DISEASE RESISTANCE IN PLANTS

SECOND EDITION

J. E. Vanderplank

Disease Resistance in Plants

Second Edition

J. E. VANDERPLANK

Plant Protection Research Institute
Pretoria, Republic of South Africa

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Preface

Most of the chapters in this book include information not available when the first edition appeared in 1968. Its comprehensive coverage will be of interest to plant pathologists and plant breeders. Both are concerned with developing new cultivars possessing genetic resistance to diseases; both contribute special skills. But skills alone are not enough. To be properly employed relevant genetic, epidemiologic, biochemical, and biometric principles must be understood. The aim of this work is to help provide such an understanding.

It is known that an incompatibility can exist between high resistance and high yields of grain, fruits, tubers, and bolls. These are metabolic sinks that sometimes induce a loss of resistance. Evidence indicates that the loss, when it occurs, is in horizontal resistance and that vertical resistance escapes sink-induced losses. The desire to obtain the greatest possible yields is perhaps a reason why plant breeders prefer vertical resistance if it is available and can be stabilized by stabilizing the selection of the pathogen. In conjunction with lost resistance, sugar seems to be the most important substance drained by the sink, which suggests that to maintain high yields the best forms of resistance are those enhanced by low sugar content. They are more likely to be found against biotrophy than necrotrophy. This book includes discussions on stabilizing selection, sugar, biotrophy, and necrotrophy.

This publication coincides with a burst of activity in plant genetic engineering, which provides new methods for manipulating genetic material. It will aid plant pathologists and plant breeders to discover what genetic material they wish to manipulate. The nature of resistance and resistance genes is investigated using information not analyzed elsewhere and with results that are a necessary prerequisite for the breeding of resistant varieties by genetic engineering.

Breeding for disease resistance involves two organisms: the host plant and the pathogen. The breeder changes the host; in doing so he may also change the pathogen if host and pathogen are genetically interlocked as they are in a gene-for-gene system. The pathogen changes by reflection from the host. The plant breeder can put genetic reflection to use in order to weaken the pathogen and reduce disease. Reflective genetic engineering of the pathogen may yet become an important part of plant breeding for disease control. A major portion of one chapter is devoted to a discussion of the effect of reflected virulence on the structure of the pathogenic population.

Agronomists and horticulturists must know the disease resistance of the varieties they recommend, and disease resistance is one of the features that determines acceptability to farmers. This book, by comprehensively covering disease resistance and stressing its limitations as well as its advantages, helps provide a suitable basis for making the proper agronomic and horticultural recommendations.

I thank Drs. N. H. Luig and R. A. McIntosh for sending me recent reports on wheat stem rust in Australia.

J. E. Vanderplank

Preface to the First Edition

The purpose of this book is to inquire into the role of resistance in plant disease. It discusses the nature of resistance and how it can best be used to protect crops from disease.

The chapters contain much that is new. First, it is shown that there are two types of pathogenic races and of pathogenicity which correspond to the two types of resistance. The evidence for this seems clear and incontrovertible. Second, the strength of genes for vertical resistance in host plants is measured. This is done by introducing the concept of relative half-lives of matching pathogenic races. The argument is simple and the evidence direct. Third, in discussing vertical resistance to obligate parasites, host–host–pathogen systems replace the host–pathogen systems ordinarily considered in current literature. This conforms with the self-evident fact that there must be two or more host genotypes within the epidemic area if there is to be satisfactory vertical resistance to obligate parasites that do not live long outside their host plants. The change to host–host–pathogen systems will be found to clarify many practical problems in the use of resistance. Fourth, evidence is presented that in its effects on disease a change in the polygenic horizontal resistance of the host plants is often identical with a change in the aggressiveness of the pathogen or of that in the environment. It seems that the genes of horizontal resistance are often not special resistance genes, but ones concerned in the normal metabolic processes of healthy plants. For this reason there may be large untapped reserves of horizontal resistance in many crops. Fifth, a theory is given of vertical and horizontal resistance: Resistance is vertical if to overcome it the pathogen must become less aggressive on susceptible varieties of the host; it is horizontal if to overcome it the pathogen

must become more aggressive on susceptible varieties as well. The theory fits the known facts.

