differences between the two treatments of humidity decreased with increase of day temperature from 10 to 30°C.

It was found that these patterns of epidemic development derived from interactions between dispersal and survival of dispersed sporangia under various conditions of temperature and humidity. Dispersal was many times higher under the low than under the high day humidities; survival of dispersed sporangia was better under humid conditions. The effect of these two contrasting phenomena on epidemic development depended on the prevailing temperature. Until day maxima reached 25°C the adverse effect of low humidity on survival was not very important and the survival factor was not critical. The epidemics were then chiefly dominated by the factor of abundant spore dispersal. Only at daily maxima of 30°C did survival become the critical factor obliterating the advantages of abundant dispersal.

This case demonstrated the occurrence of two ecological peaks in late blight development: the higher one under continuous moisture, and the lowest one under dry rather than humid conditions. The continuous moisture peak is probably typical of very wet areas where all the stages of the pathogen's life cycle meet nearly optimal conditions. Dispersal by means of splashing is followed by germination and infection and some sub-optimal factor in the environment need not be compensated by another. The lower peak is probably typical of semi-arid areas in which blight develops owing to phenomena of compensation. At different temperatures either dispersal or survival of dispersed sporangia plays the dominant role and compensates for a sub-optimal level of another factor (10).

B. Powdery mildew on squash. Powdery mildews are probably the most typical pathogens in dry habitats, because infection and sporulation do not need external moisture. However, powdery mildews are also abundant in wet habitats (5). To understand some of the reasons enabling their world-wide distribution, epidemics of *Sphaerotheca fuliginea* on squash were induced in growth chambers (7). They proceeded somewhat better under dry than under humid conditions, but the differences were rather small. These results became understandable when isolated events in the pathogen's life cycle were studied. It was found that dryness favoured sporulation and dispersal, but both processes occurred under a wide range of conditions. High humidity favoured spore survival and infection but these processes too proceeded quite well in dryness (&). Since neither process was limited to a narrow range of meteorological factors there was nothing to compensate for, while the wide range of environmental conditions in which these processes occur confers an obvious advantage on the pathogen, allowing a world-wide distribution of this disease.

This presentation is an attempt to demonstrate the importance of some environmental and biological factors affecting disease development in sub-tropical and semi-arid conditions. In both, the environmental factor has received greater attention in laboratory and field studies. The interactions between environmental and biological factors were tested in the laboratory only and in too few cases for a general rule to be formulated. Further discoveries of interactions like these may lead to a conclusion that there is more than one ecological pathway leading to disease development, that these are rather flexible ways with some alternative routes and that each factor important in disease development must be evaluated not only in relation to the absolute effects, but also in relation to its potential interaction with all other factors.

J. Rotem
Department of Plant Pathology,
Waite Agricultural Research Institute,
Glen Osmond, S.A. 5064

(Dr. Rotem has been visiting the University of Adelaide on sabbatical leave. He is Senior Plant Pathologist in the Division of Plant Pathology, Volcani Center, Bet Dagan, Israel and Professor in the Department of Life Sciences, Bar Ilan University, Ramat Gan, Israel. The Editor is grateful to Dr. Rotem for contributing this invited paper.)

REFERENCES

(1) Bashi, Esther and Rotem, J. (1974) — Adaptation of four pathogens to semi-arid habitats as conditioned by penetration rate and germinating spore survival. *Phytopathology* **64**: 1035-1039.

(2) Bashi, Esther, Rotem, J. and Putter, J. (1973) — The effect of wetting duration, and of other environmental factors, on the development of *Stemphylium botryosum* f. sp. *lycopersici* in tomatoes. *Phytoparasitica* **1**: 87-94.

(3) Cohen, Y. and Rotem, J. (1971) — Dispersal and viability of sporangia of *Pseudoperonospora cubensis*. *Trans. Br. Mycol. Soc.* **57**: 67-74.

(4) Crosier, W. (1934) — Studies in the biology of *Phytophthora infestans* (Mont.) de Bary. *N.Y. Agric. Exp. Stn. Mem.* **153**.

(5) Hirota, K. (1966) — Host range and geographical distribution of the powdery mildews. *Niigata University, Japan.*

(6) Palti, J. and Rotem, J. (1973) — Epidemiological limitations to the forecasting of downy mildews and late blight in Israel. *Phytoparasitica* **2**: 119-126.

(7) Reuveni, R. and Rotem, J. (1974) — The effect of humidity on epidemiological patterns of the powdery mildew *(Sphaerotheca fuliginea)* on squash. *Phytoparasitica* **2**: 25-33.

(8) Rotem, J. (1968) — Thermo-xerophytic properties of *Alternaria porri* f. sp. *solani*. *Phytopathology* **58**: 1284-1287.

