Neth. J. Pl. Path. 75 (1969) 45–52

Pathogenic races, host resistance, and an analysis of pathogenicity

J. E. VAN DER PLANK

Department of Agricultural Technical Services, Pretoria, South Africa

Accepted 31 July, 1968

Abstract

There are two kinds of pathogenic races: those that interact differentially with varieties of the host plant, and those that do not. There are two kinds of resistance in the host: vertical resistance, when varieties of the host interact differentially with races of the pathogen, and horizontal resistance, when differential interaction is absent. There are two kinds of pathogenicity, defined here as virulence and aggressiveness, differentially interacting races differing in virulence, other races in aggressiveness. In relation to strong resistance genes, unnecessary virulence in a race reduces its fitness to survive and, with obligate parasites, also its aggressiveness; i.e. with obligate parasites unnecessary virulence and aggressiveness appear to be negatively correlated.

Introduction

The retirement of an eminent plant pathologist is a fitting occasion for pausing and looking back over the accumulated knowledge in one of his fields of special interest. Professor A. J. P. Oort's retirement seems a particularly appropriate opportunity for looking at host–pathogen relations, a topic in which he has had a strong and productive interest. As a facet of this we may choose the relation between races of the pathogen and varieties of the host. Both pathogen and host vary genetically, and the relation between races and varieties is a cardinal part of plant pathology.

It is now more than 70 years ago that Eriksson (1894) drew attention to formae speciales within a species of a pathogen. Thus, the formae speciales *tritici* and *avenae* of *Puccinia graminis* cause stem rust in wheat and oats, respectively. Just over 50 years ago Stakman and Piemeisel (1917) went a step further and showed that within a forma specialis there are races that differ in their ability to attack different varieties of a crop. Thus, *P. graminis* f. sp. *tritici* contains a number of races that can be distinguished by the way they attack different varieties of wheat. Since then a great number of races within both species and formae speciales of the pathogen have been described, and a pattern of the relation between races of the pathogen and varieties of the host has begun to emerge.

Two kinds of pathogenic races

There are races of the pathogen that interact differentially with varieties of the host, and races that do not. They are distinct.

Races (0), (1) and (2) of *Phytophthora infestans* are examples of differentially interacting races. Race (0) can attack only potato varieties without *R* genes; race (1) can attack varieties without an *R* gene and varieties with the gene R_1, but not varieties with

the gene R_2; race (2) can attack varieties without an R gene and varieties with the gene R_2, but not varieties with the gene R_1.

To give just one other example, race 1 of *Fusarium oxysporum* f. sp. *lycopersici* can attack varieties of *Lycopersicum esculentum* but not varieties derived from *L. pimpinellifolium* accession 160. Race 2 can attack both *L. esculentum* and derivatives of *L. pimpinellifolium* accession 160, but not some of *L. peruvianum*.

The test for differentially interacting races is that it is always necessary to have more than one host genotype to identify them fully. "Differential varieties" are now common tools for identification.

As examples of races that do not interact differentially, we can again use *Phytophthora infestans* and *Fusarium oxysporum* f. sp. *lycopersici*.

Paxman (1963) set out to determine whether races of *P. infestans* would become specially adapted to a potato variety if they were grown continuously on it. He used varieties without R genes. From a naturally infected tuber of the variety Red Skin he isolated a race which he called 30RS; from a naturally infected tuber of the variety Kerr's Pink he obtained a race 31KP; and from a naturally infected tuber of the variety King Edward he obtained a race 32KE. He subcultured these races, each on its original variety, i.e. he subcultured 30RS on Red Skin, 31KP on Kerr's Pink, and 32KE on King Edward. He started to make his tests after 90 cycles of subculturing, i.e. race 30RS had been on Red Skin for 90 cycles plus the unknown number of cycles it had been on Red Skin before it was isolated. As the criterion in his tests he used the rate of spread of mycelium in tuber tissue. He estimated the rate of spread of each of the three races not only in the variety of origin, e.g. 30RS in Red Skin, but also in the other two varieties, e.g. 30RS in Kerr's Pink and King Edward as well. He also tested a fourth race, of unspecified origin. The analysis of variance showed that there was a highly significant difference between the races, i.e. he was dealing with clearly different races of *P. infestans* as measured by their ability to spread through tuber tissue. The analysis also showed that there was a highly significant difference between the three varieties, i.e. Red Skin, Kerr's Pink and King Edward differed clearly in their resistance to the spread of mycelium in their tubers. But there was no evidence of a significant interaction, races × varieties. Judged by the criterion he used, races had not been trained to become specially adapted to the varieties; there was no differential interaction between pathogenic races and host varieties.

