to resistance against plant pathogens, and can include both vertical (race-specific) and horizontal (race-non-specific) resistance as defined for pathogens by van der Plank (1963, 1968). There is strong evidence that plants have vertical resistance to some of the highly specialized insect pests such as phloem feeding aphids. This resistance has the same attributes as vertical resistance to pathogens. It is often controlled by single, identifiable genes and shows a gene-for-gene interaction as in pathogens, in fact involving the same family of resistance proteins in the plants (NBS-LRRs)(reviewed in Broekgaarden et al., 2011). Long ago, hessian fly was considered to have a gene-for-gene interaction with wheat (Sidhu, 1975; Gallun et al., 1975). Wheat-hessian fly and medicago-bluegreen aphid interactions involve a hypersensitive necrosis reaction like that in plant diseases. And, as with plant diseases, there is ample evidence that this vertical resistance to insects breaks down with its widespread deployment in the field.

Most of the resistance of plants to insects discussed in the literature fits the category of horizontal resistance (reviewed in Yencho et al., 2000). This can be explained by the fact that most insect pests have a far less intimate association with their host than the microbial pathogens: most insects just eat the host tissue. Most resistance in plants against insect attack is partial, inherited quantitatively, and is relatively stable. In fact, horizontal resistance to insects is easier to understand than horizontal resistance to pathogens because the mechanisms are more obvious and easily observed. If an insect is attracted to feed on a plant, and there is no immediate hypersensitive response that prevents it, then there are likely to be a multitude of constitutive or induced factors involved in the insect-plant interaction that either allow the insect to feed unimpeded and rapidly build up its population to damaging levels or restrict its feeding and so control its population on the plant. In reviewing the topic, Beck (1965) concluded that "It is doubtful that any example of resistance can be explained on the basis of a single simple biological characteristic of the plant. The multiplicity of factors exerting influences on the insect-plant relationship precludes the formulation of meaningful all-inclusive generalizations." Plants produce a wide range of secondary plant compounds such as alkaloids, tannins, essential oils, flavones and phenolics that can inhibit the build-up of insect populations, while not necessarily making the plants immune to attack (Beck, 1965; Levin, 1976). Many morphological and physical properties of plants such as density of sticky secretory glandular trichomes, density of hooked trichomes, and tissue toughness due to silica or lignin content may reduce herbivory and/or digestibility and consequently the build-up of insect populations on the

plants (e.g. Tingey, 1979). In response to insect attack, solanaceous plants produce proteinase inhibitors that enhance their resistance to insects (Heath et al., 1997). Most of these traits are likely to be inherited quantitatively (Yencho et al., 2000). Also, like horizontal resistance to plant pathogens, they are liable to erosion if the insect population is able to adapt to particular mechanisms. Thus, many secondary plant compounds that probably initially conditioned resistance to particular insects have become specific attractants for insects that have adapted to their presence. This process has been especially evident in relation to the glucosinolates in the Brassicaceae (Hopkins et al., 2009).

7. The contribution of molecular biology

Several studies of the molecular basis of vertical resistance against several types of parasites (bacteria, fungi and insects) have provided evidence that the gene-for-gene interaction involves a specific molecular system, and that the different R genes, both within and across species, fall within similar gene families (Ellis et al., 2007; Broekgaarden et al., 2011). In other words, the basic incompatibility process involves the expression of variants of the same interactive molecular system. This is a special molecular recognition system that could only have developed through a long period of co-evolution between the host and parasite in a situation where the two types of dominant genes (R-resistance, A-avirulence) had a selective advantage when interacting in genetically diverse populations of host and parasite in the region of evolution of the crop. There is evidence that this specific recognition system does not occur in new-encounter diseases in which there has been insufficient time for such a system to develop. This accounts for the fact that in such diseases (e.g. vascular streak dieback of cocoa, possibly late blight of *S. tuberosum* ssp. *tuberosum*) only horizontal and not vertical resistance has been found naturally.

Another exciting development from the use of recombinant DNA technology is the use of DNA molecular markers for important plant traits such as yield and resistance (Young 1996; Yencho et al., 2000). Mapping and development of DNA markers for Quantitative Trait Loci linked to horizontal resistance could enable the Vertifolia Effect of van der Plank (1963, 1968) and referred to by Black (1970) to be avoided; that is, it could enable the selection of vertical resistance while ensuring that the background horizontal resistance is not lost. While R genes can be added to a variety, and combined as in the effective strategy for wheat stem rust in Australia (McIntosh, 1976), the varieties can be monitored using DNA analysis to ensure that the underlying horizontal resistance is not lost. This is an important development as the presence of a high level of horizontal resistance in vertically resistant varieties reduces the chances of rapid breakdown of vertical resistance, and it reduces the damage done if the resistance does break down. If the vertical resistance is underpinned by good horizontal resistance, breakdown of vertical resistance, as seen with the emergence of a race like Ug99, is not likely to be catastrophic.

