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# INVESTIGATIONS INTO THE INHERITANCE OF THE IMMUNITY FROM PHYTOPHTHORA INFESTANS DE B. OF SOLANUM DEMISSUM LINDL.

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## 1. Introduction

Potato late blight, caused by the fungus *Phytophthora infestans*, is occurring practically all over the world, as is shown by a map in the book of Gäumann (4). According to recent information late blight is also present in the Belgian Congo (1) and on the island of Celebes, which is not yet indicated on this map.

The damage to the potato crop, caused by the disease, is important, ranging from slight to very severe, according to climatic conditions. In the Netherlands reduction in yield caused by foliage attack, loss of attacked tubers and costs of spraying together are estimated at 20 million guilders a year. If blight immune varieties, equalling in yielding ability the present susceptible varieties, could be widely grown, the value of about 13.000 ha (= 32.000 acres) or about $6\frac{1}{2}\%$ of the total acreage under potatoes in the Netherlands might be saved (7). So breeding immune varieties is worth trying.

## 2. Differentiation of phytophthora infestans

In several countries (Germany, U.S.A., Great Britain, U.S.S.R., Java) the breeding of potatoes immune from late blight is endeavoured. The differentiation of the fungus in pathogenetically differing races was an unexpected and unpleasant complication, which turned formerly immune varieties into susceptible ones. Although there is little agreement and also little evidence as to how these races come forth, by way of mutation or by way of adaptation, the races behave as stable entities if maintained on proper host plants.

When the breeding of potatoes for immunity from late blight at the Plant Breeding Station of the Centraal Bureau, the National Agricultural Cooperative Wholesale Society, started in 1943 two races of the fungus were known in the Netherlands, the "old" race and the S-race of Müller (10), occurring on the W-varieties, indicated by the author as N 1 and N 2 resp. (N means Netherlands). This reference was chosen in order to have a clear distinction from the letters of Müller (10), Black (2, 3) and Mills & Peterson (9) and the figures of Lehmann (6). From then on until 1952 7 new races were isolated, viz. races N 4, N 5 and N 7 in 1945; race N 6 in 1946; N 3 in 1947; N 8 in 1949 (found in a greenhouse) and N 9 in 1950. These races can be differentiated by means of a test series (table 1).

The susceptibility and immunity of several hundreds of clones were investigated during the period 1943–1952; all results were in agreement with the general outlines of table 1. All clones could be classed in 9 host groups according to their susceptibility to the 8 races.

TABLE 1. REACTIONS OF TEST SERIES DIFFERENTIATING THE RACES OF PHYTOPHTHORA INFESTANS FOUND IN THE NETHERLANDS

| Host group | Test series | Races of *P. infestans* [1] | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | N 1 | N 2 | N 4 | N 5 | N 6 | N 7 | N 8 | N 9 |
| I | *S. tuberosum* | − [3] | − | − | − | − | − | − | − |
| II | 43154–5 [2] | + [4] | − | + | + | + | − | − | + |
| III | 4431–5 | + | + | − | + | − | − | − | − |
| IV | 44158–4 | + | + | + | − | − | + | − | − |
| V | 4414–2 | + | + | + | + | − | + | − | − |
| VI | 46174–30 | + | + | + | + | + | − | − | + |
| VII | 4651–2 | + | + | + | + | + | + | − | + |
| VIII | 4737–33 | + | + | + | + | + | + | + | − |
| IX | 4739–58 | + | + | + | + | + | + | + | + |

[1]) Race N 3 has been omitted as there is only very little difference between this race and N 1.
[2]) This means: seedling 5, selected out of the progeny of cross 154 made in '43.
[3]) − = susceptible.
[4]) + = immune.

From table 1 may be deduced some important facts:

*a.* Susceptibility to race N 1 is always correlated with susceptibility to all other races.
*b.* Immunity from one of the "new" races is always connected with immunity from the "old" race N 1.
*c.* Immunity from the races N 4 or N 5 may be combined with susceptibility to race N 2.
*d.* Clones, immune from race N 6 are always immune from the races N 5, N 4 and and N 1, but may be susceptible to the races N 2, N 7, N 8 and/or N 9.
*e.* Clones, immune from race N 7 are always immune from the races N 2, N 4 and N 1, but may be susceptible to the races N 5, N 6, N 8 and/or N 9.
*f.* Immunity from race N 8 includes immunity from all the other races, except race N 9.
*g.* Clones, immune from race N 9 may be susceptible to race N 8 and even the races N 2 and N 7.
*h.* Clones, immune from the races N 8 and N 9 are always immune from all other races.

These facts, characterizing the host groups and the spectra of the blight races, are shown by the following diagram of the relationships and the differences between the blight races (fig. 1).

