Breeding for Resistances to Potato Virus Y and Potato Virus A

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Introduction

Among the many traits that a potato breeder must select in a breeding program are resistances to viruses. Resistance to virus is not always high on the priority list because virus infection is considered to be a problem that may be effectively dealt with by seed programs. Indeed it often appears that problems with viruses occur in cycles. A build-up in the incidence of a particular virus is addressed by a corresponding vigilance, elimination of infected stock, and increased investment in detection.

Potato virus Y (PVY) and Potato Virus A (PVA) are potyviruses transmitted in a non-persistent manner by aphids or mechanically. In the field aphid transmission is the usual means of spread. Symptoms range from mild mosaic to severe necrosis leading to plant death. Spread of PVY and PVA may occur particularly early in the growing cycle before roguing can remove infected plants. It may be that potyviruses have entered a new age of intractability. In particular, Potato virus Y (PVY) appears to be almost uncontrollable across all the Seed Districts of the United States. Potato leafroll virus (PLRV), is more amenable to control by seed hygiene and insecticidal prevention of spread. Furthermore, the appearance of the tobacco necrotic strain of PVY (PVY-N) has increased the difficulty of detection of PVY in seed programs due to its tendency toward latency in most varieties.

The potato originated in South America and was distributed to other regions of the world after European contact. There are several thousand cultivars and over two hundred wild species composed of thousands of accessions presently available as a source of genes controlling traits of interest. During the twentieth century, numerous sources of resistance to virus have been explored and a number of have been thoroughly exploited.

Sources of Resistance to Potyviruses in Potato

Extreme resistance to PVY has been found in *Solanum stoloniferum* (Ross, 1957; 1960; Delhey, 1975) and *Solanum tuberosum* ssp *andigena* (Table 1) (Muñoz et al., 1975, Galvez and Brown, 1980). Both sources of resistance provided a monogenic dominant source of resistance (Ry_{sto} and Ry_{adg}, respectively). Surpluses of PVY resistant progeny that are often found after mechanical inoculation may be explained by an unmapped necrosis gene "*Ny*" that is hypostatic to Ry_{adg} (Valkonen et al., 1994a). The *S. stoloniferum* source

coincidently carries with immunity to potato virus A (PVA), a potyvirus with similar attributes (Ross, 1960). The *S. tuberosum* ssp *andigena* source displays a hypersensitive reaction to PVA (Fernandez-Northcote, 1990). Nearly all breeding clones and varieties possessing the *S. stoloniferum* source are male-sterile, presumably due to cytoplasmic male sterility, although there are some exceptions to this. Both sources have been mapped to identical locations on chromosome 11 (Figure 1) (Brigneti et al., 1997; Hamalainen et al., 1997).

Further mapping revealed that the immunities to PVY and PVA are due to closely linked but distinct genes in both sources (Barker, 1996; Hamalainen et al., 1998). A number of genes were detected in extensive studies of the wild species sources of PVY and PVA resistance by Cockerham (1970). Extreme resistance to PVY and PVA controlled monogenically was found in *S. hougasii* (Ry_{hou}). Hypersensitivity to both viruses controlled in a monogenic dominant fashion was found in *S. chacoense*, and *S. microdontum* (Ny_{chc}) and in *S. demissum* (Ny_{dms}). It is likely that further surveys of wild germplasm would find numerous monogenes controlling hypersensitive or extreme resistances to PVY, PVA, and other potyviruses.

The reaction of potato breeding clones harboring the Ry_{sto} has been characterized histologically using tobacco etch virus (TEV) that expresses a GUS gene when the genome is transcribed. After inoculation, systemic spread of the virus is not detectable although replication of virus occurs in initially infected cells. Necrotic streaks that become apparent in leaf veins starting 3 days after inoculation are the visible manifestation of a hypersensitive reaction that apparently stops spread of the virus. This reaction is elicited by several distinct potyviruses including tobacco etch virus (TEV) normally not found in potato (Hinrichs et al., 1998). Ry_{sto} is apparently effective against all known strains of PVY including the "common (PVY-O)" "necrotic (PVY-N)"and the causative agent of "tuber necrotic ringspot disease" (PVY-NTN) (Chrzanowska and Muchalski, 1999).

Identification of Potyvirus Resistance in the Tri-State Breeding Program

The breeding activities in the Northwest have utilized a broad range of germplasm including sources with single gene resistances discussed above. At the Prosser Station we have been evaluating the resistance by grafting virus-infected scions onto test plants and serologically testing for virus in the new shoots that grow on the grafted stem (Table 2). We have classified numerous breeding clones as being immune to one or more of the potyviruses. The recent discovery that resistance to PVY and PVA is controlled by separate genes seems to be borne out by the discovery of clones that are immune to one or the other but not both. However immunity to both is much more common than immunity to just one virus. It has also been gratifying to confirm that immunity to PVY-O is always accompanied by immunity to PVY-N.

Conclusions

In the future we will continue to graft test new breeding products to clearly identify which ones are immune to which potyviruses. We will also be testing the reaction of our "immune" parents to the total range of PVY strains and isolates emphasizing those that fall into the PVY-N group to determine if any resistance breaking strains have been collected.

