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# Inheritance of resistance to potato viruses Y and A in progeny obtained from potato cultivars containing gene *Ry*: evidence for a new gene for extreme resistance to PVA

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**Abstract** Extreme resistance in cultivated potato (*Solanum tuberosum*) to potato viruses Y and A (PVY and PVA) conditioned by the presence of *Ry* genes introduced from *Solanum stoloniferum* was described by Cockerham (1970). Cockerham detailed a number of genes which controlled a variety of reactions, including extreme resistance to both viruses (i.e. little or no visible reaction of plants and no viral replication following graft and manual inoculation) controlled by gene $Ry_{sto}$. In the present study, cvs 'Pirola' and 'Barbara', which contain a *Ry* gene, were found to have extreme resistance to PVY isolates from the ordinary ($PVY^O$), veinal necrosis ($PVY^N$) and potato tuber necrotic ringspot ($PVY^{NTN}$) subgroups, and PVA. The inheritance of this phenotype was examined in seedling progenies obtained by crossing 'Barbara' and 'Pirola' with susceptible cultivars. Segregation data for resistance to PVY and PVA in a progeny involving cv 'Pirola' best fitted a genetical model of one gene controlling extreme resistance to both PVY and PVA, although the possibility that there are two genes, each controlling resistance to one virus but closely linked, cannot be excluded. Segregation data from progenies involving cv 'Barbara' best fitted a genetical model in which there are two independent genes, one controlling extreme resistance to PVA and PVY and a second gene controlling extreme resistance to PVA but not to PVY. This previously unrecognised gene conferring extreme resistance to PVA only, should be given the notation *Ra* in keeping with nomenclature used for other resistance genes.

**Key words** Extreme virus resistance · Potyviruses · Genetics · Genes *Ry* and *Ra* · New gene

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## Introduction

Two distinct potyviruses, potato viruses Y (PVY) and A (PVA), were recognised many years ago to infect cultivated potato (*Solanum tuberosum*). Control of these aphid-borne viruses can be difficult, and the most effective means of preventing their spread in potato crops is by the use of durable resistance genes. A range of primitive *Solanum* species have resistance to PVY and PVA controlled by major genes (reviewed by Valkonen 1994), several of which have been transferred to *S. tuberosum* cultivars. The resistance can be classified into two main types depending on the reaction of plants following inoculation: necrotic (hypersensitive reaction), conferred by the *N* genes; and extreme resistance (very little or no visible effect) (Delhey 1975), conferred by the *R* genes. Following inoculation, virus can usually be recovered from plants with *N* genes but not from those with *R* genes. *Ny* and *Na* genes conferring hypersensitive resistance to PVY and PVA, respectively, occur individually in several cultivars, while a few cultivars contain both genes. *S. tuberosum* ssp. *andigena* and *S. stoloniferum* are sources of *R* genes to potyviruses. Clones containing *Ry* genes from *S. tuberosum* ssp. *andigena* are resistant to PVY but susceptible to PVA (Ross 1986). The extreme resistance to PVY in the polymorphic Mexican species *S. stoloniferum* was first reported by Cockerham (1943), Hawkes (1945) and Ross and Baerecke (1950). This extreme resistance is expressed to a range of PVY strains and also to PVA. However, comprehensive extreme resistance to both PVY and PVA was only one of seven phenotypic responses to be recognised by Cockerham (1970) in plants containing *Ry* genes from *S. stoloniferum*. Other phenotypes included necrotic and susceptible reactions to inoculation with one or both of the viruses. Cockerham (1970) found that the phenotypes observed in reaction to PVY and PVA are determined by five genes at three loci. The most valuable of these is a gene controlling extreme resistance to both viruses, which Cockerham (1970) designated as

$Ry_{sto}$ and which is similar in effect to gene $Ry$ described by Ross (1960).

Approximately 20 cultivars, have been bred that contain $Ry$ genes, including cultivars from The Netherlands, Poland, Hungary and Germany (Ross 1986). Jones (1990) reported on the reaction of cvs 'Pirola' and 'Corine' to inoculation with PVY and PVA. However, there seem to have been few, if any, other investigations that have examined in detail the inheritance of these genes and their control of resistance to PVY and PVA. This paper describes the behaviour of $Ry$-containing cvs 'Barbara', 'Corine', 'Fanal' and 'Pirola' to inoculation with isolates of PVY and PVA and an investigation of the inheritance of resistance to PVY and PVA in cvs 'Pirola' and 'Barbara'.