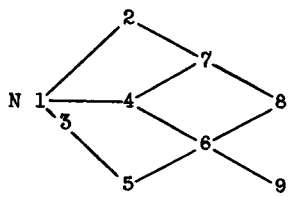


FIG. 1. DIAGRAM OF RELATIONSHIPS AND DIFFERENCES BETWEEN THE RACES OF THE LATE BLIGHT FUNGUS FOUND IN THE NETHERLANDS.

### 3. Inheritance of immunity

The mode of inheritance of the immunity of potatoes, derived from *Solanum demissum* by means of repeated backcrossing with cultivated varieties of *S. tuberosum*, was investigated by inoculating seedlings from crosses between these potatoes and varieties of *S. tuberosum* or other derivatives of *S. demissum*. Records were made of the number of attacked and of the nonattacked seedlings, from which hypotheses regarding the inheritance were formulated.

Young seedlings are transplanted from the seedbed into boxes and, after some time, inoculated by atomizing all over the plants a suspension containing zoospores and sporangia of a certain race or of certain races of *P. infestans*. Immediately after inoculation they are placed in moist chambers, where the leaves will keep wet the next 24 hours or even longer. Then the glass windows on top of the chambers are removed in order to keep the plants in the best possible conditions. When infection lesions have become clearly visible, the glass is replaced and within 24–48 hours there are plenty of sporangiophores on the attacked plants, 5–7 days after inoculation. Whereas the nonattacked, immune plants do not show anything at all or only very small pin-point, black, necrotic spots, indicating that the fungus does penetrate the leaves, but that the plants resist further development of the mycelium in the leaves (hypersensitive reaction).

In several experiments seedling plants were inoculated several times successively. It is thought that the necrotic hypersensitive reactions to a certain blight race in a previous inoculation will not influence the reactions of the same seedlings to another blight race in the following inoculation. Müller, Meyer & Klinkowski (14), Meyer (8), Müller & Börger (13) and Müller & Behr (12) have shown that in the necrotic cells compounds are produced which inhibit the growth of the *Phytophthora* mycelium, but also that of some other fungi. The necrotic spots formed as a consequence of a hypersensitive reaction upon the penetration of a certain blight race will be inaccessible to an other blight race. However, if inoculations are made properly, only a small percentage of the leaf area will be necrotic. As there is no or only very slight transport of the necrotic compounds, the normal green tissue of the leaves will be able to react as if there had been no previous inoculation. The time, elapsed between two successive inoculations has always been sufficient for all attacked plants to develop symptoms. As a consequence of these repeated inoculations and a prolonged stay in moist chambers some seedling plants succumb to root and foot diseases, others turn yellow and die, probably because of shortage of light, and still others are broken during the making of records of the numbers of attacked and nonattacked plants.

#### *A. Inoculations of seedlings of S. tuberosum*

These inoculations were run as checks alongside other experiments. Seedlings resulting from spontaneous pollination of the variety Katahdin were used. Not one plant was found to be immune from the blight races used (table 2). There appears to be no factor for immunity in the variety Katahdin, as far as can be determined by the present methods.

#### *B. Factor $R_8$*

It is evident from the data of table 3 that all susceptible plants have been eliminated

TABLE 2. REACTIONS OF SEEDLINGS FROM 47218 [1], INOCULATED WITH RACES OF P. INFESTANS

| Series | Race | – | + |
|---|---|---|---|
| A | N 1 | 98 | 0 |
| B | N 1 | 48 | 0 |
| C | N 1 | 94 | 0 |
| D | N 2 | 100 | 0 |
| E | N 2 | 92 | 0 |
| F | N 6 | 101 | 0 |
| G | N 7 | 98 | 0 |
| H | N 6 + N 7 | 70 | 0 |

[1]) 47218 = Katahdin SP (spontaneously pollinated).

in the first inoculation, whatever race may be involved. This can be explained by assuming one single dominant factor $R_8$ for immunity from the races N 1 to N 8 inclusive. In the crosses 4736, 4744, 4753, 4754 and 4765 a 1:1 segregation is expected theoretically. In the case of the 47179 and 4898 spontaneous pollinations a 1:3 segregation is expected if the pollination has actually been a self-pollination. $\chi^2$-test and P values, calculated according to PATTERSON (15), indicate that the hypothesis of one dominant factor $R_8$ is in agreement with the observed facts.

After race N 9 was found, clones possessing factor $R_8$ only invariably appeared to be susceptible to this race N 9. Factor $R_8$ was observed to occur in certain varieties of *S. demissum*, whereas other varieties and all their derivatives, obtained by backcrossing with susceptible *S. tuberosum* varieties, were susceptible to N 8.

