Ciba Foundation Symposium 133

PLANT RESISTANCE TO VIRUSES

A Wiley – Interscience Publication

1987

JOHN WILEY & SONS

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PLANT RESISTANCE TO VIRUSES

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Symposium on Plant resistance to viruses, held at the Ciba Foundation, London, 31 March—2 April 1987
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Introduction

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A symposium of this kind needs some introductory comments to set the scene and to point to topics that seem especially to merit consideration. Let me first emphasize that our discussions will take place against a background of the knowledge that virus diseases still cause large yield losses in many crop species. Therefore, progress in solving the scientific problems posed by the need to increase virus resistance in crop plants has important implications for agriculture.

Crop losses attributable to virus diseases are greatest in, but by no means confined to, areas with warmer climates that favour the reproduction and activity of virus vectors. They are especially serious in developing countries in the sub-tropics and tropics. In tropical Africa, for example, maize streak and groundnut rosette viruses can cause devastating disease epidemics, and cassava mosaic is responsible for crop losses which are conservatively valued at more than two hundred million pounds per annum. Over the years, three main categories of control measure have been adopted for preventing virus-induced crop losses. The first type aims to remove virus sources, for example by producing virus-free planting stocks of vegetatively propagated plants, or by removing volunteer plants or plant propagules left from previous crops. The second type is concerned with preventing virus spread, usually by killing or interfering with the activity of virus vectors. The third type, which is the most economical for farmers, is to grow virus-resistant varieties of crops.

In this symposium we are concerned only with virus resistance. This choice seems both appropriate and timely for two main reasons. The first is that the environmental consequences of applying large amounts of vector-killing pesticides to crops are becoming increasingly evident, and public pressure has grown, and can be expected to continue to grow, for their more restricted and more discriminating use. A further factor is that, in some areas where pesticides have been widely used, pesticide-resistant vector organisms have become common (Table 1). A consequence of these developments is that the scope for using crop protection chemicals to prevent virus spread may decrease. The second reason is that recent advances in molecular biology and

TABLE 1 Increases in insecticide resistance of hopper vectors of rice viruses in Taiwan

Insecticide	*Increase in insecticide resistance*[a]	
	Nephotettix cincticeps (green rice leafhopper)[b]	*Nilaparvata lugens (brown planthopper)*[c]
Malathion (organophosphate)	× 27–452	× 288–526
Carbaryl (carbamate)	× 12–79	—
Propoxur (carbamate)	—	× 19–46
Permethrin (pyrethroid)	× 1–6	× 71–121

[a] Range of factors of increase in insecticide resistance of field populations relative to a susceptible control population.
[b] Data from Kao et al (1982).
[c] Data from Chung et al (1982).

biotechnology offer the prospect, not only of speeding up progress in the more conventional approaches to producing virus-resistant plants, but also of exploiting completely new approaches.

Interest in the resistance of varieties of plants to virus diseases probably goes back for at least a couple of centuries, to a time that pre-dated the recognition of viruses as a separate class of pathogens. Although the correct interpretation of old writings can be debatable, an apparently relevant observation is to be found in a book by Marshall (1790) on the rural economy of the Midland counties of England. Marshall recognized varietal differences in potato in the occurrence of 'curledtop', the disease that we now attribute to infection with potato leafroll virus. He wrote: 'The old varieties, formerly in cultivation, dwindling in produce, and being, at length, in a manner destroyed, by the disease of curledtop, two new varieties were introduced. . . . The consequence has been, the disease vanished with the old sorts, and is now (1786) and in *this* neighbourhood, where no other sort is in ordinary cultivation, in a manner forgot'. Although it is uncertain whether the events recorded by Marshall represented the replacement of infected stocks with virus-free clones of other cultivars, or with virus-resistant cultivars, it seems unlikely that all the old infected stocks would have been discarded in the same year, and the virus reservoirs therefore removed simultaneously, and more probable that the new varieties were much more resistant to, or tolerant of, infection than the old ones. This kind of selection for superior performance in the field has proceeded, unconsciously as well as consciously, for centuries. As a result many of the varieties of crop plants in cultivation in areas where these crops have been exposed to a prevalent virus for a long period are cultivars which are tolerant of infection. This is found, for example, in native potato genotypes grown in the Andean region of Peru (Jones 1981). Simi-

larly, the earliest attempts to breed improved crop plants relied on selection, now more often intentional, to eliminate the most readily infectible and sensitive types.