Future crosses at Prosser will emphasize the concentration of PVY immunity genes in order to produce "duplex" parents that will confer immunity on 80% of the progeny in a cross. Building up the copy number in breeding parents can make the whole process of breeding more efficient by practically eliminating the need for screening.

Table 1. Germplasm sources of resistance to PVY and PVA

Virus	Germplasm source	Type of resistance	Gene symbol
PVY	*S. stoloniferum*	Extreme resistance	Ry_{sto}
PVY	*S. tuberosum* ssp. *andigena (?)*	Extreme resistance	Ry_{adg}
PVY	*S. phureja*	Relative resistance	Polygenic
PVY	Sect. Etuberosum *S. palustre, S. etuberosum, S. fernandezianum*	Variable	Not determined
PVY	*S. demissum,*	Hypersensitivity to PVY and PVA	Ny_{dms}
PVY	*S. chacoense*	Hypersensitivity	Ny_{chc}
PVY	*S. hougasii*	Extreme resistance	Ry_{hou}
PVA	*S. stoloniferum*	Extreme resistance	Ra_{sto}

Table 2. Response of Tri-State Program breeding clones and varieties to graft inoculation testing with PVY strains and PVA.

Clone	*PVY-O*	*PVY-N*	*PVA*
R241-16 (adg)	Immune	Immune	Susceptible
A93570-13 (adg)	Immune	Immune	Hypersensitive
A88625-10 (adg)	Immune	Immune	Hypersensitive
A88617-6	Immune	Immune	Susceptible
Bzura (sto)	Immune	Immune	Immune
PA98V1-1 (sto)	Immune	Immune	Immune
PA98V2-1 (sto)	Immune	Immune	Immune
Russet Burbank	Susceptible	Susceptible	Susceptible
Bannock	Susceptible	Susceptible	Immune
Shepody	Susceptible	Susceptible	Immune

adg = Derived from the *Solanum tuberosum* ssp *andigena* source PVY immunity.
sto = Derived from the *Solanum stoloniferum* source of PVY immunity.

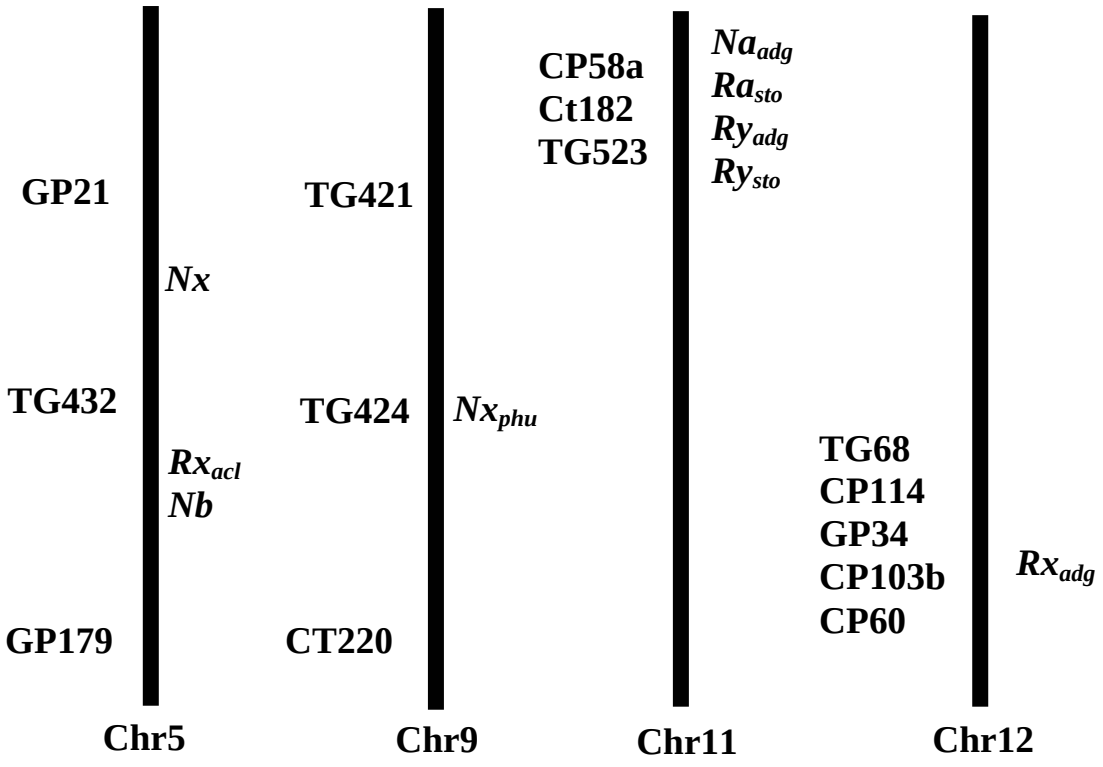

Fig. 1. Genomic map of known genetic factors controlling virus resistance. Molecular markers are to the left and virus resistance genes are to the right of the chromosome bars.

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