Table 1. The reaction between races of *Fusarium oxysporum* f. sp. *lycopersici* and the tomato varieties Bonny Best and Marglobe (data of Wellman and Blaisdell, 1940)

Growth of race in culture	*Reaction severity**		*Pathogenicity rank*	
	Bonny Best	*Marglobe*	*Bonny Best*	*Marglobe*
Raised fluffy	10.4	7.5	1	1
Raised sclerotial	8.7	6.3	2	2
Intermediate raised	8.3	4.5	3	3
Intermediate appressed	6.3	4.1	4	4
Appressed, slimy	4.7	3.0	5	5

* Scale in which 0 = no disease and 15 = severe disease

Tabel 1. Reactie tussen fysio's van Fusarium oxysporum f. sp. lycopersici en de tomatevarieteiten Bonny Best en Marglobe (gegevens ontleend aan Wellman and Blaisdell, 1940)

Wellman and Blaisdell (1940) collected races of *Fusarium oxysporum* f. sp. *lycopersici* in tomato fields over a wide area of the United States, and grouped them according to their habit of growth in culture. The growth varied from raised and fluffy at the one extreme to appressed and slimy at the other. These races they tested against the tomato varieties Bonny Best, which is very susceptible, and Marglobe, which has some resistance. Their findings, reproduced in Table 1, were recorded on an arbitrary scale in which 0 means no infection and 15 means that all the plants were killed while they were still young. Disease varied from a high average of 10.4 for fluffy races on Bonny Best to a low of 3.0 for slimy races on Marglobe. The races varied markedly from one another, the fluffy races being the most pathogenic on both varieties, and the slimy races being the least. The two varieties differed markedly in susceptibility. But there was no evidence of any differential interaction between races and varieties. The races all followed the same ranking for pathogenicity on the two varieties; the two varieties followed the same ranking for susceptibility to all the races, Bonny Best being always the more susceptible.

All the races collected by Wellman and Blaisdell almost certainly belonged to race 1 in the classification based on differential interaction, i.e. they were almost certainly unable to attack derivatives of *L. pimpinellifolium* accession 160. At the time these races were collected there were no commercial tomato varieties derived from *L. pimpinellifolium* accession 160. Race 2 was not found in commercial tomato fields until 1960 (although it had been found earlier in a glasshouse and experimental plots). The classification of races not differentially interacting is independent of the differential interaction of another set of races.

Races that cannot be differentiated by their interaction with different varieties have sufficiently distinctive reactions on a single variety to be distinguished thereby. The five races in Table 1 could have been distinguished by their reaction on either Bonny Best alone or Marglobe alone.

Two kinds of resistance in the host

Corresponding with the two kinds of pathogenic races are two kinds of host resistance. Differential interaction between races of the pathogen and varieties of the host concerns the host as much as it does the pathogen. The host has differentially interacting resistance (or susceptibility) and we call this vertical resistance. A vertically resistant variety has greater resistance relatively to some races of the pathogen than to others. A potato variety with the gene R_1 is vertically resistant to race (0) or (2) of *Phytophthora infestans*, but not to any race with the number 1 in its designation. A variety with the gene R_2 is vertically resistant to race (0) or (1), but not to any race with the number 2 in its designation. Differentially interacting races and vertical resistance in the host are two sides to the same phenomenon, and we use one term or the other according to whether we are stressing the pathogen or the host.

When races of the pathogen do not interact differentially with varieties of the host, the varieties, if they differ, differ in horizontal resistance. Horizontal resistance is spread evenly against all races. The tomato variety Marglobe is more resistant than Bonny Best to all races, be they fluffy or slimy or intermediate in culture.

If the theory of van der Plank (1968, chapter 11) is correct, the number of varieties capable of differing in (polygenic) horizontal resistance to a pathogen is practically limitless.

Two kinds of pathogenicity

It is proposed, as a convention, that when races of the pathogen and varieties of the host interact differentially the races be said to differ in virulence. When they do not interact differentially, it is proposed that they be said to differ in aggressiveness. Thus, race (1) of *Phytophthora infestans* is more virulent than race (0); fluffy races of *Fusarium oxysporum* f. sp. *lycopersici* are more aggressive than slimy races.
There is no known evidence for a positive correlation between virulence and aggressiveness, and the two concepts of pathogenicity must be kept separate.