In the years 1950 and 1951 a total of 33.840 seedlings from crosses in which only one parent was supposed to be simplex $R_8$, were inoculated with race N 8; 16.098 or 47,6% appeared to be immune. In 1952 out of 6189 seedlings 2982 or 48,2% were immune.

In the progeny of crosses of two clones, each having one factor $R_8$, theoretically 75,0% of the seedlings are expected to be immune from race N 8. In 1952 1878 seedlings from such crosses were inoculated with race N 1 and 1444 of them or 76,9% appeared to be immune. The same percentage of immune seedlings is expected in spontaneous progenies, supposing self-pollination. 994 seedlings from such progenies were inoculated with race N 1 in 1952, of which 743 or 74,7% were not attacked.

Among seedlings resulting from spontaneous fecundation (probably mainly self-fertilization) individuals with two factors $R_8$ on the four loci are expected to occur. Some were actually found, as may be concluded from the data of table 4.

The observations suggest a 1:5 segregation, that can be explained by assuming duplex $R_8$ in the mother plants. The factorial scheme will run as follows:

parents: $R_8R_8r_8r_8$ $r_8r_8r_8r_8$
gametes: $R_8R_8 + 4R_8r_8 + r_8r_8$ $r_8r_8$
zygotes: $R_8R_8r_8r_8 + 4R_8r_8r_8r_8 + r_8r_8r_8r_8$

Thus 5 out of 6 seedlings or 83% will be immune. P values indicate the correctness of the hypothesis.

TABLE 3. SEGREGATION IN PROGENIES OF $R_8R_8R_8R_8$ × RECESSIVE, INOCULATED WITH RACES OF P. INFESTANS

| (1949, 1950) Series | 1st inoculation | | | | 2nd inoculation | | | | 3rd inoculation | | | | 4th inoculation | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Race | − | + | %+ | Race | − | + | %+ | Race | − | + | %+ | Race | − | + | %+ |
| 4736 A | N 1 | 24 | 16 | 40 | N 7 | 0 | 16 | 100 | N 6 | 0 | 16 | 100 | | | | |
| B | N 2 | 57 | 39 | 41 | N 7 | 0 | 38 | 100 | N 6 | 0 | 38 | 100 | | | | |
| C | N 5 | 25 | 26 | 51 | N 6 | 0 | 26 | 100 | N 2 | 0 | 17 | 100 | N 7 | 0 | 17 | 100 |
| 4744 A | N 1 | 57 | 39 | 41 | N 2 | 0 | 39 | 100 | N 7 | 0 | 39 | 100 | N 6 | 0 | 38 | 100 |
| B | N 1 | 26 | 20 | 43 | N 2 | 0 | 20 | 100 | N 6 | 0 | 20 | 100 | N 7 | 0 | 20 | 100 |
| C | N 2 | 47 | 49 | 51 | N 7 | 0 | 49 | 100 | N 6 | 0 | 49 | 100 | | | | |
| 4753 A | N 7 | 13 | 10 | 43 | N 8 | 0 | 10 | 100 | | | | | | | | |
| B | N 8 | 13 | 20 | 61 | | | | | | | | | | | | |
| 4754 A | N 7 | 3 | 3 | 50 | N 8 | 0 | 3 | 100 | | | | | | | | |
| 4765 A | N 1 | 17 | 15 | 47 | N 2 | 0 | 15 | 100 | N 7 | 0 | 15 | 100 | N 6 | 0 | 15 | 100 |
| B | N 1 | 35 | 25 | 42 | N 2 | 0 | 23 | 100 | N 6 + N 7 | 0 | 23 | 100 | | | | |
| C | N 2 | 50 | 45 | 47 | N 7 | 0 | 44 | 100 | N 6 | 0 | 44 | 100 | | | | |
| D | N 7 | 51 | 44 | 46 | N 6 | 0 | 34 | 100 | | | | | | | | |
| 47179 A | N 7 | 6 | 14 | 70 | N 8 | 0 | 12 | 100 | | | | | | | | |
| B | N 7 | 9 | 20 | 69 | N 8 | 0 | 16 | 100 | | | | | | | | |
| C | N 8 | 19 | 45 | 70 | | | | | | | | | | | | |
| 4898 A | N 7 | 2 | 7 | 78 | N 8 | 0 | 7 | 100 | | | | | | | | |
| B | N 7 | 17 | 40 | 70 | N 8 | 0 | 40 | 100 | | | | | | | | |

4736 $P > 0{,}05$; 4744 $P > 0{,}10$; 4753 $P > 0{,}50$; 4754 $P = 1{,}00$; 4765 $P > 0{,}10$; 47179 $P > 0{,}30$; 4898 $P > 0{,}40$.

