Cytological and Histology

Cytological Comparison of Specific (R3) and General Resistance to Late Blight in Potato Leaf Tissue

René Gees and Hans R. Hohl

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ABSTRACT


A cytological comparison was made of several potato cultivars inoculated with the late blight fungus Phytophthora infestans to study possible differences in phenotypic expression of general and specific resistance genes. The host cultivars used had different levels of general resistance and, additionally, differed by the presence or absence of the specific resistance gene R3. It was noted that: 1) penetration frequency did not differ among the cultivar-pathogen combinations; 2) up to 90% of the penetrations of the leaf tissue occurred in the stomata complex; 3) the time required for lesions to start expanding tended to increase in cultivars containing the R3 gene; 4) speed of tissue colonization following this latent period was fairly uniform in all cultivars where growth beyond the inoculation site occurred; 5) all cells initially penetrated by the pathogen turned brown during incompatible interactions, 31-98% turned brown during compatible interactions; 6) sporulation expressed as spores of sporangia formed per unit area of infected tissue was similar in all cultivars; and 7) high general resistance may have a phenotype indistinguishable from a hypersensitive reaction evoked by specific resistance genes.

Additional key words: horizontal resistance, vertical resistance.

MATERIALS AND METHODS

Cultivars of Solanum tuberosum L. with and without the R3 gene were used. They differed in their level of general resistance as recorded in the 1979 index of European potato varieties (31), where 1 denotes the highest and 9 the lowest level of resistance. In practice, 1-3 may be considered as high, 7-9 as very low levels of resistance. In this paper, the first number after the name of the cultivar indicates the level of general resistance of the leaf tissue, while the presence or absence of the R3 gene is indicated by “3” and “r,” respectively; Alpha 3/r, Erntestolz 3/r, and Tasso 4/r have no specific resistance gene but a high level of general resistance. Dekama 2/3 and Eba 3/3 possess the R3 gene and a high level of general resistance; Pentland Beauty 7/3 and the R3 differential (obtained from L. J. Turkensteen, Wageningen, Holland) have the R3 gene but low general resistance. Finally, Holde 7/r and Bintje 7/r have neither an R gene nor appreciable levels of general resistance. The expected field disease reactions are shown in Table 1.

The potato plants were grown in pots in the greenhouse during the winter months and in the field during the growing season. Seven to 10 wk after planting, the fourth to sixth leaf below the youngest fully expanded leaf was excised and used. Only the terminal leaflet and the adjoining lateral pair of leaflets were used. The leaflets were separated from each other, surface-sterilized by immersion in a 1% aqueous sodium hypochlorite solution, and rinsed several times in sterile water. They were placed on moistened filter paper in sterile plastic petri dishes. The surfaces of the leaflets were carefully dried in a laminar flow sterile bench with the lids of the petri dishes temporarily removed.

Two races of the pathogen P. infestans (Mont.) de Bary were used: strain 134, a complex race (with known virulence genes 1, 2, 3, 4, 7, and 8, collected in Costa Rica by the second author) compatible with all potato cultivars used, and strain 193 (race 0, without known virulence genes, obtained from L. C. Turkensteen, Wageningen, Holland) incompatible with cultivars possessing R genes. The isolates were grown on rye-dextrose agar for 10-14 days at 16 C. Occasionally they were inoculated into potato tuber tissue and reisolated. For inoculation, sporangial suspensions in sterile deionized distilled water (SDDW) were incubated 2-3 hr at about 4 C in a refrigerator to release zoospores. The concentration of the zoospore suspension was then adjusted using a hemacytometer.
For controls, SDDW was used alone.

One- or two-drop inoculations were made on each leaflet in a central intercostal field on either side of the midrib. To prevent the inoculation drop from spreading, it was confined within a thin ring of sterile petroleum jelly. The petroleum jelly was applied using the blunt end of a glass tube with an internal diameter of 5.4 mm. No ring was needed on the lower surface because the inoculation drop was held in place by surface tension alone.

The spread of the infection was measured daily using a dissecting microscope and transmitting light. Because the lesions developed in a circular fashion, the radius of the necrotized area parallel to the long axis of the leaflet was measured and used as the parameter for lesion spread.

Sporulation was assessed after 6 days. Infected and sporulating areas were determined planimetrically. The sporangia were washed from both sides of the leaflet and their concentration was determined in a hemacytometer.

