in the parasite and two alleles. They are *Px/Rx, Px/rx, px/Rx* and *px/rx*. The incompatibility specified by *Px/Rx* has to be only great enough to be distinguishable from *Px/rx*. Until recently the three genotypes for compatibility, namely, *Px/rx, px/Rx* and *px/rx* were considered to be identical in host-parasite interactions. It has now been found (4) that *px/Rx* may not give the same degree of compatibility as *Px/rx* or *px/rx*.

Three cultures of *E. graminis* f. sp. *tritici* have now been found which give infection type 4 on wheat plants with either gene *Pm4* or *pm4*. Therefore, by identification, these cultures have the corresponding gene *p4*. One culture (MS-2) has slightly slower primary infection kinetics on plants with *Pm4* than on plants with *pm4*. MS-2 does not have genes for slow growth because it has normal infection kinetics on plants with *pm4*. One culture (MS-3) has an infection efficiency of 40% on plants with *Pm4* but an infection efficiency of 80% on plants with *pm4*. A third culture ($KhxCc^7$) has an infection efficiency of 20% on plants with *Pm4* but 80% on plants with *pm4*. Here then are two traits, slow development of the parasite and lower infection efficiency, which are generally considered to be a part of horizontal, or field, resistance. An increase in the generation time and a reduction in the number of successful infections will certainly affect the rate with which an epidemic will develop. Upon inoculation with culture MS-1 with *P4*, the host gene *Pm4* is a gene for infection type 0. Upon inoculation with culture MS-2, *PM4* is a gene for high infection type but slower development of disease. Upon inoculation with cultures $KhxCc^7$ or MS-3, *Pm4* is a gene for high infection type but with fewer pustules. Segregation of *Pm4* vs *pm4* is easy to see with culture MS-1 but difficult to see with cultures MS-2, $KhxCc^7$, or MS-3. Is *Pm4* a major gene or a minor gene? The culture of *E. graminis* f. sp. *tritici* used would have determined how *Pm4* would have been classified!!

Clearly the phenotype of a host *R* gene is determined by the selection of the culture of the parasite. There is probably no such thing as a host gene for hypersensitivity, or infection type X, or reduced infection efficiency. The infection type observed is the result of the genotypes of both host and parasite.

Careful analysis in several laboratories has continued to accumulate evidence that field resistance (or horizontal resistance, or non-specific resistance, or whatever you want to call it) is controlled by the same kinds of genes, the same kinds of genetic interactions, as genes controlling infection type. Different kinds of techniques are needed to see effects on infection efficiency or slow growth, as compared to effects on infection type, but the basic genetics turns out to be consistent with gene-for-gene interactions as described by Flor.

On theoretical grounds, it has been suggested that not all genes should follow the gene-for-gene relationship (1). But analysis of the naturally occurring variability indicates that essentially all of it does. The results of numerous detailed studies in several laboratories over the past few years has lent greater credence to the statement that *non-specific resistance* (field, horizontal, generalized, etc.) *is that resistance which hasn't yet been shown to be specific.*

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(Professor Ellingboe has beer. at the University of Sydney whilst on sabbatical leave from the Department of Botany and Plant Pathology, Michigan State University, East Lansing, Michigan 48824. The Editor is grateful to Professor Ellingboe for contributing this viewpoint.)

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REGIONAL NEWS

New Zealand Branch

Dr. C. Gardner Shaw, Professor of Forest Pathology, Washington State University, Pullman, Washington is spending August-December at the University of Auckland with Prof. F.J. Newhook and Dr. J.B. Corbin. He is taking part in the teaching programme and taking the opportunity to visit his son Terry Shaw who recently joined the staff of Forest Research Institute, Rotorua and is working on *Armillariella* in pine plantations being established on cut-over native forest.

A.C.T. Branch

Dr. I.A.M. Cruickshank has received the Ruth Allen Award of the American Phytopathological Society for 1975.

The Ruth Allen Award, consisting of a certificate and the income from the Ruth Allen Memorial Fund is given for outstanding contributions to the science of plant pathology. This award is made to "individuals who have made an innovative research contribution that has changed or has the potential to change, the direction of work in any field of plant pathology".

The comprehensive series of studies of the Phytoalexin Research Group under the leadership of Dr. I.A.M. Cruickshank beginning in 1960 provided the primary stimulus for the current interest in the involvement of fungitoxic host-plant compounds, arising as a result of host-parasite interactions, being involved in disease resistance in plants. Important results from Cruickshank's laboratory have included the isolation and identification of such compounds produced by peas and beans and an extensive study on physiological factors affecting their formation and the clarification of the roles they appear to play in the living plant. They have also isolated and identified fungal compounds which are formed during the early stages of fungal growth that may play an important role in the stimulation and control of the formation of the fungitoxic plant defense compounds.

As a direct result ot the work of the Phytoalexin Research Group world-wide interest has occurred in this area of plant pathology. It is now clear that disease resistance in plants is dependent on complex biochemical interactions between the plant and its parasites. A better knowledge of the biosynthesis of the fungitoxic compounds could open the way to the manipulation of plant metabolism through chemotherapy to new and improved methods of disease control through the controlled activation of the defense systems in genetically susceptible varieties of plants. Knowledge derived from this area of research may also provide a physiological basis for selection in plant breeding programs for disease resistance. It may allow selection within genetically resistant progenies for higher degrees of disease resistance than is possible by present selection methods.