Two topics introduced in my previous book ("Plant Diseases: Epidemics and Control," Academic Press, 1963) now receive more attention. The effect of resistance on the progress of disease in the field has a chapter to itself; disease progress curves are used to illustrate the effect. Stabilizing selection is the main topic of two chapters. It is at the core of stable vertical resistance conferred by strong genes. In emphasizing stabilizing selection the book departs from the conventional treatment of pathogenic races. Instead of an emphasis on how races arise, the theme of countless papers in the literature, it places the emphasis on how fit races are to survive after they have arisen, a theme hitherto much neglected.

The theory of multilines is examined, particularly in relation to stabilizing selection. The great unanswered question is whether there are enough strong genes to maintain adequate resistance in the long run.

The work is intended for plant pathologists, because sooner or later they must deal with matters of resistance; for plant breeders, because breeding for resistance is one of the important reasons for creating new varieties; and for those interested in ecology, because nowhere is natural balance better illustrated than by the interaction of plant host, pathogen, and environment.

The chapters interlock because resistance theories are becoming consistently integrated. However, without too much repetition, I tried to make each chapter an independent unit to reduce the need for tedious cross references.

Pretoria, South Africa *J. E. Van der Plank*
June 1968

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Introduction

Our daily bread comes from wheat fields protected against disease by genetic resistance; chemical protection is practically limited to seed dressings. Our meat, dairy products, and eggs come ultimately from maize, soybeans, pastures, and fodder crops protected against disease by genetic resistance. Our food is sweetened by sugar; a change from susceptible to resistant cultivars saved the sugarcane industry almost worldwide from mosaic disease, and the sugar beet industry in areas west of the Rocky Mountains from curly top disease. So the story continues, with genetic resistance being seen as a pillar of agriculture.

Disease resistance has come from many sources. It has come from transgressive segregation and from other varieties within the crop species. Examples are innumerable. It has come from different but related species and genera. Already at the beginning of the century Orton had transferred to watermelons (*Citrullus lanatus*) the resistance of stock citrons (*C. vulgaris*) to *Fusarium oxysporum* f. sp. *niveum*. Modern tomato cultivars have gained disease resistance from other *Lycopersicon* spp. Hexaploid bread wheat has been fortified by genes from tetraploid and diploid *Triticum*, and intergenerically from *Secale*, *Agropyron*, and *Aegilops*. Knott and Dvořák (1976) have reviewed the use of alien germ plasm for developing disease-resistant cultivars. The advantage of using alien germ plasm is great; an almost limitless source of resistance is tapped. The difficulties are equally great. Fertilization is often difficult; crosses fail soon after fertilization; and hybrid plants may die before flowering or may be sterile. The interspecific and intergeneric

transfer of a gene for disease resistance involves the transfer of a chromosome segment from a donor species or genus to a recipient. The substitution of an alien segment may cause undesirable duplications or deletions, and the segment is likely to carry unwanted genes linked to the wanted resistance gene. Getting rid of the unwanted genes requires crossing-over, and this becomes more difficult when the donor is foreign and the chromosomes are not homologous but only homeologous. Nevertheless, despite the difficulties, there is a large body of literature of successful and useful donations by alien species and genera.

Natural mutation to resistance occurs. In potatoes, mutation from smooth-skinned to russet-skinned tubers commonly brings with it increased resistance to scab caused by *Streptomyces scabies*. Mutagens have been used to introduce resistance, as in peppermint (*Mentha piperita*) to *Verticillium albo-atrum* (Murray, 1969; Todd *et al.*, 1977). Stolons of the susceptible peppermint cultivar Mitcham were treated with neutrons or X-rays to give mutants both resistant and of high horticultural quality. Other examples are quoted in a review of mutagenesis by Simons (1979).