A staining and clearing method was used to study infection 24 hr after inoculation. The technique originally described by Shipston and Brown (30) has been modified by Heath (11) and by Wilson and Coffey (39). After rinsing in distilled water, leaf disks 4 mm in diameter were treated with 0.01% of aniline blue in 0.067 M K2PO4·3H2O of pH 12. The disks were examined in a Zeiss photomicroscope II equipped with epifluorescence (exciter filter BP 405/6, interference beam splitter FT 425, and barrier filter LP 435) and a high-pressure mercury vapor lamp (HBO 50 W). Photographs were taken on Kodak Technical Pan 2415 and Kodak Plus X Pan 35 mm film.

For scanning electron microscopy, tissue 24 hr following inoculation was fixed for 4 hr in 3% glutaraldehyde in 0.067 M phosphate buffer of pH 6.8 and postfixed in 1% osmium tetroxide overnight. After dehydration in an acetone series, the leaf disks were dried by the critical point method and coated with a layer of gold/palladium. The specimens were examined in a Cambridge S4-10 scanning electron microscope.

All experiments were repeated at least once. The drawings and photographs represent typical situations and examples.

RESULTS

Onset and rate of lesion expansion. After inoculation of the upper leaf surface with race 6, the cultivars with the R3 gene reacted in a hypersensitive fashion as expected. Holde and Binjte, the two varieties without R genes and with low general resistance, developed extensive lesions. The rates of expansion of the lesions were approximately linear (Fig. 1). Ernestolz and Tasso, also without R genes but with high general resistance, reacted hypersensitively and identical to the R3 cultivars by forming small necrotic flecks 24 hr after inoculation with no further disease development. This observation was made from plants grown in the greenhouse as well as in the field. Alpha reacted in this and subsequent experiments similarly to the most susceptible cultivars Binjte and Holde, despite its reported high general resistance. L. C.

Table 1. Reactions expected from inoculation of potato cultivars with two races of the pathogen Phytophthora infestans

<table>
<thead>
<tr>
<th>Cultivar</th>
<th>Genotype</th>
<th>Expected reaction with race 6*</th>
<th>Expected reaction with race 1, 2, 3, 4*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Holde</td>
<td>7/r</td>
<td>S</td>
<td>S</td>
</tr>
<tr>
<td>Binjte</td>
<td>7/r</td>
<td>S</td>
<td>S</td>
</tr>
<tr>
<td>Alpha</td>
<td>3/r</td>
<td>GRb</td>
<td>GRb</td>
</tr>
<tr>
<td>Ernestolz</td>
<td>3/r</td>
<td>GR</td>
<td>GR</td>
</tr>
<tr>
<td>Tasso</td>
<td>4/r</td>
<td>GR</td>
<td>GR</td>
</tr>
<tr>
<td>Pentland Beauty</td>
<td>7/3</td>
<td>R</td>
<td>S</td>
</tr>
<tr>
<td>R3 differential</td>
<td>-/3</td>
<td>R</td>
<td>S</td>
</tr>
<tr>
<td>Eba</td>
<td>3/3</td>
<td>R</td>
<td>GR</td>
</tr>
<tr>
<td>Dekama</td>
<td>2/3</td>
<td>R</td>
<td>R</td>
</tr>
</tbody>
</table>

*GR = Susceptible, S = specific resistance (hypersensitive reaction), GR = high general resistance.

bBehaved as S, see Results.

Turkensteen has reported that Alpha is strongly influenced by environmental conditions in its disease expression (Turkensteen, personal communication).

In the compatible interactions, the rate of expansion with a given inoculum density was not significantly different among the cultivars (Fig. 1). However, with 70 zoospores per square millimeter, lesions started to expand earlier and their rate of expansion was larger compared with the lower inoculum density (15 zoospores per square millimeter).

Inoculations of the lower (abaxial) leaf surface with 45 zoospores per square millimeter of race 0 (Fig. 2) gave results similar to those obtained with the upper leaf surface. The R gene cultivars reacted hypersensitively, and in the compatible reactions the starting time and rates of lesion expansion did not significantly differ from each other. In these experiments only plants from the greenhouse were involved, and cultivars Ernestolz and Tasso were not included in the experiments.