4736 = 442–8 (*S. demissum* 29 A × Frühmölle × Opperdoese Ronde × Katahdin) × Katahdin.
4744 = 4411–2 (*S. demissum* 29 A × Frühmölle × Opperdoese Ronde × JK 3843) × Earlaine.
4753 = 4420–3 (*S. demissum* 29 A × Frühmölle × Frühmölle × Jubel) × Alpha.
4754 = 4420–3 × Earlaine.
4765 = 4428–2 (*S. demissum* 29 A × Frühmölle × Frühmölle × Fransen) × Flava.
47179 = 443–7 (*S. demissum* 29 A × Frühmölle × Opperdoese Ronde × Koopmans Blauwe) SP.
4898 = Black 1257a (7) SP.
JK 3843 = Bevelander × Veenhuizen 31185.
442–8, 4411–2, 4420–3, 4428–2, 443–7 and Black 1257a (7) are immune from the races N 1 to N 8 inclusive and susceptible to race N 9 (host group VIII, table 1).

TABLE 4. SEGREGATION IN PROGENIES OF $R_8R_8R_8R_8$ × recessive

| Cross | Parentage | Race | − | + | %+ | P |
|---|---|---|---|---|---|---|
| 49240 | 4768–36 × Earlaine . . . . . | N 8 | 35 | 146 | 81 | > 0,30 |
| 49241 | 4768–36 × Frühmölle . . . . . | N 8 | 29 | 146 | 83 | 1.00 |
| 51245 | 4768–36 × 47233–34 . . . . . | N 1 | 15 | 85 | 85 | > 0,60 |
| 5121 | 47179–9 × Alpha . . . . . | N 1 | 11 | 53 | 83 | > 0,80 |
| 50372 | 47185–22 × Koopmans Blauwe . . . | N 8 | 57 | 230 | 80 | > 0,10 |
| 51294 | 47185–22 × 47233–34 . . . . . | N 1 | 68 | 329 | 83 | > 0,80 |

### *C. Factor* $R_9$

A number of crosses were made with the varieties Erika and Robusta, belonging to the W-group of MÜLLER and fitting into host group II (table 1). 3664 seedlings from these crosses were inoculated in 1944 with race N 1, 1835 or 50,1% proved to be

immune from this race (table 5). This may be explained by assuming one factor for immunity, present in simplex condition in Erika and Robusta. Later, all seedlings retained from these crosses appeared to be immune from the races N 4, N 5, N 6 and N 9 and to be susceptible to the races N 2, N 7 and N 8 without exception. The factor concerned is therefore referred to as $R_9$.

TABLE 5. SEGREGATION IN PROGENIES OF W-VARIETIES AND DERIVATIVES, INOCULATED WITH RACE N 1 OR N 9 OF P. INFESTANS

| (1944) Cross | Parentage | Race | − | + | %+ | P |
|---|---|---|---|---|---|---|
| 43146 | Berlikumer geeltje × Erika | N 1 | 77 | 70 | 48 | > 0,50 |
| 43147 | Bloemgraafje × „ | N 1 | 46 | 34 | 43 | > 0,10 |
| 43148 | Allerfrüheste Gelbe × „ | N 1 | 34 | 37 | 53 | > 0,70 |
| 43149 | Bravo × „ | N 1 | 43 | 40 | 48 | > 0,70 |
| 43150 | Deva × „ | N 1 | 101 | 130 | 56 | > 0,05 |
| 43151 | Duivelander × „ | N 1 | 154 | 136 | 47 | > 0,20 |
| 43152 | Eersteling × „ | N 1 | 128 | 106 | 45 | > 0,10 |
| 43154 | Eigenheimer × „ | N 1 | 81 | 94 | 54 | > 0,30 |
| 43155 | Erika × Allerfr. G. | N 1 | 38 | 63 | 62 | < 0,05 |
| 43156 | Erika × Koopm. Bl. | N 1 | 48 | 39 | 45 | > 0,30 |
| 43157 | Erika × Opperd. R. | N 1 | 255 | 193 | 43 | < 0,05 |
| 43158 | Flava × Erika | N 1 | 40 | 58 | 59 | > 0,05 |
| 43159 | Fransen × „ | N 1 | 102 | 131 | 56 | > 0,05 |
| 43160 | Frühmölle × „ | N 1 | 214 | 227 | 51 | > 0,50 |
| 43162 | Geelblom × „ | N 1 | 24 | 35 | 59 | > 0,10 |
| 43164 | Present × „ | N 1 | 173 | 184 | 52 | > 0,50 |
| | | | 1558 | 1577 | 50 | > 0,70 |
| (1944) 43165 | Robusta × Opperd. R. | N 1 | 271 | 258 | 49 | > 0,50 |
| (1952) 51362 | 43154–5 × 47233–34 | N 9 | 240 | 258 | 52 | > 0,05 |
| 51363 | 43154–5 × Bato | N 1 | 49 | 52 | 51 | > 0,70 |
| 51364 | 43154–5 × Libertas | N 1 | 355 | 363 | 51 | > 0,70 |
| | | N 9 | 33 | 34 | 51 | > 0,90 |
| | | | 677 | 707 | 51 | > 0,40 |
| (1952) 51448 | 47232–41 [1] × 47222–21 | N 9 | 484 | 502 | 51 | > 0,50 |
| 51449 | 47232–41 × 47223–6 | N 9 | 273 | 236 | 46 | > 0,10 |
| 51450 | 47232–41 × 47223–75 | N 9 | 40 | 46 | 53 | > 0,50 |
| 51451 | 47232–41 × 47231–21 | N 9 | 747 | 724 | 49 | > 0,50 |
| 51452 | 47232–41 × 47233–34 | N 9 | 394 | 357 | 48 | > 0,10 |
| | | | 1938 | 1865 | 49 | > 0,20 |

