Effect of infection by viruses on vegetative and reproductive growth of sweet cherry on Damil and Inmil rootstocks

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ABSTRACT: The effect of infection with *Prunus necrotic ringspot virus* (PNRSV) and *Prune dwarf virus* (PDV) on vegetative and reproductive growth of sweet cherry trees (*Prunus avium* L.) was investigated. Infected trees were smaller and the growth reduction was more severe in Lapins than in Sam; when trees were infected in the nursery (early) compared to an infection two years later in the orchard; and when trees were grafted on the dwarfing Inmil rootstock compared to the more vigorous Damil. Tree mortality after establishment in the orchard was not a problem and only one virus infected tree died during the 5-year observation period. Reduced vigour was accompanied by increased blind wood formation. While the infected trees had a higher generative spur density the total number of generative spurs per tree was less than in the virus free controls, thus reducing yield. In addition the germination of pollen from infected trees was reduced. In general PNRSV has little or no significant influence on vegetative or reproductive behaviour, while PDV significantly reduced both characteristics. The combined infection had dramatic effects, especially on the dwarfing Inmil rootstock with the susceptible cv. Lapins. After four years in the orchard 10% of the initially virus free trees were infected by PDV and 48% by PNRSV.

Keywords: cherries; viruses; *Prunus avium* L.; *Prune dwarf virus*; *Prunus necrotic ringspot virus*; tree growth; yields

The planting of sweet cherry has increased during the last decade with the availability of dwarfing rootstocks, but high-density planting systems require higher investments, and higher returns are expected. In some cases this return is not realised due to reduced growth, bud and even tree mortality caused by virus infections (DESVIGNES 1990; GILLES, VERHOYEN 1992; MINK, JONES 1996). Quantitative data on the relationship between virus infection and the effect on the reproductive and vegetative development of sweet cherry trees is limited. According to MINK (1995) *Prunus necrotic ringspot virus* (PNRSV) and *Prune dwarf virus* (PDV) are common in stone fruit. In France 10 to 30% of the sweet cherry trees are infected by PNRSV or PDV, respectively (DESVIGNES 1990). In Belgium 80 to 100% of older cherry orchards are infected by PNRSV (GILLES, VERHOYEN 1992). Both viruses spread through pollen (MINK 1995; MINK, JONES 1996). As virus infected sour cherry (KRYCZYNSKY et al. 1992) and plum trees (BLAŽEK et al. 2000) can easily infect virus free trees, the contamination can increase considerably with time.

Considering the lack of knowledge of virus infections on the performance of sweet cherries growing on dwarfing rootstocks, we followed the effects of infection by PNRSV, PDV on the vegetative and reproductive performance of newly planted sweet cherry trees. Cultivar susceptibility and rootstock effects were also investigated.

MATERIAL AND METHODS

Virus free material of *Prunus avium* cv. Lapins was grafted on the virus free Damil (GM 61/1) (*Prunus* × *dawyckensis*) and Inmil (GM 9) (*P. incisa* × *P. serrula*) rootstocks in the winter 1990. Simultaneously, Sam (*P. avium*) was grafted onto Inmil rootstocks only. Virus inoculation involved budding of infected buds on the main axis of the tree at the end of the first growth season in the nursery (1990). Eight trees of each cultivar and rootstock combination were infected with PNRSV, PDV, and a combination of both (PDV + PNRSV). The trees were allowed to grow for another year in the nursery (in 1991). The 2-year-old nursery trees were planted in autumn 1991 at the Fruitteeltcentrum, Rillaar, Belgium (52° N), in randomised complete blocks, at a spacing 4 × 2 m for trees on the dwarfing Damil rootstock, and 4 × 1 m for trees on the extremely dwarfing Inmil rootstock. Additional virus free trees were planted and inoculated with PNRSV, PDV and PDV + PNRSV after the first year of growth in the orchard, in August 1992. Virus free trees that were not inoculated by budding served as controls. The performance of these trees in terms of tree mortality, shoot growth, flower bud formation, pollen viability and yield on infected and virus free trees was followed over five years. The infection of virus free trees by pollen transfer was followed by regular ELISA testing (PEUSENS 1996).