Inoculation of upper leaf surface with the complex race. Figure 3 shows that onset and rates of lesion expansion were similar among the cultivars tested. The results from the compatible interactions obtained following inoculation of the upper leaf surface with race 0 (Fig. 1) also were similar. With the R3 cultivars, lesions 96 hr after inoculation tended to be smaller. However, these differences were not statistically significant (P > 0.05). With plant material from the greenhouse, the degree of homogeneity was even higher, although the rates of lesion expansions were lower and lesions started to expand somewhat later (about 90 hr compared with 65–80 hr in Fig. 3).

The inoculation of the lower leaf surface with the complex race (Fig. 4) resulted in a much reduced starting period but also in a somewhat reduced rate of lesion expansion compared with all other situations described above. The reduced speed of lesion expansion was also observed after inoculation of the upper leaf surface. It may be attributed to the fact that the plants used in this experiment were grown in the greenhouse. Lapwood (16) and Malcolmson (23) made the same general observation.

Production of sporangia on infected leaves. Sporangial densities were determined 6 days after inoculation (Fig. 5). No significant differences were found among the cultivars with either method.

![Fig. 1. Lesion size plotted against time (hpi) after inoculation (top) of upper leaf surfaces with race 6 of Phytophthora infestans. Upper group of lines: 70 zoospores per square millimeter of inoculated leaf surface; lower group of lines: 15 zoospores per square millimeter of inoculated leaf surface. Cultivars with open symbols reacted hypersensitively. Extrapolation of the lines to the time axis gives a rough estimate of the time lapsed before symptoms appeared.](image-url)
The values in Figure 5 are combined counts of the infected leaf areas from both sides of the leaf. The results show that all the infected tissues were produced about the same density of sporangia and that, therefore, the total number of sporangia per leaf was predominantly determined by the area colonized by the pathogen.

**Cytological observations.** The results discussed in this section were from inoculated tissue of the upper leaf surface that had been cleared and stained. Twenty-four hours after depositing the inoculum, about 3–9% of the zoospores were recovered as cysts adhering to the leaf surface. The clearing and staining probably had removed a large portion of nonpenetrating cysts. Of the adhering cysts, 10–70% (0.3–6% of the initial inoculum applied) had started penetration. No correlation could be found between the numbers of adhering cysts and the host-pathogen combination used.

Five different sites of epidermal penetrations were observed: a) stomatal guard cells, b) stomatal opening, c) epidermal cells adjoining the stomatal guard cells, d) other epidermal cells, and e) hair and gland cells. Penetrations of stomatal openings and of hair and gland cells were rarely observed. When the fungus entered the stomatal opening, it usually turned at right angle and penetrated the guard cell.

Most of the penetrations occurred in the stomatal complexes irrespective of the race-cultivar combination used. The relative number of penetrations in these complexes was higher than the relative leaf area they occupied. Penetration frequency of the stomatal complex was 160–370% of the value expected assuming random distribution of penetrations (Table 2). The stomatal complex includes the guard cells and the immediately adjoining epidermal cells (39). Penetrations were preceded by appressorium

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**Fig. 2.** Lesion size plotted against time (hours) after inoculation (hpi) of lower leaf surfaces with race 0 of *Phytophthora infestans*. Cv. Pentland Beauty, Dekama, and the R3 differential reacted hypersensitively (open symbols). Inoculum: 45 zoospores per square millimeter of inoculated leaf area.

**Fig. 3.** Lesion size plotted against time (hours) after inoculation (hpi) of the upper leaf surface with the complex race of the pathogen compatible with all cultivars. Inoculum: 50 zoospores per square millimeter of inoculated leaf area.

**Fig. 4.** Lesion size plotted against time (hours) after inoculation (hpi) of the lower leaf surface with the complex race of the pathogen compatible with all cultivars. Inoculum: 40 zoospores per square millimeter of inoculated leaf area.

**Fig. 5.** Density of sporangia (number per square millimeter of infected leaf area) produced 6 days after inoculation of potato leaflets with *Phytophthora infestans*. Solid bar = complex race; stippled bar = race 0. LSD = Least significant difference with $P > 0.05$. H = Holde, B = Bintje, A = Alpha, Er = Erntestolz, T = Tasso, P = Pentland Beauty, R3 = R3 differential cultivar, D = Dekama, E = Eba.
formation (Figs. 6–9) and occurred preferentially into guard and other epidermal cells from above an anticlinal wall adjoining a neighboring epidermal cell. This site preference is possibly due to the depression furrow present between adjoining epidermal cells (Figs. 6–10).