[1]) 47232–41 = Triumf × Aquila.

In 1952 three progenies from 43154–5 (= Eigenheimer × Erika) were inoculated, 51362 with race N 9, 51363 with race N 1 and 51364 with N 1 and N 9. In all four cases the theoretically expected 50% was approximated closely, according to the

P values. The results indicate that one factor ($R_9$) controls immunity from the races N 1 and N 9. 5 progenies of 47232–41 (= Triumf × Aquila), immune from the races N 1, N 4, N 5, N 6 and N 9, susceptible to N 2, N 7 and N 8, from crosses with clones, susceptible to all races of the blight fungus as are the common varieties of *S. tuberosum*, were inoculated with race N 9. The results may be explained by assuming one factor in 47232–41. This factor will originate from Aquila, one of the German W-varieties and is therefore assumed to be factor $R_9$ also.

That one factor ($R_9$) may be responsible for immunity from the races N 1, N 4, N 5, N 6 and N 9 is further illustrated by the data of table 6.

TABLE 6. SEGREGATION IN 47174 [1]), INOCULATED WITH RACES OF P. INFESTANS

| (1949) Series | 1st inoculation | | | | 2nd inoculation | | | | 3rd inoculation | | | | 4th inoculation | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Race | – | + | %+ | Race | – | + | %+ | Race | – | + | %+ | Race | – | + | %+ |
| A . . . . | N 1 | 24 | 79 | 77 | | | | | | | | | | | | |
| B . . . . | N 1 | 22 | 72 | 77 | | | | | | | | | | | | |
| C . . . . | N 1 | 29 | 67 | 70 | | | | | | | | | | | | |
| D . . . | N 1 | 23 | 71 | 76 | N 5 | 0 | 71 | 100 | N 2 | 71 | 0 | 0 | | | | |
| E . . . . | N 6 | 23 | 74 | 76 | N 2 | 68 | 0 | 0 | | | | | | | | |
| F . . . . | N 6 | 24 | 74 | 76 | N 4 | 0 | 74 | 100 | N 5 | 0 | 74 | 100 | N 2 | 74 | 0 | 0 |

[1]) 47174 = 43160–12 (Frühmölle × Erika) spontaneously (self-) pollinated.
$P > 0{,}95$.

In selfing an $R_9r_9r_9r_9$-individual a progeny is expected in which theoretically 75% of the seedlings will carry one or two $R_9$-factors. This explanation fits well to the observed data ($P > 0{,}95$). The susceptibility of all seedlings to race N 2 was not altered into immunity by previous inoculations, resulting in necrotic reactions.

A number of spontaneously (self-) pollinated SP progenies was inoculated in 1952. The results are summarized in table 7.

TABLE 7. SEGREGATION IN SP PROGENIES OF $R_9R_9R_9R_9$ CLONES AND VARIETIES.

| Progeny | Derivation | Race | – | + | %+ | P |
|---|---|---|---|---|---|---|
| 51365 | 43154–5 . . . . . | N 9 | 43 | 135 | 76 | > 0,70 |
| | | N 1 | 355 | 1148 | 76 | > 0,20 |
| 51367 | 43160–12 . . . . . | N 9 | 46 | 136 | 75 | > 0,90 |
| | | N 1 | 208 | 699 | 77 | > 0,10 |
| 51368 | Aquila . . . . . | N 9 | 152 | 497 | 77 | > 0,30 |
| | | N 9 | 124 | 325 | 72 | > 0,20 |
| | | N 1 | 154 | 476 | 76 | > 0,70 |
| 51369 | Jakobi . . . . . | N 9 | 63 | 151 | 71 | > 0,10 |
| | | N 9 | 50 | 171 | 77 | > 0,30 |
| | | N 1 | 250 | 683 | 73 | > 0,20 |

The results fit in with the general scheme of one dominant factor $R_9$. Jakobi is one of the newer German varieties, also belonging to the group of W-varieties (MÜLLER, 11).