(9) Rotem, J. and Ben-Joseph, J. (1970) — Evaporation rate as an indicator for potato late blight development in plots of different foliage density. *Plant Dis. Reptr* **54**: 768-771.

(10) Rotem, J. and Cohen, Y. (1974) — Epidemiological patterns of *Phytophthora infestans* under semi-arid conditions. *Phytopathology* **64**: 711-714.

(11) Rotem, J., Cohen, Y. and Putter, J. (1971) — Relativity of limiting and optimum inoculum loads, wetting durations, and temperatures for infection of *Phytophthora infestans*. *Phytopathology* **61**: 275-278.

(12) Rotem, J. and Palti, J. (1969) — Irrigation and plant diseases. *Ann. Rev. Phytopathology* **7**: 267-288.

(13) Rotem, J., Palit, J. and Lomas, J. (1970) — Effects of sprinkler irrigation at various times of the day on development of potato late blight. *Phytopathology* **60**: 839-843.

Horizontal Resistance : an Artifact of Experimental Procedure?

Flor (2,3) was the first one to do a detailed, quite comprehensive analysis of both the inheritance of resistance and susceptibility in plants to a given disease and the inheritance of virulence and avirulence in the parasite. Flor found that a gene in flax could give resistance only if there was a complementary gene for avirulence in the flax rust fungus. If the parasite lacked the gene for avirulence, the host gene could not confer resistance. If the host gene was for susceptibility, whether the pathogen had the corresponding gene for avirulence or virulence was inconsequential. The host plant was susceptible or, one could say, the pathogen was virulent. A plant with gene *R1* will be resistant only to parasite strains with gene *P1*. A plant with the gene *R2* will be resistant only to parasite strains with *P2*. A plant with genes *R1 r2* will be resistant to parasite strains with *P1 P2* or *P1 p2* but not to strains with *p1 P2* or *p1 p2*. *R1*, therefore, recognizes *P1* and not *p2*. A plant with genes *r1 R2* will be resistant to parasite

strains with *P1 P2* or *p1 P2* but not to strains with *P1 p2* or *p1 p2*. *R2*, therefore, recognizes *P2* but not *P1*. The specificity of the mutual recognition between host and parasite led Flor to formulate the gene-for-gene theory. The necessity for mutual recognition in determining the interactions between host and parasite makes it necessary to give both parasite and host genotypes in discussions of host-parasite interactions. Parasite/host genotype *P1/R1* specifies incompatibility between host and parasite (host resistant and parasite avirulent). Parasite/host genotypes *P1/r1*, *p1/R1* and *p1/r1* specify compatibility. The simplest interpretation of the data presented by Flor is that specific mutual recognition is for incompatible relationships between host and parasite.

It has taken about three decades for plant pathologists and breeders to realize that the gene-for-gene relationship isn't just a peculiarity of the rust diseases of plants, or the obligate parasites of plants, or the predominantly obligate parasites of plants, or the fungal diseases of plants, or fungal and bacterial parasites of plants or fungal, bacterial and insect parasites of plants, or just parasites of plants. There is reason to believe that the gene-for-gene relationship holds for some parasites and their animal hosts, e.g., the scrapie disease of sheep.

Flor identified the *R* genes in flax based on the effect on infection type following inoculation with selected races of the rust fungus. He used the system of recording infection type, a set number of days after inoculation, developed by Stakman *et. al.* (5) for wheat stem rust. Plants with infection types 0, 0;, 1 and 2 are considered to be resistant and plants with infection types 3 and 4 are considered to be susceptible. The usual basis for distinguishing between resistance and susceptibility is the correlation with yield of the economically important plant part under conditions of an induced epidemic. Plants which give infection types 0, 0,, 1 or 2 with the strains of parasites used for inoculation usually do not suffer a great loss in yield when grown in a disease nursery where an epidemic was created. Plants which give infection types 3 or 4 with the strains used for inoculation usually suffer a significant loss in yield when grown in similar conditions. The test for resistance, therefore, is the ability to survive and yield under conditions of an epidemic.

The selection for resistance under conditions of a created epidemic does not allow for the detection of genes which affect the rate with which an epidemic can develop on a given plant genotype. There are many reports of cultivars of various plant species which suffer great losses in yield if grown under conditions of an epidemic, but when grown as commercial crops do not seem to get as much disease. Cultivars which support the slow build-up of disease are considered to have "field resistance".

Van der Plank (6) divided resistance into two categories, namely, vertical and horizontal resistance. Vertical resistance was considered to be controlled by a limited number of genes and to be specific for given strains of the parasite. Vertical resistance follows the gene-for-gene relationship. Horizontal resistance was considered to be controlled by many genes (i.e. polygenic inheritance) and to be effective against all strains of the parasite. Because vertical resistance was controlled by few genes, it was considered to be undependable because mutations at only a few genes, the complementary genes, in the parasite would render the resistance ineffective. Horizontal resistance was considered to be dependable because it didn't select for mutations at complementary loci in the parasite. The many genes in the host were considered to be effective against the parasite species.