## Materials and methods

### Plant material

All plant material was grown in soil-less potting compost in an aphid-proof glasshouse at approximately 20 °C. Plants of virus-free potato cultivars were grown from tubers propagated in the glasshouse. Seedling progenies produced from crosses between 'Barbara' ($Ry$) and either 'Flourball' or 'Dr Macintosh' (susceptible cultivars) and between 'Pirola' ($Ry$) and 'Dr Macintosh' were sown, and each seedling plant (genotype) was grown until it was large enough for propagation by stem cuttings. Virus resistance tests on progenies were made with plants grown from stem cuttings.

### Virus isolates and inoculation

An isolate of $PVY^N$ was obtained from field-grown plants of potato cv. 'Record' (Barker 1994); $PVY^O$ and PVA were SCRI stock isolates. Isolates of $PVY^N$, $PVY^O$ and PVA were maintained in potato by vegetative propagation in cvs 'Record', 'Craigs Snow-White' and 'Majestic', respectively, and transmitted by manual inoculation as necessary to *Nicotiana tabacum*. Two Spanish isolates from the potato tuber necrotic ringspot disease subgroup ($PVY^{NTN}$) from potato cvs 'Hermes' and 'Picasso' were obtained from Dr J. Legorburu (Vitoria-Gasteiz, Spain) and maintained in *N. tabacum*.

Virus was transmitted to potato test plants by graft inoculation using scions (shoot apices) from infected potato plants which were cleft-grafted onto stems of test plants from which the shoot apex had been removed. Foliage of shoots which subsequently developed from the axillary meristems was used for resistance testing. Potato test plants were manually inoculated using freshly extracted sap from infected *N. tabacum* plants (1 g leaf/5 ml water) rubbed onto corundum-dusted leaves.

### Resistance tests

A standard test was used to assess resistance of the cultivars and genotypes derived from the progeny of the crosses. Plants were graft or manually inoculated when they were approximately 300 mm tall. Young foliage of each inoculated plant was tested twice by ELISA between 20 and 35 days after inoculation. Viral replication was also assessed by an infectivity assay in which indicator plants of *N. benthamiana* and *N. clevelandii* were manually inoculated with sap extracted from foliage of test plants. In addition, attempts were made to recover virus from selected test plants by 'return grafting' in which scions were grafted into virus-free susceptible indicator plants of potato (for PVY) or tomato (for PVA). Infection in indicator plants was assessed by ELISA 3–4 weeks after inoculation. In all cases when virus was not detected by ELISA of leaf tissue from potato test plants, neither was it possible to recover it by infectivity assay or 'return grafting' to virus-free susceptible plants. However, infectious virus was always recovered from plants in which it was detected by ELISA. Failure to detect virus by ELISA and to recover infectivity from inoculated plants was taken as an indication of resistance (immunity) to virus replication. Plants in which necrotic symptoms developed were classified as susceptible or resistant on the basis of whether virus was recoverable and not on the appearance of symptoms.

### Enzyme-linked immunosorbent assay

PVY was assayed by the antibody-trapped antigen form of indirect enzyme-linked immunosorbent assay (ELISA) as described by Barker et al. (1993). Microtitre plates were coated with anti-$PVY_Y$-globulin prepared from a polyclonal antiserum to $PVY^O$ coat protein, which traps particles of $PVY^O$, $PVY^N$ and $PVY^{NTN}$. This polyclonal antiserum and a detecting monoclonal antibody that reacts with the coat protein of $PVY^O$, $PVY^N$ and $PVY^{NTN}$ was obtained from the Scottish Agricultural Science Agency, East Craigs, Edinburgh. Leaf tissue was disrupted in a Pollähne roller press (1 g leaf tissue/10 ml extraction buffer). Samples of each extract were tested in duplicate wells. PVA was detected by double-antibody sandwich ELISA as described by Singh and Barker (1991) using a polyclonal antibody kindly donated by the Department of Agriculture, Belfast, Northern Ireland.