*D. Factor $R_7$*

A third mendelian factor, designated by $R_7$, was postulated in order to explain segregations in another group of crosses. Factors $R_8$ and $R_9$ could be studied in progenies of clones, possessing only $R_8$ and $R_9$ resp. However, factor $R_7$ can only be dealt with in progenies involving factor $R_9$ as well, no progenies involving only $R_7$ being analysed yet.

TABLE 8. SEGREGATIONS IN THE PROGENY OF $R_7R_7R_7R_7R_9R_9R_9R_9 \times$ RECESSIVE 4972 [1])

| Series | 1st inoculation | | | | 2nd inoculation | | | | 3rd inoculation | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Race | – | + | %+ | Race | – | + | %+ | Race | – | + | %+ |
| A . . . | N 1 | 24 | 72 | 75 | N 6 | 21 | 51 | 71 | N 2 | 23 | 21 | 48 |
| B . . . | N 1 | 34 | 64 | 65 | N 4 | 0 | 63 | 100 | N 2 | 23 | 33 | 59 |
| C . . . | N 1 | 25 | 67 | 73 | N 5 | 20 | 34 | 63 | N 4 | 0 | 21 | 100 |
| D . . . | N 2 | 40 | 34 | 46 | N 7 | 0 | 19 | 100 | N 4 | 0 | 13 | 100 |
| E . . . | N 7 | 51 | 45 | 47 | N 6 | 21 | 24 | 53 | N 8 | 22 | 0 | 0 |
| F . . . | N 4 | 21 | 74 | 78 | N 7 | 28 | 46 | 62 | N 5 | 14 | 17 | 55 |
| G . . . | N 4 | 24 | 70 | 74 | N 5 | 21 | 48 | 70 | N 6 | 0 | 32 | 100 |
| H . . . | N 5 | 42 | 51 | 55 | N 6 | 0 | 49 | 100 | N 7 | 23 | 26 | 53 |
| I . . . | N 5 | 54 | 41 | 43 | N 6 | 0 | 41 | 100 | N 7 | 21 | 18 | 46 |
| J . . . | N 6 | 33 | 62 | 65 | N 2 | 30 | 30 | 50 | N 7 | 0 | 29 | 100 |
| K . . . | N 8 | 97 | 0 | 0 | | | | | | | | |
| L . . . | N 8 | 96 | 0 | 0 | | | | | | | | |
| M . . | N 8 | 96 | 0 | 0 | | | | | | | | |

[1]) 4972 = 4539–6 (*S. demissum* 29A × Frühmölle × Flava × Jubel) × Flava. Clone 4539–6 is susceptible to race N 8 only (host group VII, table 1).

Inoculations with the races N 1 and N 4 yield about 75% immune seedlings, whereas N 4 after N 1 did not give any attacked seedlings. This indicates that immunity from the races N 1 and N 4 is due to the same factor(s). The same may be concluded for N 5 and N 6 and also for N 2 and N 7. Two factors for immunity seem to operate here, one for immunity from N 1, N 4, N 5, N 6 (and N 9) and one for immunity from N 1, N 2, N 4 and N 7. The first is already known as $R_9$ (has at least the same effect), the second will be referred to as $R_7$. With this hypothesis the factorial scheme will run as follows:

parents: $R_7r_7r_7r_7R_9r_9r_9r_9$ $r_7r_7r_7r_7r_9r_9r_9r_9$

gametes: $R_7r_7R_9r_9 + R_7r_7r_9r_9 +$

$+ r_7r_7R_9r_9 + r_7r_7r_9r_9$ $r_7r_7r_9r_9$

zygotes: $R_7r_7r_7r_7R_9r_9r_9r_9 + R_7r_7r_7r_7r_9r_9r_9r_9 +$

$+ r_7r_7r_7r_7R_9r_9r_9r_9 + r_7r_7r_7r_7r_9r_9r_9r_9$

Upon inoculation with the various races the following segregations are expected (table 9).

Of the P values calculated on this basis, only that for series B, first inoculation and that for series J, first inoculation, are smaller than 0,05. In general the hypothesis fits

in well with the observed data, as may be seen from table 10, in which similar segregation ratios have been added up.