This first phase of plant improvement was succeeded by one in which attempts were made to breed virus-resistant forms by selecting and crossing appropriate parents, and then making selections from among their progeny, backed, where possible, by knowledge about the genetic control of resistance. Good progress was sometimes made without detailed knowledge of the genetic control, as instanced by the programme of breeding sugar beet for resistance to curly top in the United States (Carsner 1933).

Where the range of genetic variation found in a crop species does not include the required degree of virus resistance, this can sometimes be identified in a related species. Efforts to introduce the resistance genes into such crop plants are a feature of the third phase of breeding for virus resistance. For example, the R_y gene for extreme resistance to potato virus Y (Ross 1960) has been transferred by breeders from the primitive species *Solanum stoloniferum* to *S. tuberosum*. Once introduced into suitable parental material, such dominant genes are relatively easy to include in breeding programmes. Recessive genes can also be of value, as exemplified by the resistance of groundnuts to groundnut rosette disease, a property thought to be controlled by two recessive genes (K.R. Bock & S.M. Nigam, unpublished results). The growing knowledge of the genetics of resistance is described in detail by Fraser in this symposium: he considers, on the one side, the genetics of resistance in the plant and, on the other, the genetics of virulence in the virus.

Recent research has built on these foundations in two main ways. First, modern techniques (including electron microscopy, protoplast methodology, and biochemical and serological analysis) have enabled resistance mechanisms within a species to be examined at the cellular and cell-free levels as well as in tissues and intact plants. As a result of these and other analyses, it has become clear that resistance mechanisms can be assigned to two principal categories: innate resistance and induced resistance. *Innate resistance* is heritable and constitutive, and can take a range of forms, some of which are becoming much better understood (see Bruening et al and Nishiguchi & Motoyoshi, this volume). *Induced resistance* is not constitutive and is expressed only after it is activated by some previous infection or treatment. Cross-protection between virus strains is one such example (Sherwood, this volume). Recent information on virus non-specific induced local, and induced systemic, resistances is dealt with in the papers on pathogenesis-related (PR) proteins and antiviral factor (AVF). As an aside, I feel that the designation 'pathogenesis-related' for the PR proteins is unfortunate because these proteins can be produced in substantial amounts without the intervention of a pathogen (Fraser 1981) and they seem more related to stress, or perhaps to ageing, than merely to pathogenesis.

The second important development stems from the application of molecular biological and genetic engineering techniques, and is bringing totally new approaches to virus resistance within our grasp. On the one hand, attempts are under way to isolate virus resistance genes that occur naturally in plants and to define them in molecular terms as a preliminary to transferring them to other plant cultivars or species. On the other hand, we now have examples of at least two successful approaches to enhancing virus resistance by transforming the genome of plants with nucleotide sequences copied from the genetic material of the viruses themselves. In parallel with these genetic engineering approaches to resistance we have the possibility of using viruses to introduce non-viral genetic material into plant cells. These developments, which will be discussed in the last part of this symposium, are the first few small fruits from the application of what is a radically new addition to the range of techniques available for improving plants. As with more conventional kinds of resistance, these novel kinds must be tested carefully for their durability in field conditions, and possible side-effects and environmental hazards must also be assessed.

There is now, therefore, a more impressive array of approaches to improving virus resistance than has been available before, and we may be entering an age in which virus diseases will be controlled as effectively as bacterial diseases are today. However, viral genomes have probably survived and evolved over long periods and it would be surprising if plant viruses lacked the genetic flexibility to generate new forms capable of surviving in a world of plant breeders, virologists and genetic engineers. For the present, I hope that by describing, discussing and drawing together the many recent findings that are relevant to virus resistance, clearer views will emerge both of the scientific problems that are most urgently in need of solution, and also of the experimental approaches that seem suitable for solving them. A whole range of scientific problems come to mind, some more tractable than others. They include the following:

To what extent are kinds of resistance mechanism related to kinds of genetic control in the plant?
How can knowledge about PR proteins and antiviral factors be exploited?
What is the mechanism of cross-protection between virus strains, and does it differ in different virus groups?
How can virus resistance genes in plants best be defined and characterized at the molecular level?
What are the prospects for new or improved genetic engineering approaches to enhancing virus resistance?
How durable in field conditions are different kinds of resistance likely to be?
How do viruses overcome the effects of resistance genes?
What are the main barriers to progress, and how can they be removed?