## Results

### Reaction of potato cultivars to inoculation with $PVY^O$ and $PVY^N$

At least four plants of each cultivar, were graft-inoculated and four were manually inoculated with each of the isolates ($PVY^O$ and $PVY^N$). Plants of cultivars not containing gene $Ry$ occasionally developed systemic mosaic symptoms following manual or graft inoculation, although such symptoms could be difficult to identify. Virus was readily detected in these plants by ELISA and recovered by the infectivity assay (Table 1). No symptoms associated with virus multiplication developed in plants containing $Ry$ genes following manual inoculation with either $PVY^O$ or $PVY^N$. However, plants of all cultivars, particularly 'Barbara', occasionally developed fine necrotic streaks on the veins of the abaxial leaf surface following virus inoculation or 'mock' inoculation with virus-free sap. It is concluded that such symptoms were a response to stress. Following graft inoculation with $PVY^O$ or $PVY^N$, plants of cvs 'Barbara' and 'Pirola' developed a few necrotic streaks in the stems, and occasionally a few leaves of apical shoots became necrotic and died, but virus was not detected in these plants by ELISA, the infectivity assay or 'return grafting' (Table 1). Barker and Harrison (1984) and Jones (1990) described similar localised necrotic reactions in cv 'Pirola' inoculated with PVY. Such necrotic symptoms did not develop on all occasions, and it seems likely that their development may be subject to differences between isolates and environmental conditions. Plants of cvs 'Fanal' and 'Corine' remained symptom-free after graft inoculation. It was not possible to detect PVY in inoculated plants of $Ry$-containing cultivars, by ELISA, infectivity assay or 'return grafting' (Table 1).

**Table 1** Reaction of a selection of potato cultivars to inoculation with PVY and PVA

| Potato clones | Known resistance genes to: | | Virus recovery[a] and (symptoms)[b] following inoculation of potato clones with potyviruses[c] | | | |
|---|---|---|---|---|---|---|
| | PVY | PVA | $PVY^N$ | $PVY^o$ | $PVY^{NTN}$ | PVA |
| Barbara | *Ry* | – | −(none) | −(none) | −(none) | −(LN) |
| Corine | *Ry* | – | −(none) | −(none) | −(none) | +(GN) |
| Dr Macintosh | – | *Na* | +(none) | +(none) | NI | +(GN) |
| Fanal | *Ry* | – | −(none) | −(none) | −(none) | −(none) |
| Flourball | – | – | +(none) | +(none) | NI | +(none) |
| Maris Piper | – | *Na* | +(none) | +(MM) | +(MM) | +(GN) |
| Nicola | – | – | NI | NI | +(MM) | NI |
| Pirola | *Ry* | – | −(none) | −(LN) | −(none) | −(LN) |

[a] Virus recovery determined by ELISA and infectivity assay of inoculated plants. +, Virus detected by both methods; −, no virus detected

[b] GN, Generalised necrosis in leaves on shoots above the graft union leading to death of apical meristems and eventual abscision of necrotic leaves and shoot death; LN, limited necrosis in some plants developing in the stem (often under the graft union) and occasionally with a very limited amount of necrosis in the apical leaves but never leading to death of shoots; MM, mild mosaic

[c] NI, Not inoculated

The possibility was considered that although $PVY^N$ would not replicate in plants of cv 'Barbara', virus might move passively through the phloem sieve tubes of short grafted stem sections and infect a susceptible shoot grafted onto the 'Barbara' stem. Stem sections (20 mm long) of virus-free 'Barbara' were grafted onto $PVY^N$-infected stock plants of cv 'Maris Piper', and a virus-free shoot apex of 'Maris Piper' was grafted onto the stem of 'Barbara'. PVY was not detected by ELISA of the receptor scions of 'Maris Piper' 6 weeks after grafting, although if virus-free stem sections of 'Maris Piper' were used instead of those of 'Barbara', virus was readily detected.

## Reaction of potato cultivars to inoculation with $PVY^{NTN}$

Le Romancer and Kerlan (1992) reported that plants of cv 'Corine' were hypersensitive following inoculation with $PVY^{NTN}$. Two Spanish isolates of $PVY^{NTN}$ were manually inoculated to four *Ry*-containing cultivars (four plants of each cultivar per isolate), but none developed symptoms or supported virus replication, although plants of cvs 'Maris Piper' and 'Nicola' were readily infected (Table 1).