TABLE 9. SEGREGATION RATIOS EXPECTED IN 4972 ACCORDING TO THE HYPOTHESIS

| 1st inoculation | | 2nd inoculation | | 3rd inoculation | |
|---|---|---|---|---|---|
| N 1, N 4 | 1:3 | N 5, N 6, N 9 | 1:2 | N 2, N 7 | 1:1 |
| N 1, N 4 | 1:3 | N 2, N 7 | 1:2 | N 5, N 6, N 9 | 1:1 |
| N 5, N 6, N 9 | 1:1 | N 2, N 7 | 1:1 | | |
| N 2, N 7 | 1:1 | N 5, N 6, N 9 | 1:1 | | |
| N 8 | $\sim$:0 | | | | |

TABLE 10. $\chi^2$-TEST OF THE DATA OF TABLE 8.

| Ratio | Observed | Expected | $\chi^2$ | P |
|---|---|---|---|---|
| 1:1 | 329:348 | 338.5 :338.5 | 0.563 | $> 0{,}40$ |
| 1:2 | 113:212 | 108.3 :216.6 | 0.293 | $> 0{,}50$ |
| 1:3 | 128:347 | 118.75:356.25 | 0.961 | $> 0{,}30$ |

In the hypothesis $R_7$ and $R_9$ are postulated as different, independent genes. However there is another possibility, that $R_7$ and $R_9$ are allelomorphs of one gene. In this case the constitution of clone 4539–6 should be written $R_7R_9rr$. Then the factorial scheme would run as follows:

parents: $R_7R_9rr$ $rrrr$
gametes: $R_7R_9 + 2R_7r + 2R_9r + rr$ $rr$
zygotes: $R_7R_9rr + 2R_7rrr + 2R_9rrr + rrrr$

It is evident that other segregation ratios are to be expected (table 11). $\chi^2$-test gives some P-values that make this last hypothesis very improbable (table 11).

TABLE 11. $\chi^2$-TEST OF THE DATA OF TABLE 8, IF FACTORS $R_7$ AND $R_9$ ARE SUPPOSED TO BE ALLELOMORPHS OF ONE GENE.

| Ratio | Observed | Expected | $\chi^2$ | P |
|---|---|---|---|---|
| 1:1 | 220:233 | 226.5:226.5 | 0.373 | $> 0{,}50$ |
| 2:3 | 113:221 | 130 :195 | 3.705 | $> 0{,}05$ |
| 1:5 | 128:347 | 79 :396 | 36.379 | $< 0{,}01$ |
| 2:1 | 132:136 | 178.6: 89.3 | 30.483 | $< 0{,}01$ |

## 4. DISCUSSION

Three independent genetical factors have been postulated in order to explain the inheritance of the immunity from *Phytophthora infestans* in the above mentioned and similar cases. These factors are:

$R_7$, giving immunity from the races N 1, N 2, N 4 and N 7;
$R_8$, giving immunity from the races N 1, N 2, N 4, N 5, N 6, N 7 and N 8;
$R_9$, giving immunity from the races N 1, N 4, N 5, N 6 and N 9.

Clones, possessing one of these factors in simplex condition are expected to give theoretically 50 % immune seedlings in the progeny of crosses with recessive varieties.

With only a few exceptions this hypothesis could explain the observed data. In progenies resulting from selfing (or progenies from spontaneously pollinated berries) of clones of this simplex type 75 % immune seedlings are expected; this appeared also to be in agreement with the observed data.

Among the immune seedlings of these progenies individuals with two factors will occur. It was not investigated whether these duplex-seedlings did occur in the theoretical 1:2 ratio to the simplex-seedlings, but some were actually found (table 4). Because of the tetraploid nature of the potato plant, these duplex-individuals do not yield a complete immune progeny in crosses with recessives as would be the case in diploid plants. In tetraploid plants this will only be so in crosses with plants, possessing 4 factors (quadriplex and homozygous) or 3 factors (triplex). The observed segregations (table 4) approximate closely a 1:5 ratio, which is theoretically expected from the factorial scheme given just below table 4. This points, at least in these crosses, to the possibility of a pairing at random between chromosomes of both sets of two genoms if these are supposed to exist in plants of this interspecific origin. This means: allosyndesis in an allotetraploid plant (conjugation of homologous chromosomes of different parents) or the plants are autotetraploids (SIRKS, 16). The last possibility may or may not apply to *S. tuberosum*, it does not to plants of interspecific origin. Nevertheless, the free pairing indicates that the genoms of *S. demissum* and *S. tuberosum* are similar enough with respect to their chromosomes to form bivalents (HOWARD & SWAMINATHAN, 5).

The hypothesis of pairing at random is not in agreement with the results of BLACK (3), who is inclined to assume both autosyndesis and allosyndesis in approximately equal frequencies in duplex-plants. However, some of his results seem to fit in better with the hypothesis of pairing at random than to his assumption of preferential pairing.