Alternative names

To emphasize these relations one can use the names "differential resistance" for vertical resistance, "uniform resistance" for horizontal resistance, "differential pathogenicity" for virulence, "uniform pathogenicity" for aggressiveness, and "differential races" and "uniform races" for the two kinds of races. Thus one can write, e.g. that the tomato variety Marglobe is uniformly more resistant to fusarium wilt than the variety Bonny Best, and that fluffy races of *F. oxysporum* f. sp. *lycopersici* are uniformly more pathogenic than slimy races.

A negative correlation between virulence and the fitness of a race to survive; the relation between the fitness of a race to survive and its aggressiveness

Mexico is probably the home of *Solanum demissum*, the species from which all *R* genes for blight resistance used by potato breeders have been derived. It is probably also the home of *Phytophthora infestans*, which reproduces itself there both sexually and asexually, and occurs as abundant races.
Graham et al. (1959) surveyed the races of *P. infestans* on *S. demissum* and *S. demissum* × *S. tuberosum* hybrids. More than 50 isolates were studied, and the majority were found to be of races (1,2,4) and (1,2,3,4). On *S. demissum* and its hybrids virulent races abound; the races have to be highly virulent if they are to survive on *S. demissum*

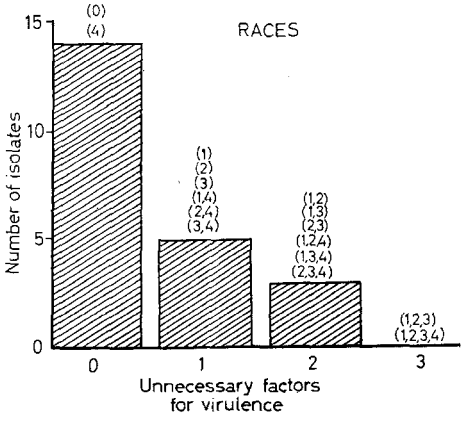

Fig. 1. The abundance of races of *Phytophthora infestans* on Criolla varieties of potato in Mexico. The races are grouped according to their ability to match the resistance genes R_1, R_2 and R_3. The Criolla varieties have no *R* genes. From the data of Graham et al. (1959).

Fig. 1. Het voorkomen van fysio's van Phytophthora infestans op Criolla aardappelrassen in Mexico. De fysio's zijn gerangschikt naar hun vermogen de resistentiegenen R_1, R_2 en R_3 te doorbreken. Ontleend aan gegevens uit Graham et al. (1959).

with its usually many *R* genes. *S. tuberosum* is not native to Mexico, but was introduced by man centuries ago into the environment of *S. demissum* and its attendant virulent races of *P. infestans*. These old varieties of *S. tuberosum*, the Criolla varieties, like all varieties of true *S. tuberosum*, have no *R* genes.

Graham et al. (1959) surveyed the races of *P. infestans* on Criolla varieties. Their results are reproduced in Fig. 1. Races are grouped according to their virulence with respect to the genes R_1, R_2 and R_3. The commonest races found by Graham et al. were (0) and (4). These cannot attack any variety with the genes R_1, R_2 or R_3. They are therefore shown as having no unnecessary virulence, i.e. no more virulence than is needed to attack varieties such as the Criolla varieties without the genes R_1, R_2 or R_3. Next commonest was the group of races, (1), (2), (3), (1,4), (2,4) and (3,4). Each of these races can match one of the genes R_1, R_2 and R_3 and on varieties such as the Criolla varieties without these genes can be said to have one unnecessary factor for virulence. Next commonest as a group were races (1,2), (1,3), (2,3), (1,2,4), (1,3,4) and (2,3,4) each of which can match two of the genes R_1, R_2 and R_3 and can be said to have two factors for virulence unnecessary for attacking the Criolla varieties. Least common were races (1,2,3) and (1,2,3,4), which can be said to have three unnecessary factors for virulence. The greater the number of unnecessary factors for virulence, the less fit the race was to survice; and because the association between *P. infestans* and the Criolla varieties is an ancient one, it can be assumed that the relation between unnecessary virulence and fitness to survive is no mere transient phase.