## Reaction of potato cultivars to inoculation with PVA

At least four plants of each cultivar were graft-inoculated, and four were manually inoculated with PVA. Following manual or graft inoculation of cv 'Flourball', virus was readily detected by ELISA although the plants developed no obvious symptoms. Following graft-inoculation with PVA, plants of 'Dr Macintosh' and 'Maris Piper', known to contain gene *Na*, developed severe systemic necrosis which eventually resulted in the death of young shoots, a typical hypersensitive reaction, and virus was readily detected by ELISA (Table 1). However, plants of 'Dr Macintosh' and 'Maris Piper' were not susceptible to manual inoculation with PVA. Large necrotic local lesions developed on manually inoculated levels of cv 'Corine', and plants also developed severe systemic necrosis (hypersensitive reaction) following either manual or graft inoculation. Virus was readily detected in such plants by ELISA (Table 1). None of the other *Ry*-containing cultivars reacted to manual inoculation with PVA, and virus could not be recovered from plants by ELISA or infectivity assay. However, in initial tests the reaction of 'Barbara' and 'Pirola' to graft inoculation with PVA was erratic. In more extensive tests, approximately 20 plants of each cultivar were graft-inoculated over about seven occasions. In some plants, but not in others, a very limited necrosis developed in the stem under the graft union, and a limited amount of necrosis was found in young shoot tips and leaves. However, this necrotic reaction was much less extensive than that which occurred in plants of 'Corine', 'Maris Piper' and 'Dr Macintosh' and, in contrast to these cultivars, virus was not detected by ELISA, infectivity assay or 'return grafting'. Plants of cv 'Fanal' never developed necrosis following graft inoculation.

## Reaction of a progeny from a cross between 'Pirola' and 'Dr Macintosh' to inoculation with PVY and PVA

Plants of 29 genotypes from the progeny of a cross between cvs 'Pirola' (*Ry*) and 'Dr Macintosh' (susceptible) segregated into two groups based on their resistance following graft inoculation with $PVY^N$ and PVA. Eleven of the genotypes were susceptible to PVA and $PVY^N$, but the other 18 genotypes were resistant to both viruses (Table 2). Inoculation with $PVY^N$ did not induce symptoms in any plants. However, approximately half of the genotypes developed necrotic symptoms (similar to those produced in plants of 'Dr Macintosh') following

**Table 2** Phenotypic ratios for virus resistance in progeny 92.VT.49 derived from cross 'Pirola' × 'Dr Macintosh'

| | Genetic models tested[a] | Segregation following inoculation[b] with: | | | | Segregation following inoculation with $PVY^N$ and PVA | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | $PVY^N$ only | | PVA only | | Phenotype with Y/A reaction: | | | |
| | | Res. | Sus. | Res. | Sus. | Res/res | Res/sus | Sus/res | Sus/sus |
| Observed data | | 18 | 11 | 18 | 11 | 18 | 0 | 0 | 11 |
| Expected ratios | 1 | 1 : 1 | | 1 : 1 | | 1 : 1 : 1 : 1 | | | |
| | 2 | 1 : 1 | | 1 : 1 | | 1 : 0 : 0 : 1 | | | |
| | 3 | 1 : 1 | | 1 : 1 | | 0 : 1 : 1 : 0 | | | |
| $\chi^2$ (*P*) | 1 | 1.69 (0.1–0.5) | | 1.69 (0.1–0.5) | | N/A[c] | | | |
| | 2 | „ | | „ | | 1.69 (0.1–0.5) | | | |
| | 3 | „ | | „ | | N/A | | | |

[a] Model 1: two independent loci, one of which controls extreme resistance to PVY and the second which controls extreme resistance to PVA. Model 2: either one locus controlling extreme resistance to PVY and PVA; or two loci controlling extreme resistance as in model 1 but linked in coupling phase. Model 3: two loci controlling extreme resistance as in model 1 but linked in repulsion phase

[b] Two plants of each genotype were graft-inoculated with each of two viruses, $PVY^N$ and PVA. Resistance is defined as no virus detected in inoculated plants by ELISA or recovered by infectivity assay

[c] N/A, $\chi^2$ test for a particular genetical model is not appropriate because of missing or additional phenotype class(es) in progeny