It is hypothesized that horizontal resistance is due to any aspect of plant biology that happens to slow down the invasion and sporulation of a pathogen in a basically compatible interaction. This understanding opens up a vast array of functions that could be altered by recombinant DNA techniques in a subtle way that may confer partial resistance on the host, rather than continuing the preoccupation with vertical resistance which we know the pathogen can overcome.

8. Conclusion

Our personal experience conditions how we see the world, including the world of science with which we work. van der Plank (1963) developed his ideas from life-long experience of breeding for resistance to late blight in potato, in which R genes were not effective. They broke down rapidly. Breeding for resistance to late blight relied more on horizontal resistance, and van der Plank was impressed by horizontal resistance. Workers with wheat stem rust grew up with direct personal experience of one of the great biological phenomena discovered in our time, vertical resistance involving the gene-for-gene recognition of a plant species and its co-evolved parasite. This resistance was genetically simple and made a spectacular difference; addition of a single Mendelian gene could convert a very susceptible variety into an immune one and completely protect a crop that had suffered regular devastating epidemics down through history. It is no wonder that researchers were excited by it and worked so hard to exploit it. Even the breakdown of resistance with which they had to contend was such a striking phenomenon, with incredible practical importance on the farms, that this only added to the excitement of the endeavor; researchers not only had to track the resistance genes in the host but also the virulence genes in the pathogen. All the emphasis was placed on making vertical resistance work, with considerable success in the case of wheat stem rust through breeding several resistance genes into each variety, keeping track of virulence changes in the rust populations, and continuously breeding varieties with new resistance genes. Horizontal resistance to wheat rusts was paid little attention during the grand quest to make vertical resistance work in practice. The inbreeding nature of wheat and the other small grain cereals made it harder to accumulate horizontal resistance as occurred in some outbreeding crops such as maize and cocoa. As a consequence, the researchers involved were not enthusiastic about van der Plank's synthesis and many continue to ignore his insights. The present author, introduced to plant pathology through study of vascular streak dieback of cocoa in Papua New Guinea and Southeast Asia, saw the functioning and value of horizontal resistance at first hand. It was easily selected for in an outbreeding, genetically diverse crop exposed to severe epidemics. Indeed, resistance was selected by farmers and agronomists before the cause of the disease was known. Although the pathogen reproduces only sexually and is therefore likely to be highly variable, it has not been necessary, and indeed the biology of the fungus has made it impossible, to be concerned about 'races'. It has proved durable, protecting cocoa from a potentially devastating pathogen and allowing the region to develop over 50 years into the second most important cocoa producing region after West Africa. As a result, this author has been impressed by van der Plank's concept of horizontal resistance.

Long ago, the father of the early work on vertical resistance in the cereals, E.C. Stakman (1957, 1958, 1964), called for greater use of horizontal resistance against cereal rusts. Hooker (1967) suggested that, in developing disease resistance in crops, "perhaps man did not properly assess the resources at his disposal or employed the wrong tactics in their usage." In the light of his experience with the maize rusts he concluded that "If the system prevailing in maize and maize rust is applicable to other host-pathogen systems, then genes for specific hypersensitive-based resistance should be avoided or used only as a minor supplement to a high level of generalized resistance. As many modes of generalized resistance as possible should be combined to produce multimodal resistant varieties." In van der Plank's (1968) and Robinson's (1979) terms, horizontal resistance should be built up in crops as a primary objective and as the foundation of disease management, with vertical

resistance being added as necessary, along with cultural control measures and targeted use of pesticides, as part of an IPM strategy.

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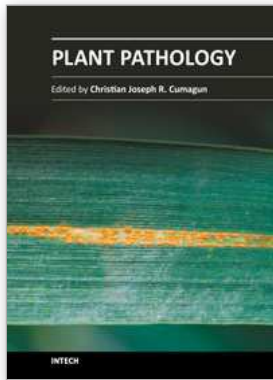
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Plant Pathology

Edited by Dr. Christian Joseph Cumagun

ISBN 978-953-51-0489-6

Hard cover, 362 pages

Publisher InTech

Published online 04, April, 2012

Published in print edition April, 2012

Plant pathology is an applied science that deals with the nature, causes and control of plant diseases in agriculture and forestry. The vital role of plant pathology in attaining food security and food safety for the world cannot be overemphasized.

How to reference

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P.J. Keane (2012). Horizontal or Generalized Resistance to Pathogens in Plants, Plant Pathology, Dr. Christian Joseph Cumagun (Ed.), ISBN: 978-953-51-0489-6, InTech, Available from:

<http://www.intechopen.com/books/plant-pathology/horizontal-or-generalized-resistance-to-pathogens-in-plants>

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Phone: +86-21-62489820
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