This personal list is by no means exhaustive but I hope it will serve to provoke thought and discussion when considering the points made by the main contributors to the symposium.

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Genetics of plant resistance to viruses

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Abstract. This paper concerns the genetics of resistance used by the plant breeder to produce cultivars resistant to viruses. Non-host immunity, and resistance induced in normally susceptible individuals, are discussed only where they may share mechanisms with cultivar resistance. Conclusions about the genetics of resistance and of virulence (the ability of a virus isolate to overcome a specific resistance gene) are drawn from a survey of 63 combinations of hosts and viruses, and from comparisons with the predictions made from various theoretical models of host–virus interactions. Most resistance mechanisms that result in virus localization appear to involve an inducible, positive inhibitor of virus replication or spread, which tends to be temperature sensitive. Resistance mechanisms which permit some systemic spread of virus tend to be incompletely dominant (gene-dosage dependent) and are determined by quantitative interactions between host- and virus-specified functions. Completely recessive resistance is rare, and may involve a negative mechanism where the resistant plant lacks a susceptibility function. Most of the resistance genes considered have been overcome by virulent isolates of virus; extreme durability is rare. It appears easier for viruses to mutate to overcome dominant localizing resistance than recessive immunity mechanisms.

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Crop losses caused by plant virus diseases can be controlled in various ways. Infection can be prevented by good hygiene, use of virus-free seed and control of vectors. Viruses can be eliminated from universally infected cultivars or clonally propagated lines by tissue culture techniques. However, in the absence of any chemical treatment analogous to the use of fungicides, the only strategy of control that can be applied directly to field crops is breeding for host resistance.

Full exploitation of resistance in crop protection depends on an understanding of the genetical and biochemical mechanisms involved, and of the nature of the plant–pathogen interaction. In this paper I shall summarize knowledge of the genetics of resistance, and of resistance-breaking behaviour in the virus. I shall indicate where genetic information suggests possible mechanisms. Finally, I shall stress some of the limitations of disease control

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by classical breeding for resistance, and speculate on how the approach could be expanded.

Types of resistance

There has been some confusion in the literature over nomenclature, because of the diversity of plant resistance mechanisms and of the corresponding phenomena in the virus. It is therefore useful to begin with some definitions. Resistance mechanisms can be separated into three broad groups, which operate at different levels of complexity of the host population.

In *non-host resistance* all individuals of a species are completely unaffected by a particular virus; on inoculation the virus produces no symptoms or detectable multiplication. Two genetic models can be suggested. In the positive model, the 'non-host' contains a gene or genes completely effective against all tested isolates of the virus. Holmes (1955) suggested as many as 20 to 40 genes with additive effects, but there is no direct evidence for such polygenic resistance systems in plants, and in any case they would be very difficult to handle in breeding programmes. Bald & Tinsley (1967) suggested a negative mechanism; a species is a non-host because it lacks certain 'susceptibility factors' required by the virus for full pathogenesis. Again there is little direct evidence to support this, but a possible site of action would be cooperation of host- and virus-coded subunits to form a functional replicase. 'Negative' mechanisms of non-host resistance could only be exploited in plant breeding by the modification or deletion of some existing host function. Finally, recent evidence shows that protoplasts, isolated from some plants considered to be non-hosts, can support virus multiplication (e.g. Huber et al 1981). This suggests that at least some cases of non-host immunity may be mediated by physical barriers to infection at the cell wall or epidermis.

Cultivar resistance occurs within a host species. Resistant individuals contain a gene or genes conferring resistance to a virus which affects susceptible members of that species. This is the type of resistance most used by the plant breeder, and is the main subject of this paper. The corresponding effect in the virus is *virulence* — the ability to overcome a specific resistance gene and thus cause disease in a resistant plant.