**Table 3** Phenotypic ratios for virus resistance in progeny 93.V.32 derived from cross 'Barbara' × 'Flourball'

| | Genetic models tested[a] | Segregation following inoculation[b] with: | | | | Segregation following inoculation with $PVY^N$ and PVA | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | $PVY^N$ only | | PVA only | | Phenotype with Y/A reaction: | | | |
| | | Res. | Sus. | Res. | Sus. | Res/res | Res/sus | Sus/res | Sus/sus |
| Observed data | | 25 | 28 | 37 | 16 | 25 | 0 | 12 | 16 |
| Expected ratios | 1 | 1 : 1 | | 1 : 1 | | 1 : 1 : 1 : 1 | | | |
| | 2 | 1 : 1 | | 1 : 1 | | 1 : 0 : 0 : 1 | | | |
| | 3 | 1 : 1 | | 1 : 1 | | 0 : 1 : 1 : 0 | | | |
| | 4 | 1 : 1 | | 3 : 1 | | 2 : 0 : 1 : 1 | | | |
| $\chi^2$ (*P*) | 1 | 0.17 (0.5–0.9) | | 8.32 (< 0.01) | | N/A[c] | | | |
| | 2 | „ | | „ | | N/A | | | |
| | 3 | „ | | „ | | N/A | | | |
| | 4 | „ | | 0.76 (0.1–0.5) | | 0.77 (0.5–0.9) | | | |

[a] Model 1: two independent loci, one of which controls extreme resistance to PVY and the second which controls extreme resistance to PVA. Model 2: either one locus controlling extreme resistance to PVY and PVA; or two loci controlling extreme resistance as in model 1 but linked in coupling phase. Model 3: two loci controlling extreme resistance as in model 1 but linked in repulsion phase. Model 4: two independent loci, one controlling extreme resistance to PVY and PVA and the second locus controlling extreme resistance to PVA only

[b] Two plants of each genotype were graft-inoculated with each of two viruses, $PVY^N$ and PVA. Resistance is defined as no virus detected in inoculated plants by ELISA or recovered by infectivity assay

[c] N/A, $\chi^2$ test for a particular genetical model is not appropriate because of missing or additional phenotype class(es) in progeny

inoculation with PVA, both in the susceptible and the resistant classes. However, PVA was recoverable from the necrotic plants of the susceptible genotypes but not from those that were resistant. In this cross and that between 'Barbara' and 'Dr Macintosh', PVA-necrotic genotypes were classified as susceptible if virus was recoverable. The ratio of resistant to susceptible genotypes best fitted a genetical model of one locus controlling resistance to both PVY and PVA (Table 2). However, the possibility that there are two loci, each controlling resistance to one virus but linked in coupling phase, cannot be excluded.

## Reaction of a progeny from a cross between 'Barbara' and 'Flourball' to inoculation with PVY and PVA

Plants of 53 genotypes from the progeny of a cross between cvs 'Barbara' (*Ry*) and 'Flourball' (susceptible) segregated into three groups based on their resistance to graft inoculation with $PVY^N$ and PVA. Sixteen genotypes were susceptible to both $PVY^N$ and PVA, 25 genotypes were resistant to both viruses and 12 genotypes were susceptible to $PVY^N$ but resistant to PVA; no plants were susceptible to PVA but resistant to $PVY^N$

**Table 4** Phenotypic ratios for progeny 92 VT.46 derived from cross 'Barbara' × 'Dr Macintosh'

| | Genetic models tested[a] | Segregation following inoculation[b] with: | | | | Segregation following inoculation with $PVY^N$ and PVA | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | $PVY^N$ only | | PVA only | | Phenotype with Y/A reaction: | | | |
| | | Res. | Sus. | Res. | Sus. | Res/res | Res/sus | Sus/res | Sus/sus |
| Observed data | | 14 | 17 | 25 | 6 | 14 | 0 | 10 | 7 |
| Expected ratios | 1 | 1 : 1 | | 1 : 1 | | 1 : 1 : 1 : 1 | | | |
| | 2 | 1 : 1 | | 1 : 1 : | | 1 : 0 : 0 : 1 | | | |
| | 3 | 1 : 1 : | | 1 : 1 : | | 0 : 1 : 1 : 0 | | | |
| | 4 | 1 : 1 : | | 3 : 1 : | | 2 : 0 : 1 : 1 | | | |
| $\chi^2$ (*P*) | 1 | 0.29 (0.5–0.9) | | 11.65 (<0.01) | | N/A[c] | | | |
| | 2 | ,, | | ,, | | N/A | | | |
| | 3 | ,, | | ,, | | N/A | | | |
| | 4 | ,, | | 0.53 (0.1–0.5) | | 0.87 (0.5–0.9) | | | |