Incompatible interactions. All the R3 cultivars as well as the r cultivars Tasso and Ernestolz with a high general resistance reacted hypersensitively against race 0 (Figs. 11 and 12). This conclusion is based on microscopic observations and lesion expansion data. The frequency of penetrated cells of these cultivars did not differ from that obtained in the compatible situations. After 24 hr, the hyphae and the wall of all initially penetrated cells had turned brown (Figs. 11 and 12). Usually a thin annular layer of wall appositions surrounded the invaded cells. These appositions formed on the walls of the healthy cells surrounding the necrotic cell (Fig. 12) but sometimes also formed on the wall of the necrotic cell. Even though the pathogen grew well within the invaded cell and formed swollen hyphal branches, it very rarely penetrated a second epidermal cell or a palisade cell. An exception occurred when the pathogen first penetrated a stomatal guard cell. In this case it normally also invaded the neighboring guard cell (Fig. 11).

Compatible interactions. After 24 hr, the fungus had left the original site of infection and invaded additional epidermal and/or palisade cells (Fig. 13). This was the case in all compatible combinations. However, the cultivars differed in the percentage of browning of the first cell penetrated by the pathogen (subsequent invasions of neighboring tissue were not considered). Table 2 shows that Dekama formed the highest percentage of browned cells, followed by the other R3 cultivars. In these cultivars lesion expansion tended to be delayed, yet, in general, this higher degree of browning of the R3 cultivars did not result in an overall higher level of resistance against the complex race as measured by the spread of the lesions (Figs. 3 and 4).

Nonpenetrating germ tubes. Growing over the leaf surface, germ tubes frequently formed appressoria (Figs. 6–8, 14, 15). From these germ tubes often resumed growth to form another appressorium at some distance from the first. These thickenings usually occurred over anticlinal epidermal walls outside the stomatal complex and were frequently accompanied by the formation of penetration pegs (checked at 100 hr after inoculation). The pegs never developed into infection hyphae and were always accompanied by formation of wall appositions and browning of the host walls at the site of attempted penetration (Figs. 14 and 15). These sites were termed “abortive penetrations” (Fig. 16). Abortive penetrations were observed in all cultivar-pathogen combinations and their incidence was not correlated with levels of incompatibility.

<table>
<thead>
<tr>
<th>Cultivar</th>
<th>General-specific resistance</th>
<th>Total penetrations of stomatal complex (%)</th>
<th>Germ tubes penetrating guard cells (%)</th>
<th>Relative area of stomatal complex (%)</th>
<th>Relative penetration frequency (%)</th>
<th>Browed penetrated cells (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Holde</td>
<td>7/r</td>
<td>96</td>
<td>40</td>
<td>29</td>
<td>3.7</td>
<td>34 a</td>
</tr>
<tr>
<td>Binjic</td>
<td>7/r</td>
<td>94</td>
<td>38</td>
<td>50</td>
<td>1.9</td>
<td>35 a</td>
</tr>
<tr>
<td>Alpha</td>
<td>3/r</td>
<td>90</td>
<td>36</td>
<td>40</td>
<td>2.3</td>
<td>31 a</td>
</tr>
<tr>
<td>Ernestolz</td>
<td>3/r</td>
<td>87</td>
<td>50</td>
<td>29</td>
<td>3.0</td>
<td>46 ab</td>
</tr>
<tr>
<td>Tasso</td>
<td>4/r</td>
<td>84</td>
<td>35</td>
<td>52</td>
<td>1.6</td>
<td>53 a</td>
</tr>
<tr>
<td>Pentland Beauty</td>
<td>7/3</td>
<td>86</td>
<td>30</td>
<td>25</td>
<td>3.4</td>
<td>70 bc</td>
</tr>
<tr>
<td>R3 differential</td>
<td>?/3</td>
<td>80</td>
<td>40</td>
<td>33</td>
<td>2.7</td>
<td>86 ed</td>
</tr>
<tr>
<td>Dekama</td>
<td>2/3</td>
<td>89</td>
<td>40</td>
<td>49</td>
<td>1.8</td>
<td>98 d</td>
</tr>
</tbody>
</table>

aThe means are the averages of two experiments including five leaf disks per cultivar. The inoculum consisted of 50 spores per square millimeter of inoculated leaf area. The counts were made 24 hr after inoculation.

bFirst number denotes general resistance (from 1 to 9, with 1 = highest resistance and 9 = lowest resistance); ? = not known but low resistance; /r = no R gene present; /3 = R3 gene present.

cPenetrations to relative surface area of stomatal complexes.

dPercentage of initially penetrated cells which turned brown. Means followed by the same letter are not statistically different (*P = 0.05) according to Duncan's multiple range test.