The idea of a stable form of disease resistance stimulated a tremendous amount of interest amongst plant pathologists and breeders involved in practical problems of disease control through breeding. There appeared to be a Utopian solution to plant disease problems, even if selection for horizontal resistance was very difficult and inheritance a quantitative trait. Many programs were started where a major effort was to get away from breeding for vertical resistance and to breed for horizontal resistance. Identification of races of parasites lost priority. The concept of horizontal resistance was accepted as proven fact.

The gene-for-gene theory and the pattern that emerges from gene-for-gene interactions has been very useful for studies on host-parasite interactions. The simplest interpretation is that specific mutual recognition occurs for incompatible relationships (1). Compatible relationships are the result of lack of recognition. The possibility, therefore, of getting a handle on genes which affect host-parasite interactions which do not follow the gene-for-gene relationship is very tantalizing. It would mean getting hold of systems that affect interactions that are of a different kind from what has been worked with before.

A number of years ago Dr. E. Everson made available to me segregating generations of a number of crosses involving the wheat cultivar 'Genesee'. Genesee is considered to be susceptible to both powdery mildew and leaf rust based on infection type following inoculation, but, in commercial fields, epidemics of both powdery mildew and leaf rust seem to increase more slowly than on many other varieties. F_2 seedlings from a cross of Genesee with a variety in which the epidemic builds rapidly in the field were inoculated with culture MS-1 of *Erysiphe graminis* f. sp. *tritici*. If the inoculated plants were kept in the glasshouse the distribution of plants with different amounts of mildew was such that one could only conclude that development of mildew, as evaluated in each cross, was controlled by a large number of genes. In other words, continuous segregation was observed in the F_2 population. When a F_2 population was held in controlled environment after inoculation, discontinuous segregation was observed. One fourth of the F_2 population had abundant mildew at seven days after inoculation, and three fourths had the amount of mildew at seven days that would be expected at six and one half days. The mildew developed slowly on three fourths of the progeny. The mildew developed on one fourth of the progeny at the rate expected for a typical susceptible cultivar. Plants could be classified into two discrete classes based on the rate of development of mildew. The differences were the most dramatic at four days after inoculation. One fourth of the plants had macroscopically visible symptoms, three fourths had no macroscopically visible symptoms. The secregation ratio of three slow mildewing to one fast mildewing was consistant with a hypothesis of one dominant gene for slow mildewing. Slow mildewing, therefore, was apparently controlled by one gene, not many as the experiments in the glasshouse had indicated.

One of the characteristics of horizontal resistance is that it is supposed to be effective against all strains of a pathogen. However, one isolate of *E. graminis* f. sp. *tritici* was found which developed as rapidly on Genesee as on other varieties, even under controlled environmental conditions. These results show that the number, and specificity, of genes postulated to explain slow mildewing was dependent on the ability to control the environment sufficiently so that genotypic differences could be observed. Slow mildewing apparently follows the gene-for-gene relationship! (One aspect of this work which has not yet been completed is to show that the gene which gives slow mildewing to Genesee wheat in controlled environment is the same gene which gives slow mildewing to Genesee wheat in the field). Similar results have now been obtained with leaf rust of wheat, stem rust of wheat and stem rust of oats.

Can "major" genes be distinguished from "minor" genes? In the following paragraphs I want to give the results of one analysis of this question. The gene *Pm4* in wheat will illustrate results obtained.

There are four possible parasite/host genotypes with one locus in the host and two alleles and a corresponding locus

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.*

Albert H. Ellingboe,
Department of Plant Pathology and Agricultural Entomology
University of Sydney.
Sydney, N.S.W. 2006

(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.)

REFERENCES

(1) Ellingboe, A.H. — Genetics of host-parasite interactions. Physiological Plant Pathology, R. Heitfuss and P. Williams, ed. (in press).

(2) Flor, H.H. (1946) — Genetics of pathogenicity in *Melampsora lini*. *J. Agr. Res.* **73**: 335-357.

(3) Flor, H.H. (1947) — Inheritance of reaction to rust in flax. *J. Agr. Res.* **74**:241-262.

(4) Martin, T.J., and Ellingboe, A.H. — Differences between compatible parasite/host genotypes involving the *Pm4* locus of wheat and the corresponding genes in *Erysiphe graminis* f. sp. *tritici*. (Submitted to Phytopathology).

(5) Stakman, E.C. and Piemeisel, F.J. (1917) — Biological forms of *Puccinia graminis* on cereals and grasses. *J. Agr. Res.* **10**: 429-496.

(6) Van der Plank, J.E. (1975) — The genetic basis of plant disease epidemics. *Aust. Plant Pathology Soc. Newsletter* **4**: 27-30.

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.