[a] Model 1: two independent loci, one of which controls extreme resistance to PVY and the second which controls extreme resistance to PVA. Model 2: either one locus controlling extreme resistance to PVY and PVA; or two loci controlling extreme resistance as in model 1 but linked in coupling phase. Model 3: two loci controlling extreme resistance as in model 1 but linked in repulsion phase. Model 4: two independent loci, one controlling extreme resistance to PVY and PVA and the second locus controlling extreme resistance to PVA only

[b] Two plants of each genotype were graft-inoculated with each of two viruses, $PVY^N$ and PVA. Resistance is defined as no virus detected in inoculated plants by ELISA or recovered by infectivity assay

[c] N/A. $\chi^2$ test for a particular genetical model is not appropriate because of missing or additional phenotype class(es) in progeny

(Table 3). Plants from this progeny were also manually inoculated with $PVY^O$ and resistance assessed. Genotypes which were resistant to $PVY^N$ were also resistant to $PVY^O$, and likewise with the susceptible genotypes. Symptoms did not develop in any of the inoculated plants. On the basis of this data, the genetical model proposed for control of resistance to PVY and PVA in the cross 'Pirola' × 'Dr Macintosh' was inappropriate. The data best fitted a genetical model in which there are two independent loci, one controlling resistance to PVY and PVA and a second locus controlling resistance to PVA only (Table 3).

### Reaction of a progeny from a cross between 'Barbara' and 'Dr Macintosh' to inoculation with PVY and PVA

Plants of 31 genotypes from the progeny of a cross between cvs 'Barbara' (*Ry*) and 'Dr Macintosh' (susceptible) segregated into three groups based on their resistance to graft inoculation with $PVY^N$ and PVA. Seven of the genotypes were susceptible to $PVY^N$ and PVA, 14 were resistant to both viruses, 10 were susceptible to $PVY^N$ but resistant to PVA; no genotypes were obtained which were susceptible to PVA but resistant to $PVY^N$ (Table 4). Inoculation with $PVY^N$ did not induce symptoms in plants of the progeny from 'Barbara' × 'Dr Machintosh'. However, following inoculation of PVA, necrotic symptoms similar to those produced in plants of 'Dr Macintosh' developed in some plants. As with the cross 'Pirola' × 'Dr Macintosh', these were not confined to either susceptible or resistant genotypes. Again, the data best fitted a genetical model in which there are two independent loci, one controlling extreme resistance to PVY and PVA and a second locus controlling extreme resistance to PVA only (Table 4).

## Discussion

Resistance to PVY replication in four potato cultivars containing gene *Ry* is extremely strong. Following graft inoculation, virus cannot be detected by ELISA or recovered by either the infectivity assay or 'return graft' inoculation to susceptible test plants. In other tests (H. Barker unpublished results) on graft-inoculated plants of cvs 'Barbara' and 'Corine', PVY was not detected by immunosorbent electron microscopy or a sensitive polymerase chain reaction assay. Extreme resistance to PVY in *Ry*-containing potato cvs 'Corine' and 'Pirola' was expressed in mesophyll protoplasts inoculated *in vitro* (Barker and Harrison 1984). Only 0.1% of the protoplasts from 'Corine' and Pirola' became infected compared with 11% of those from susceptible cv 'Kerr's Pink'. Attempts to transmit PVY passively through short stem segments of cv 'Barbara' failed, presumably because PVY cannot move without replication, which is prevented in *Ry*-containing tissue. *Ry*-mediated resistance is effective against isolates from the ordinary ($PVY^O$), veinal necrosis ($PVY^N$) and tuber necrotic ringspot disease ($PVY^{NTN}$) subgroups. Le Romancer and Kerlan 1992 found that cv 'Corine', which is resistant to isolates of $PVY^O$ and $PVY^N$, was susceptible to an isolate from the $PVY^{NTN}$ subgroup. It is possible that isolates of $PVY^{NTN}$ other than the ones I have used may replicate in cv 'Corine'. However, it would be prudent to check any isolates that do so for the presence of other

potyviruses because both PVA and potato virus V cause a hypersensitive reaction in cv 'Corine' (Jones 1990). With the exception of an occasional limited and localised necrotic reaction in plants of cvs 'Barbara' and 'Pirola' graft-inoculated with PVY, *Ry*-containing cultivars were symptom-free following graft or manual inoculation of PVY.