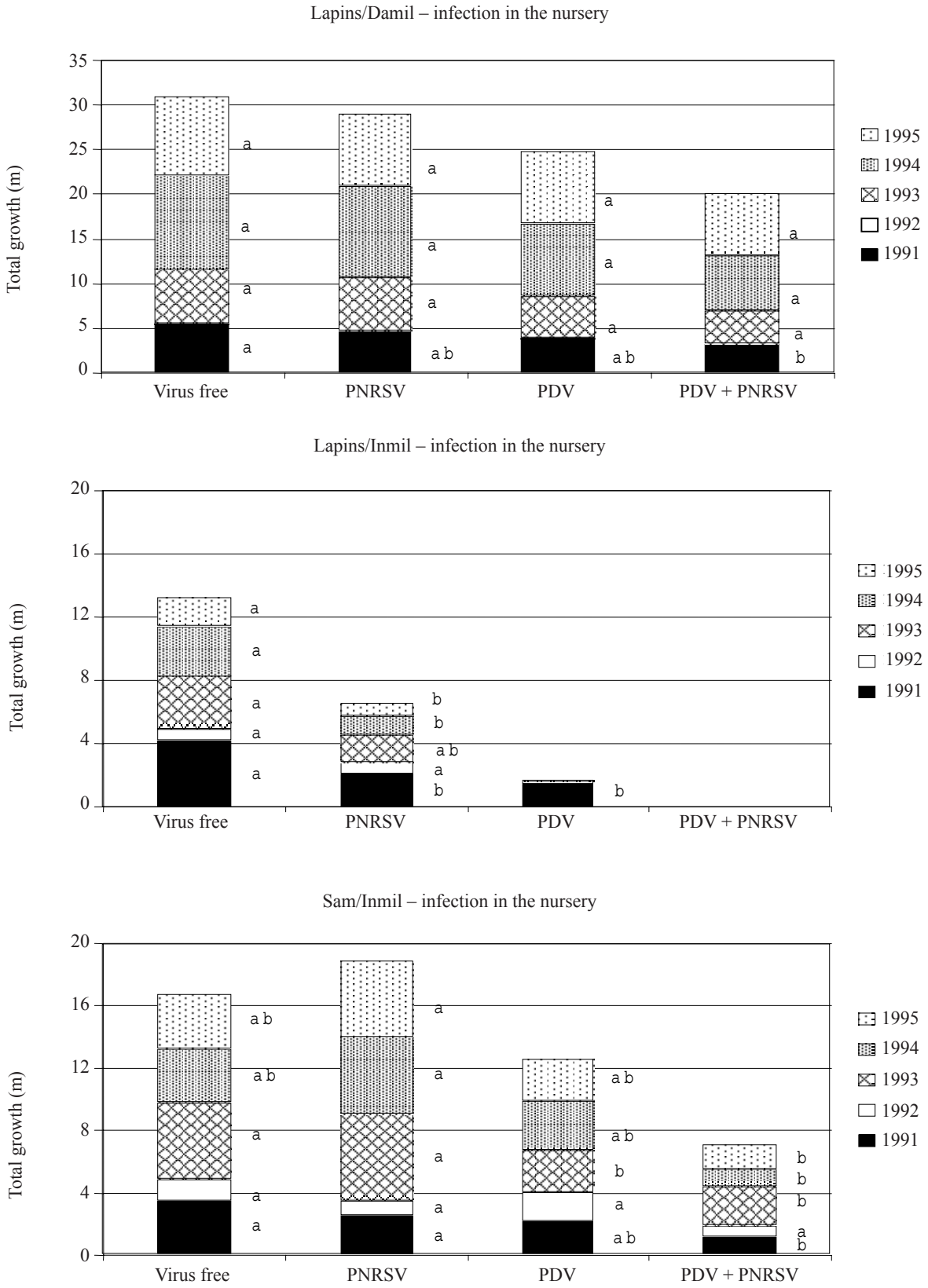

Fig. 1. The cumulative shoot growth of sweet cherry cvs. Lapins on the rootstocks Damil and Inmil and Sam on Inmil, virus free or infected in the nursery (1990) with PNRSV, PDV and PDV + PNRSV. Means separation within years by Duncan's multiple range test ($P = 0.05$)

RESULTS

Tree mortality

No sweet cherry trees died in the planting year and one PDV infected tree died during the 1992/1993 winter. Sweet cherries are less sensitive to PDV than sour cherries (DESVIGNES 1990; ANDERSONE et al. 2002). Water stress killed 4% of the trees shortly after planting. Sweet cherries are known to be intolerant of water logging (MOONS et al. 1994; WUSTENBERGS et al. 1995). Mortality of the sweet cherries after infection was negligible.