Induced resistance operates at the level of the individual, when a form of resistance is conferred on a susceptible plant by a prior inoculation, or chemical or environmental treatment. It includes effects such as acquired systemic resistance and the pathogenesis-related proteins, cross-protection, and virus-free green islands in mosaic tissue. The mechanisms are diverse; some may depend on host genes directly involved in cultivar resistance mechanisms, while others are probably indirect effects of other aspects of host metabolism (reviewed by Fraser 1987). Unlike cultivar resistance, induced resistance is not normally heritable, and must be conferred afresh on each generation.

TABLE 1 Genetics of resistance to viruses in crop species and some features of resistance gene action and virulence (derived from data in Fraser 1986, 1987)

Genetic basis:

	Number of host–virus combinations
Single dominant gene	29
Incompletely dominant (gene-dosage dependent)	10
Apparently recessive	11
Sub-total: monogenic	50
Possibly oligogenic	5
Monogenic, with possible modifier genes or effects of host genetic background	8
Sub-total: oligogenic (?)	13
Total number of host–virus combinations in sample	63

Localization[a]*:*

	Immune	*Yes*	*Partial*	*No*	*Not known*	*Total*
Dominant alleles	0	19	0	2	8	29
Incompletely dominant	0	0	4	8	0	12
Apparently recessive	5	1(?)	1	2	4	13

Temperature response[b]*:*

	ts	*tr*	*Not known*	*Total*
Dominant alleles	7	2	20	29
Incompletely dominant	1	1(?)	10	12
Apparently recessive	2	2	9	13

Virulent isolates reported:

	Yes	*No*	*Not known*	*Total*
Dominant alleles	16	1	12	29
Incompletely dominant	8	3	1	12
Apparently recessive	4	1	8	13

[a] Immune, no virus detectable; Yes, normally involving lesion formation; No, resistance permitting some systemic spread; Not known, not tested, or not reported in the literature.
[b] ts, temperature sensitive; tr, temperature resistant.

The genetics of cultivar resistance

Heritable resistance is known in numerous crop species. The numbers of genes involved have been determined by standard genetic methods, by fitting observed segregation ratios to predictions for various models. Resistance alleles have been classified as dominant, incompletely dominant or recessive,

depending on the resistance phenotype of plants homozygous or heterozygous for the resistance allele. Table 1 summarizes the genetic control of resistance in a randomly chosen sample of 63 combinations of hosts and their viruses. Fuller details and literature citations are given elsewhere (Fraser 1986).

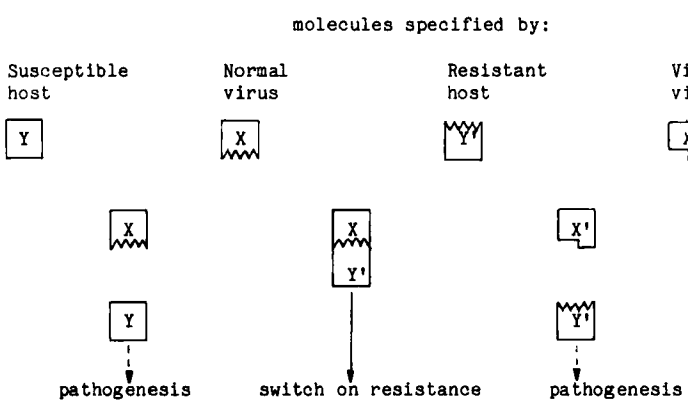
In these cultivated species, resistance is mostly inherited at a single locus. The evidence for oligogenic control, or modifier genes, is weaker. Some early examples of genetically complex resistance were later shown to be monogenic (reviewed by Fraser 1986). The early experiments were conducted under variable environmental conditions, and the proposed genetic complexity was an attempt to explain, in purely genetic terms, segregation ratios resulting from genotype–environment interactions. There are, however, a few cases of well-substantiated host genes which indirectly modify the phenotypic expression of resistance; for example, an effect of plant growth rate on resistance to barley yellow dwarf virus in barley (Jones & Catherall 1970). There are also a few examples of oligogenic resistance systems involving epistatic effects. In resistance to bean common mosaic virus (BCMV) in *Phaseolus vulgaris*, the *bc-u* locus has no antiviral effect alone, but enhances the antiviral effect of resistance genes at any of three other loci (Drijfhout 1978, Day 1984).