A hypersensitive reaction was induced in plants of cvs 'Dr Macintosh' and 'Maris Piper' following graft inoculation of PVA, and virus was readily detected by ELISA. 'Corine' was hypersensitive to manual or graft inoculation with PVA, and the virus was detected by ELISA. There was an uneven response of other *Ry*-containing cultivars to inoculation with PVA. Cultivars 'Barbara', 'Pirola' and 'Fanal' did not react to manual inoculation, but graft inoculation induced a limited and localised necrotic reaction in some plants of 'Barbara' and 'Pirola'. However, numerous attempts to detect or recover virus from plants of these three cultivars were unsuccessful.

It is concluded that despite limited necrotic reactions induced by some virus/host combinations, normal virus replication does not occur and cvs 'Pirola', 'Barbara' and 'Fanal' are immune to replication of PVY and PVA. The cause of the necrotic reaction is unknown but may be induced by virus replication in a few cells which are initially invaded from the grafted scion. These then trigger a resistance reaction and a systemic host response. Or, a defective or incomplete cycle of viral replication which does not lead to the production of infectious particles or coat protein may be responsible. The extreme resistance of cvs 'Barbara', 'Pirola' and 'Fanal' would seem to be that of phenotype 1 as defined by Cockerham (1970), i.e. no visible reaction following manual inoculation, a limited necrotic reaction following graft inoculation and no virus recovery from plants. The reaction of cv 'Corine' would be classified as phenotype 3, i.e. no visible reaction to PVY with no virus recovery, and hypersensitivity to PVA with virus being recoverable from inoculated plants (Cockerham 1970).

Theoretically, half the genotypes from progenies of the crosses 'Pirola' × 'Dr Macintosh' and 'Barbara' × 'Dr Macintosh' should inherit gene *Na* from 'Dr Macintosh'. Indeed, approximately half the plants from these crosses developed necrosis typical of that induced by gene *Na*, but the symptoms were not confined to either susceptible or resistant (with respect to PVA replication) genotypes. Thus, some genotypes were identified in which virus could not be detected but which reacted with necrosis following PVA inoculation. These results are intriguing because they suggest that in the presence of an *R* gene providing immunity to PVA, one property of gene *Na* may be elicited although viral replication does not take place. It is not known whether this is a hypersensitive response induced by gene *Na* or whether it is triggered by some initial stages of viral replication but which does not lead to normal infection.

The progeny from the cross 'Pirola' × 'Dr Macintosh' segregated into two groups with either susceptibility or resistance to PVY and PVA. The predicted genetical model for this cross (Table 2) does not conflict with the hypothesis proposed by Cockerham (1970) that gene $Ry_{sto}$ conditions extreme resistance to PVY and PVA. However, Cockerham did not identify the phenotype of susceptible to PVY and resistant to PVA, and this phenotype appears in progenies from the crosses between 'Barbara' × 'Flourball' and 'Barbara' × 'Dr Macintosh' (Tables 3 and 4). There are two major genes for extreme resistance in a genetical model which provides a good fit for the data from crosses with cv 'Barbara', one gene which like that in 'Pirola' provides extreme resistance to PVY and PVA and a second gene which provides extreme resistance to PVA only. Further tests are required to determine whether the gene for PVA resistance is present in other potato cultivars which have inherited genes from *S. stoloniferum* and whether it is allelic with gene $Ry_{sto}$. On the basis of the present evidence, this newly identified gene for extreme resistance to PVA should be given the notation *Ra* in keeping with other resistance genes. In future work aimed at developing a more precise understanding of the nature of virus/gene interactions, comparisons between *Ry* and *Ra* should prove to be of great value.

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