Other, and more extensive, surveys of races of *P. infestans* lead to the same conclusion that unnecessary virulence reduces fitness to survive. Graham et al. (1959), in their surveys of *Phytophthora infestans* in Mexico, found much the same pattern in European and United States potato cultivars without *R* genes as they did in the Criolla varieties. Schick et al. (1958) identified the races to which 209 isolates from potatoes without *R* genes from West Germany belonged; the most abundant were those that could not overcome the resistance of any of the genes R_1, R_2 and R_3, and abundance fell off with increasing virulence. Graham (1955) surveyed the blight races in Canada in 1952 and 1953, and found on varieties without *R* genes only races (0) and (4). Frandsen (1956) found that of 34 isolates from Lower Saxony and Schleswig-Holstein only one could attack the genotype R_1 and none the genotypes R_2 and R_3. The evidence in the literature seems to be entirely consistent: when *P. infestans* is isolated from ordinary commercial potato fields without *R* genes (this detail is essential), races such as (1,2,3) with much unnecessary virulence are relatively scarce, and in general the abundance of a race decreases as its unnecessary virulence increases.

Similar results have been obtained with *Puccinia graminis* on wheat. Unnecessary virulence, especially in relation to the resistance genes Sr_6 and Sr_{11}, reduces the fitness of a race to survive (Watson, 1958; van der Plank, 1968).

In the analysis of races of *Phytophthora infestans* a distinction was drawn between the genes R_1, R_2 and R_3 on the one hand an R_4 on the other. There is no evidence that unnecessary virulence in relation to R_4 greatly reduces the fitness of the race to survive, i.e. race (4) seems to be as fit as race (0) to survive on varieties without an *R* gene. The genes R_1, R_2 and R_3 are "strong" and R_4 is "weak". This distinction has long been apparent in the results of potato breeders. Each of the genes R_1, R_2 and R_3 has been used to provide commercial potato varieties with resistance to blight, but there is no example of a potato variety with successful blight resistance based on R_4 alone. (The

Table 2. The number of pustules formed on three wheat varieties by two races of *Puccinia graminis* f. sp. *tritici* (data of Katsuya and Green, 1967)

Race	*Number of pustules**			*Pathogenicity rank*		
	Little Club	*Marquis*	*Red Bobs*	*Little Club*	*Marquis*	*Red Bobs*
56	6292	4374	3594	1	1	1
15 *B-1*	5448	3065	1646	2	2	2

* Totals for two experiments and three temperatures: 15°, 20° and 25°C

Tabel 2. Aantal sporehoopjes gevormd op drie tarwerassen door twee fysio's van Puccinia graminis f. sp. tritici (gegevens ontleend aan Katsuya and Green, 1967)

gene R_4 has occasionally been used by breeders, but only in combination with other genes, e.g. with R_1 in the potato variety Virginia ,with R_3 in Vertifolia, and with R_1 and R_3 in Greta.) A detailed discussion of strong and weak resistance genes would take us too far off our course.

Unnecessary virulence in relation to strong genes reduces fitness to survive. How is aggressiveness affected? With obligate parasites such as *Puccinia graminis* or near obligate parasites such as *Phytophthora infestans* it seems reasonable to equate aggressiveness with fitness to survive, i.e. it seems reasonable to expect unnecessary virulence to reduce aggressiveness.

Consider the evidence of Katsuya and Green (1967), They compared races 15 *B*-1 (Can.) and 56 of *Puccinia graminis* f. sp. *tritici* on three wheat varieties, Little Club, Marquis and Red Bobs, all of which are susceptible vertically to the two races. Race 15 *B*-1 is virulent on several varieties which race 56 cannot attack: Arnautka, Mindum, Einkorn, Vernal and, especially, derivatives of the varieties Hope and *H* 44 which played a dominant part in the red spring wheat of North America during the 1940's and early 1950's. On varieties vertically susceptible to both, race 15 *B*-1 has more unnecessary virulence than race 56, and, accordingly, its fitness to survive is less. This is brought out clearly by the analyses of Browder (1966) of races collected from susceptible wheats in Kansas.

Katsuya and Green determined the number of pustules developed on seedlings sprayed with about equal amounts of suspensions containing 20 mg of spores of each race in 50 ml. Table 2 shows that race 56 produced more pustules on each of the three varieties. Also, against both races the varieties followed the same ranking for resistance: Little Club the least resistant, Marquis next, and Red Bobs the most resistant. On these tests of rank, there is no evidence of a differential interaction between pathogenic races and host varieties, and the difference between the two races on the three varieties is therefore primarily one of aggressiveness[1]. This is borne out by the general evidence with wheat stem rust, maize rust and potato blight that resistance to the initiation of lesions is horizontal.