Tree growth

The growth of sweet cherries was clearly reduced by PDV, but the severity of this reduction was cultivar and rootstock dependent (Figs. 1 and 2). PDV infection more severely reduced growth than PNRSV. Infection with PNRSV after planting in the orchard had little or no negative effect on growth. PNRSV only reduced growth of Lapins on Inmil, yet when infected in the nursery the presence of both viruses (PDV + PNRSV) always had the greatest negative effect. When the infections were made later (in the orchard) the negative effect of PDV + PNRSV was not worse than PDV. Infected trees had less shoots and the shoots were also shorter (data not presented).

The total shoot growth of Lapins on Damil infected with PDV + PNRSV in 1990, during the two years in the nursery, was significantly reduced (Fig. 1). Infection with PDV before and after planting reduced growth with earlier infection more severely than the latter (42% vs. 38% respectively). With Lapins the effects of

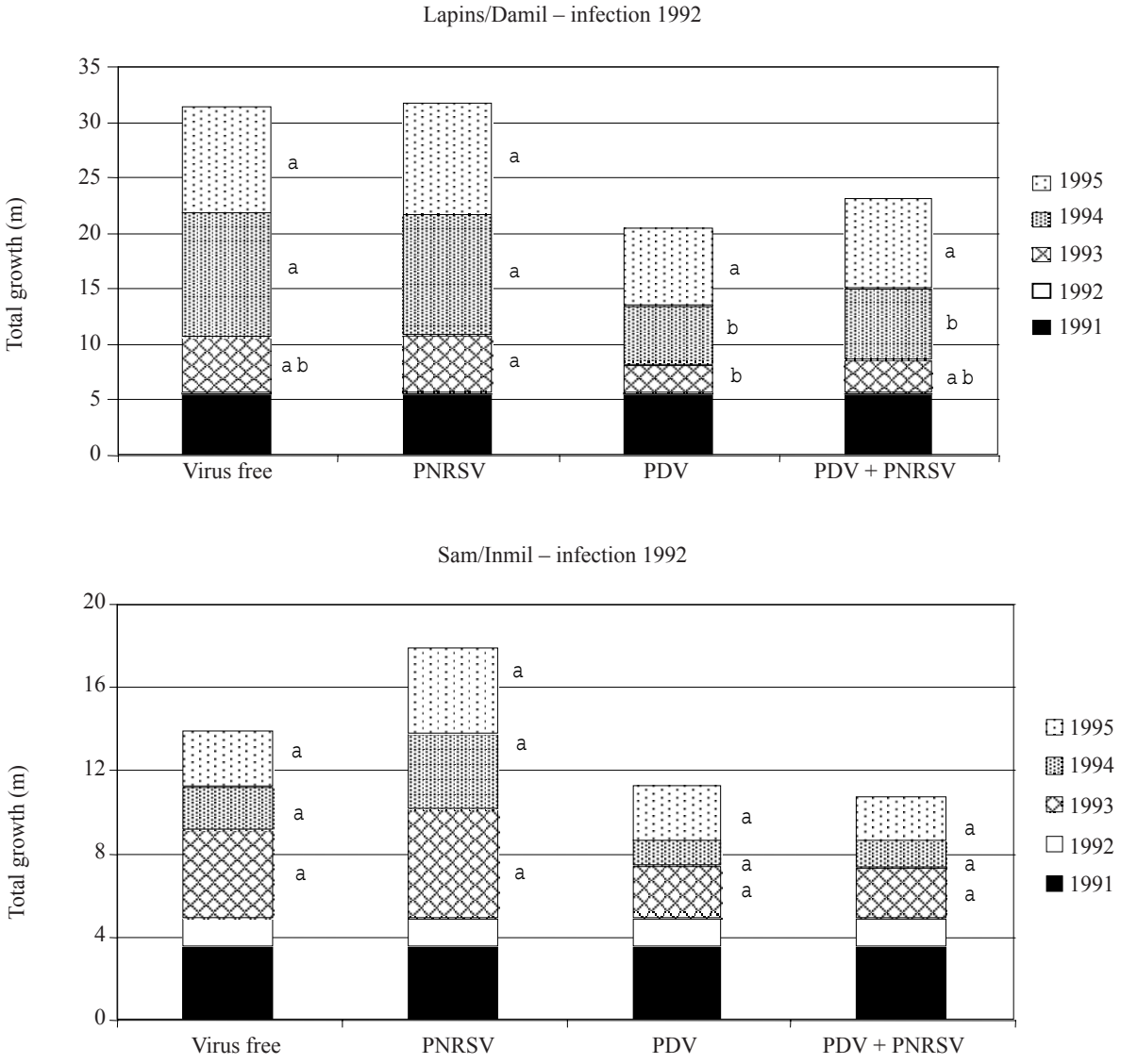

Fig. 2. The cumulative shoot growth of sweet cherry cvs. Lapins on the rootstocks Damil and Inmil and Sam on Inmil, virus free or infected in the orchard (1992) with PNRSV, PDV and PDV + PNRSV. Means separation within years by Duncan's multiple range test ($P = 0.05$)