Although Table 1 suggests that resistance to viruses in crop species is in most cases very simple genetically, it should be remembered that the resistant cultivars are often a product of deliberate breeding for this trait. The genetics of virus resistance in wild species might well be more complex, but do not appear to have been investigated. There have been attempts by breeders to construct oligogenic systems of resistance by incorporating several individual resistance genes from related wild species into the commercial cultivar. The best examples are resistance to potato virus Y in potato, and to tobacco mosaic virus (TMV) in tomato.

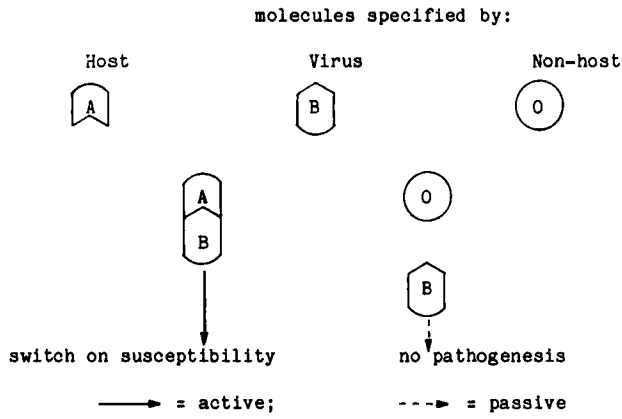
Models of resistance gene action

A useful way of analysing resistance is to make theoretical models of how resistance genes might work, and of the corresponding response of the virus. Predictions are made from these models and then tested against observations. Fig. 1 shows three types of model. In the positive model (1), the resistant plant produces an inhibitor of the viral replicative cycle, whereas in the negative model (2), the resistant plant lacks some susceptibility function normally required by the virus for pathogenesis. Both models involve some form of recognition event between host- and virus-coded functions which determine the outcome of the interaction in a 'go/no-go' manner. In the positive model, recognition switches on resistance, while in the negative model it switches on susceptibility. The third model is intermediate, in that it does not involve an all-or-nothing response like the first and second, but has a

1. **Positive model: resistance is switched on**



2. **Negative model: susceptibility is not switched on**



3. **The quantitative interaction model**

molecules specified by:

Normal virus	Virulent virus	Susceptible host	Resistant host
V	V'	H	H'

[V]	+	[H]	⇌	[VH]	susceptibility
[V]	+	[H']	⇌	[VH']	resistance
[V']	+	[H]	⇌	[V'H]	susceptibility
[V']	+	[H']	⇌	[V'H']	susceptibility

FIG. 1. Three models for interactions between host- and virus-specified molecules which may determine susceptibility or resistance.

response determined by quantitative interactions between host- and virus-specified functions. The outcome, which could be either resistance or susceptibility, depends on the concentration and nature of the recognition product formed. This in turn depends on the concentration and nature of the functions specified by the two participants in the interaction, and can be described by the mathematics of chemical reaction kinetics. The third model could involve either positive or negative mechanisms. Table 2 summarizes some predictions made by the models.

Comparison of models and observations

If we consider only the monogenic resistances, Table 1 shows that most are dominant, with smaller proportions being incompletely dominant or recessive. In fact, the proportion of incompletely dominant alleles is probably an underestimate. Resistance may appear completely recessive or completely dominant when assessed only by scoring visible symptoms, but can show clear

TABLE 2 Predictions from the resistance models

Positive models (Resistance = inhibition)	Resistance is: ■ dominant if recognition is a go/no-go event ■ gene-dosage dependent for quantitative interactions ■ never fully recessive ■ possibly temperature sensitive Virulence is: ■ when a virus function has an altered interaction with the host resistance gene function, or fails to interact
Negative models (Resistance = reduction or absence of susceptibility)	Resistance is: ■ probably recessive for a go/no-go recognition event ■ gene-dosage dependent for qualitative interactions ■ never fully dominant ■ unlikely to be temperature sensitive Virulence is: ■ the ability of the virus to multiply without the host susceptibility factor