DISCUSSION

This study deals with rates of lesion expansions and cytological aspects of the infection process obtained with surface-sterilized detached leaves that were inoculated and incubated under standard conditions. This approach is simple and efficient, and it avoids variations introduced by changing biotic and abiotic factors encountered in field and whole-plant experiments. Detached leaves might react differently from those left on the plant. However, when excised leaf assays were used in screening for late blight resistance and in fungicide studies, the results were essentially similar to those obtained in the field (18, 22, 35). Furthermore, many of our results were scored within only 1 or 2 days after the leaves were detached.

The average length of the germlings on the leaf surface did not differ among the various race-cultivar combinations tested, and there was no correlation between the frequency of germ tube penetration and the degree of host incompatibility. Tomiyama et al. (33) and Berggren (2) have reported similar results, indicating that prepenetration and penetration events per se are frequently nondiscriminating factors in specific and general resistance of late blight. However, Wilson and Coffey (39) considered the very low frequency of penetration a major factor of resistance in the highly resistant potato cultivar Pimpernel.

DeBary (5) carefully described penetration of epidermal cells. A newer finding is the increased frequency of penetration of the stomatal complex, a phenomenon noticed by Wilson and Coffey (39) in potatoes attacked by P. infestans. Lin and Edwards (20) and Johnson et al. (14) also recorded this phenomenon with Erysiphe graminis on barley. While Wilson and Coffey (39) observed cultivar-specific differences in this behavior, our data with eight different cultivars revealed no such differences. In all cultivars, roughly 90% of the penetrations occurred in the stomatal complex, which makes up only 25–52% of the upper leaf surface area. This situation is typical for both compatible and incompatible combinations. It was even more pronounced in our study than in the study by Wilson and Coffey (39). The differences might be explained by environmental influences or by the 20- to 60-fold higher inoculum density used by Wilson and Coffey, which perhaps somewhat obscured the expression of this differential trait.

Our observations suggest that the increased rate of penetration of the stomatal complex is not due to a preferential growth of germ tubes towards it but to a more successful attack of these cells by the pathogen. This is indicated by the high number of abortive penetration attempts observed outside of these complexes in both compatible and incompatible situations. In these instances the fungus produced typical appressoria (9) which, however, did not
lead to successful penetration and resulted in a renewed outgrowth of the germ tube from these swellings. Possible differences in the physical surface properties (40) or the composition of the walls might explain why fewer penetrations occurred outside the stoma complex. Stoma complexes generally arise from a common stoma mother cell. A common cell lineage could quite reasonably explain the differential behavior observed. However, differences in partial CO₂ pressure or in pH around the stomata (29) or elevated ion concentrations caused by increased peristomal transpiration (21) might also contribute to successful penetration.

The spread of the lesion beyond the site of inoculation was influenced by two factors: inoculum density and presence of the "overcome" R3 gene. The onset of spreading occurred about 20 hr earlier with 70 than with 15 zoospores per square millimeter (a reduction from 80 to 60 hr after inoculation). Hodgson (12) observed a similar effect of inoculum density on potato leaves and Hächler and Hohl (10) observed it in potato tubers. No apparent influence of inoculum density was observed in the hypersensitive interactions, at least not in the range of inoculum densities tested.

With the infections involving the complex race and cultivars carrying the R3 gene, there was a tendency for a prolonged lag phase before lesions began to expand. In addition, the percentage of first-penetrated cells that browned was higher in cultivars with the R3 gene than without it, even in compatible situations. This could indicate that the R3 gene product, although overcome by the virulence gene 3 of the complex race, was still capable of