Protoclones are the newest way of developing disease-resistant variants. Shepard *et al.* (1980) found increased resistance to the blights caused by *Phytophthora infestans* and *Alternaria solani* in clonal populations regenerated from mesophyll cell protoplasts of the potato cultivar Russet Burbank. They solved the technical problems of regenerating plants from mesophyll protoplasts and then compared a number of "protoclones," as they called them. Some were more resistant than others to *P. infestans*, and some to *A. solani*. It is still too early to state whether these resistant protoclones differ essentially from the meristem mutants known as bolters and semibolters, which often have increased resistance to blights. But, be the resistant protoclones horticulturally successful or not, the work of Shepard *et al.* marks the entry of genetic engineering into plant breeding for disease resistance. A new era has begun.

Genetic engineering is a general term used to describe cellular and molecular methods for altering the genetics of organisms. Research has developed rapidly in two directions. In the direction of tissue and cell culture, plants (and other multicellular organisms) can be reduced to single cells grown under aseptic laboratory conditions. In the direction of molecular biology, the emphasis is on recombinant DNA techniques. Single genes isolated from one organism can be introduced into another, thereby transforming the recipient genetically. Chromosomes, chromosome segments, DNA preparations, and cellular organelles can also be transferred to a recipient.

Cell cultures can be started from plant tissue by inoculation into an appropriate medium and maintained aseptically. A callus of rapidly dividing cells develops and can be maintained by repeated transfers. Callus cells inoculated

into a liquid medium with continuous agitation form a suspension culture of single cells and small cell aggregates. Protoplasts or plant cells without walls are produced by treatment with enzymes. Protoplasts have unique properties. They fuse with other protoplasts or absorb foreign genes, chromosomes, and organelles. From manipulated protoplasts whole plants can be regenerated by appropriate techniques.

At present these methods are far from routine. They work well with some species, notably tobacco, but there are unsolved difficulties with other plants such as cereals and soybeans. Where they do work, new procedures for producing disease-resistant plants are available. Protoplasts unlock variation, as the results of Shepard *et al.* (1980) show. When disease is caused by a toxin, millions of protoplasts can be screened for resistance in a small flask; they are the equivalent of thousands of acres of growing plants. Protoplast fusion or *in vitro* pollination (fertilization of placentas) can produce hybrids between species and genera of plants where normal sexual procedures fail; this increases the range of donors of resistance. Recombinant DNA methods coupled with protoplast-to-plant regeneration can be aimed at introducing single genes for resistance uncluttered by association with deleterious genes.

Techniques of genetic engineering will develop swiftly. Our particular concern is with how to use them.

Specificity resides in susceptibility. Resistance is unspecific; this is the topic of Chapter 2. Unspecific resistance is the single most fortunate fact in the whole of plant pathology. It makes susceptibility the exception, not the rule. For genetic engineers it makes the whole plant world a treasure-house of resistance genes that we call nonhost resistance genes. Putting these genes to work is an essential part of genetic engineering.

Wheat is resistant to all the rust fungi that attack maize; maize is resistant to all the rust fungi that attack wheat. Wheat is a nonhost of the maize rust fungi, and maize a nonhost of the wheat rust fungi. One of the tasks of coming years is to get rustfree wheat and maize by a selective exchange of nucleotide base sequences. But first we must learn what the relevant sequences are. The available evidence suggests that there are at least three. First, there is (the evidence suggests) a variable sequence that determines specificity in gene-for-gene systems. Second, there is a conserved sequence that seems to be involved in processes that cause the pathogen to be turned on to produce an elicitor even in some nonhost plants. Third, there are sequences that determine the tertiary structure of the coded protein and the quaternary structure of protein polymers and copolymers.

In gene-for-gene systems the variable sequence in the host determines a variable sequence in the pathogen, and a pair of resistance genes in the host determines a pair of sequences in the pathogen. This enables the host to force unfavorable epistatic interactions on the pathogen; these interactions