In general, in progenies of crosses with recessives there is an excess of recessive susceptible seedlings. BLACK (2) ascribes this to minor incompatibility factors; they will tend to disappear as the number of backcross generations to *S. tuberosum* is increasing.

Clones, possessing the postulated three genes separately, will react upon inoculation with the blight races as follows:

| | N 1 | N 2 | N 4 | N 5 | N 6 | N 7 | N 8 | N 9 |
|---|---|---|---|---|---|---|---|---|
| $R_7$ | + | + | + | − | − | + | − | − |
| $R_8$ | + | + | + | + | + | + | + | − |
| $R_9$ | + | − | + | + | + | − | − | + |

It is evident that race N 1 is not differentiated from race N 4, race N 2 not from race N 7 and race N 5 not from race N 6. From certain observations it is very probable that a fourth gene, controlling immunity from the races N 1, N 2 and N 5, must be assumed in clone 4431–5 (group III, table 1); this factor will be referred to preliminary as $R_{2,5}$. Thus:

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| $R_{2,5}$ | + | + | − | + | − | − | − | − |

Now all races can be differentiated from each other. The existence of this $R_{2,5}$ factor will be shown in a future publication.

It may be readily concluded from the above statement that immunity from the races N 8 and N 9, including immunity from all other races, may be accomplished by com-

bining in one individual the factors $R_8$ and $R_9$. This will be shown in detail in a next article.

Other authors have postulated genetical factors in explaining their observations. It is desirable to try to coordinate the hypotheses. Before this can be done a comparison of the blight races these authors have worked with has to be made.

## 5. Summary

1. 9 races of *Phytophthora infestans* have been found, of which N 3 is almost identical with the "common" race N 1. A test series has been composed. There is considerable overlapping in the spectra of the races, as a consequence of which immunity from the races N 8 and N 9 includes immunity from all the other races, known up to date in the Netherlands.
2. Three genes for immunity have been postulated:

   $R_7$, giving immunity from races N 1, N 2, N 4 and N 7;
   $R_8$, giving immunity from races N 1, N 2, N 4, N 5, N 6, N 7 and N 8;
   $R_9$, giving immunity from races N 1, N 4, N 5, N 6 and N 9.

   Segregation ratios indicate that these factors are dominant allelomorphs of three indepent genes.
3. Segregation ratios in crosses of duplex-individuals indicate the possibility of pairing at random between chromosomes of all four genoms.
4. The presence of a fourth gene, controlling immunity from the races N 1, N 2 and N 5, is very probable.

## Samenvatting

*Onderzoekingen over de vererving van de resistentie tegen Phytophthora infestans* d.B. *van Solanum demissum* Lindl.

1. De aardappelziekte kost de Nederlandse economie naar schatting gemiddeld 20 millioen gulden per jaar; dit is evenveel als de geldelijke opbrengst van 13.000 ha aardappelen of $6\frac{1}{2}$ % van het aardappelareaal.
2. Van de bijna over de gehele wereld voorkomende schimmel *Phytophthora infestans*, die de aardappelziekte veroorzaakt, zijn de laatste decennia een aantal physio's bekend geworden, die zich onderscheiden door hun spectrum (reeks van planten, die vatbaar zijn voor een bepaald physio). In Nederland werd het voorkomen van 9 verschillende physio's geconstateerd (waarvan physio N 3 zeer veel gelijkt op physio N 1 en in tabel 1 werd weggelaten).
3. De spectra van de physio's overlappen elkaar (fig. 1), zodat resistentie tegen de physio's N 8 en N 9 steeds samen zal gaan met resistentie tegen alle overige van de thans bekende physio's.
4. Ter verklaring van een aantal waarnemingen over de vererving van de resistentie, werden drie verschillende dominante onafhankelijke erfelijke factoren aangenomen:

   $R_7$, geeft resistentie tegen de physio's N 1, N 2, N 4 en N 7;
   $R_8$, geeft resistentie tegen de physio's N 1, N 2, N 4, N 5, N 6, N 7 en N 8;
   $R_9$, geeft resistentie tegen de physio's N 1, N 4, N 5, N 6 en N 9.

Volgens de voor $\chi^2$ en P berekende waarden is deze verklaring zeer waarschijnlijk juist.

5. De splitsingsgetallen in kruisingen van planten met twee dezelfde factoren maken een vrije combinatiemogelijkheid tussen de betreffende chromosomen van alle 4 genomen zeer waarschijnlijk.
6. Naast de drie onder 4 genoemde factoren bestaat zeer waarschijnlijk nog een vierde factor, die resistentie tegen de physio's N 1, N 2 en N 5 bepaalt.
7. Resistentie tegen alle thans bekende physio's wordt bereikt bij gezamenlijke aanwezigheid van de factoren $R_8$ en $R_9$.

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