Figs. 6-9. 6, Cyst with germ tube forming an appressorial swelling (as) from which a normal-looking hypha emerges, 24 hr after inoculation. (Scanning electron microscope [SEM] preparation, ×1,000) 7, Abortive penetration. The pathogen produces an appressorial swelling (as) but then continues to grow on the host surface. c = Cyst. (SEM preparation, ×1,600) 8, Site of penetration at the edge of a subsidiary cell, 24 hr after inoculation. As in Figure 7, one germ tube swells (as) shortly before forming an appressorium (a). c = Cyst. (SEM preparation, ×840) 9, Penetration of a stoma cell (s), with appressorium (a) and germ tube (gt). (SEM preparation, ×1,600)
Fig. 10. Typical responses of a compatible interaction between Phytophthora infestans and potato leaf tissue, 24 hr following inoculation. A, cv. Alpha and B, cv. Bintje were inoculated with race 0 of the pathogen. The pathogen has spread well beyond the initially infected cell. a = Appressorium, cy = cyst, co = collar surrounding basis of penetrating hyphal peg and brightly fluorescing after aniline blue staining, e = epidermal cell, iv = fungal infection vesicle diffusely fluorescing with aniline blue staining, pc = palisade cell, s = stoma cell, sc = subsidiary stomatal cell, black dots = browned area, hash marks = fluorescing after aniline blue staining.

Fig. 11. Typical responses of incompatible interactions between Phytophthora infestans and potato leaf tissue, 110 hr after inoculation. A, cv. Tasso and B, cv. Erntestolz were inoculated with race 6. In A, fungal growth did not extend beyond the initially penetrated guard cell; in B, the fungus penetrated the subsidiary cell but did not leave it. a = Appressorium, co = collar at penetration site, brightly fluorescing with aniline blue, cy = cyst, gt = germ tube, p = papilla brightly fluorescing with aniline blue, s = stoma cell, sc = subsidiary cell of stomatal complex, black dots = browned areas, hash marks = fluorescing after aniline blue staining.

Figs. 12-15. 12. Hypersensitive reaction of cv. Dekama inoculated with race 0. The guard cell and the adjoining epidermal cell are deeply browned and the neighboring epidermal cells are lined with deposits of aniline blue fluorescing material (wall appositions, evident as bright, undulating rim in the micrograph). Mixed bright field/fluorescence illumination, 110 hr after inoculation. (X325) 13. Compatible interaction of cv. Alpha inoculated with race 0. The penetrated guard cell is only slightly browned. The fungus has invaded the adjoining epidermal cell. a = Appressorium, th = transcellular hypha. (Bright field, X1,000) 14. Abortive penetration. The anticlinal walls of the epidermis below the appressorium (not in focal plane) are browned. The two epidermal cells have marked papillae. e = Epidermal cell, n = cell nucleus, p = papilla. (Bright field, X900) 15. Same as Figure 14 but photographed with combined bright field and fluorescence illumination and aniline blue staining. (X900)
While the host cultivars with the R3 gene reacted hypersensitively against race 0 of the pathogen, as had been expected, the hypersensitive-like reaction displayed by the two cultivars Ertestolz and Tasso (both lacking R genes) was unexpected. However, several authors have observed hypersensitive-like reactions in cultivars carrying no specific resistance genes of several crops (cited in 38) including potato (39). Apparently the hypersensitive reaction is not unique to specific resistance but is a general expression of highly incompatible interactions. Of the traits we and others have studied, such as browning of the cytoplasm and cell walls, formation of wall appositions (papillae [1]), and restriction of the pathogen to the foci of infection and phytotoxin formation (6), all, even rapid cell death, may be found in specific and in general resistance. Conceivably similar biochemical and cellular events form the basis of the close cytological similarity of these reactions in cultivars with and without R genes. However, in the absence of direct evidence, such an interpretation remains essentially speculative.

Our study emphasizes that phenotypic expressions of general and specific resistance in potato leaf tissue against the late blight fungus are not clearly separable on the basis of morphological and cytological criteria. General resistance may lead to situations morophologically and cytologically indistinguishable from hypersensitivity. Furthermore, the presence of specific resistance gene(s) may not necessarily lead to hypersensitivity after challenge by an avirulent pathogen as suggested by Lapwood and McKee (17) and histochemically explored by Häcker and Hohl (10) in potato tuber tissue. In a general context, the hypersensitive reaction would represent one extreme of possible combinations of individual defense reactions potentially present in hosts with either specific or general resistance. The other end would be marked by the absence of any resistance response.

Finally, our results imply a cautionary note for potato breeders. In screening for general resistance to potato blight, the indiscriminate elimination of plants with “hypersensitive” symptoms may possibly lead to loss of valuable breeding material because these plants with seemingly specific resistance R genes might actually carry a very high general resistance.

LITERATURE CITED