[1] Table 2 gives pooled totals. In detail results were consistent except that in experiment 1 at 20°C race 15 *B* produced more pustules than race 56, a result reversed in experiment 2. To turn to another matter, it is suggested for future experiments that it might be worth the trouble to count infection points instead of pustules. Pustules are not a clear linear measure of infection, because, as Petersen (1959) showed, several infection points often go into the making of one pustule, the average number of infection points per pustule increasing with the level of disease.

Katsuya and Green also measured the weight of spores produced per uredium. The results were as before: race 56 produced more spores than race 15 *B*-1 on all three varieties, and with both races uredia on Little Club produced the most spores and Red Bobs the least. Again, there was no evidence of differential interaction between races of the pathogen and varieties of the host, and differences in pathogenicity were primarily differences in aggressiveness.
To summarize, race 15 *B*-1 has more virulence than race 56, which allows it to attack varieties such as Hope which race 56 cannot attack, but it has less aggressiveness on varieties such as Little Club, Marquis or Red Bobs which are susceptible to both races. In gaining the virulence needed to overcome the resistance of Hope the pathogen lost aggressiveness on susceptible varieties. This is the essence of vertical resistance given by strong genes: it makes the pathogen lose aggressiveness as it acquires virulence. This is in contrast with horizontal resistance, which by definition the pathogen can match only if it increases aggressiveness on all varieties, susceptible as well as resistant. All this is for obligate parasites. With non-obligate parasites that survive largely as saprophytes one cannot necessarily equate fitness to survive with aggressiveness, because their fitness may be determined in the saprophytic phase. There is however evidence that with non-obligate parasites too unnecessary virulence reduces fitness to survive (van der Plank, 1968).

Samenvatting

Pathogene fysio's, resistentie van de waardplant en een analyse van pathogeniteit

Er zijn twee soorten van pathogene fysio's: die welke interactie vertonen met rassen van de waardplant, en die welke dat niet doen. Er zijn twee soorten resistentie in de waardplant: verticale resistentie, wanneer rassen van de waardplant verschillend reageren op fysio's van het pathogeen, en horizontale resistentie, wanneer dit niet het geval is. Er zijn twee soorten pathogeniteit, hier omschreven als virulentie en agressiviteit; de eerstgenoemde fysio's verschillen in virulentie, andere fysio's in agressiviteit. In verband met sterke resistentiegenen reduceert onnodige virulentie in een fysio de overlevingskans en, bij obligate parasieten, ook de agressiviteit; dit wil zeggen dat bij obligate parasieten onnodige virulentie en agressiviteit negatief gecorreleerd zijn.

References

Browder, J. E., 1966. A rapid method of assaying pathogenic potential of populations of Puccinia graminis tritici. Pl. Dis. Reptr 50: 673–676.

Eriksson, J., 1894. Über die Specialisierung des parasitismus bei den Getreiderostpilzen. Ber. dt. bot. Ges. 12: 292–331.

Frandsen, N. O., 1956. Rasse 4 von Phytophthora infestans als Feldrasse in Deutschland. Phytopath. Z. 26: 124–130.

Graham, K. M., 1955. Distribution of physiological races of Phytophthora infestans (Mont.) de Bary in Canada. Am. Potato J. 32: 277–282.

Graham, K. M., Niederhauser, J. S. and Romero, S., 1959. Observations on races of Phytophthora infestans in Mexico during 1956–1957. Am. Potato J. 36: 196–203.

Katsuya, K. and Green, G. J., 1967. Reproductive potentials of races 15B and 56 of wheat stem rust. Can. J. Bot. 45: 1077–1091.

Paxman, G. J., 1963. Variation in Phytophthora infestans. Eur. Potato J. 6: 14–23.
Petersen, L. J., 1959. Relations between inoculum density and infection of wheat by uredospores of Puccinia graminis var. tritici. Phytopathology 49: 607–614.
Plank, J. E. van der, 1968. Disease resistance in plants. Acad. Press, New York, 206 pp.
Schick, R., Schick, E. and Haussdörfer, M., 1958. Ein Beitrag zur physiologischen Specialisierung von Phytophthora infestans. Phytopath. Z. 31: 225–236.
Stakman, E. C. and Piemeisel, F. J., 1917. Biologic forms of Puccinia graminis on cereals and grasses. J. agric. Res. 10: 429–495.
Watson, I. A., 1958. The present status of breeding disease resistant wheats in Australia. Agric. Gaz. N.S.W. 69: 630–660.
Wellman, F. L. and Blaisdell, D. J., 1940. Differences in growth characters and pathogenicity of Fusarium wilt isolations tested on three Tomato varieties. Tech. Bull. U.S. Dep. Agric. 705: 28 pp.