Table 1. The number of blind nodes per metre branch length not less than 3-years-old of sweet cherry trees that were virus free or infected in the nursery (1990) with PNRSV, PDV and PDV + PNRSV

Cultivar/rootstock	Virus treatment	1993		1994	
Lapins/Damil	virus free	6.7	b	13.4	b
	PNRSV	6.7	b	17.2	ab
	PDV	9.1	a	22.3	a
	PDV + PNRSV	9.5	a	22.3	a
Lapins/Inmil	virus free	7.8	ab	9.3	b
	PNRSV	5.7	b	10.5	b
	PDV	14.8	a	59.3	a
	PDV + PNRSV	–		–	
Sam/Inmil	virus free	11.2	b	17.1	b
	PNRSV	6.8	b	32.1	ab
	PDV	14.9	ab	25.7	ab
	PDV + PNRSV	24.9	a	40.7	a

Means separation within columns for each cultivar/rootstock combination by Duncan's multiple range test ($P = 0.05$)

virus infection was more severe on Inmil than on Damil (Fig. 1). On Inmil the growth of Sam was inhibited less than Lapins (Fig. 1). With Sam the PDV + PNRSV infection was again most severe, 49% when infected in the nursery and 42% when infected in the orchard.

Blind wood formation

The effect of virus infection in the nursery (1990) on blind wood formation is shown in Table 1. Blind wood was evaluated as the number of blind nodes (without shoots or spurs) per metre of wood that is 3 years or older, in *P. avium* trees that were virus free or infected with PNRSV, PDV and PDV + PNRSV in the nursery (1990). In 1993 as well as in 1994 the number of blind nodes was significantly higher in trees infected with PDV and PDV + PNRSV. PDV enhances of bare wood formation. Bare wood formation was more severe on the weak growing Inmil rootstock.

Flower bud formation

Infection with PDV generally increased flower bud formation as seen by in increase in the density of generative spurs (Table 2). This was most evident in Lapins, particularly on Inmil. In Sam the increase in generative spur density was not always significant although the trend is still observed, especially with the PDV + PNRSV infection. The increase in generative spur density is probably correlated with the less growth observed with the virus infection, however, it should be noted that the increased generative spur density did not compensate the lack of bearing positions due to reduced tree volume. On a whole tree basis virus infected trees produced less flowers (data not presented). This was most noticeable on trees infected in the nursery where growth was the most reduced.

Pollen germination

Fruit set and yield data were not recorded in this trial but the negative effect of the infections on tree size clearly reduced yield (personal observation). This supports other findings (BLAŽEK et al. 1981). The higher flower density did not compensate. Pollen germination was tested in 1993 and PDV negatively influenced pollen germination (Table 3). Germination of the pollen grains infected with PDV was ca. 30% less than virus free pollen.

Natural infection of virus free trees

The virus infections spread rapidly from inoculated to virus free trees, especially PNRSV. The transfer of

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Cultivar/rootstock	Virus treatment	1992		1993		1994	
Lapins/Damil	virus free	9.8	b	5.8	a	5.1	b
	PNRSV	11.1	ab	6.3	a	5.3	b
	PDV	13.1	ab	7.1	a	7.9	a
	PDV + PNRSV	15.2	a	7.8	a	6.5	ab
Lapins/Inmil	virus free	13.9	a	15.0	b	7.0	b
	PNRSV	22.9	a	9.2	b	9.4	b
	PDV	17.9	a	34.3	a	54.4	a
	PDV + PNRSV	–		–		–	
Sam/Inmil	virus free	22.0	b	12.6	a	15.5	a
	PNRSV	25.3	b	17.1	a	15.6	a
	PDV	68.4	a	16.4	a	14.2	a
	PDV + PNRSV	31.9	b	29.6	a	20.3	a

Means separation within columns for each cultivar/rootstock combination by Duncan's multiple range test ($P = 0.05$)

Table 3. The effect of virus infection in the nursery (1990) or in the orchard (1992) on the percentage of pollen germination in spring 1993

Cultivar/rootstock	Virus treatment	Year of infection			
		1990		1992	
Lapins/Damil	virus free	17.8	b	20.6	a
	PNRSV	25.2	a	19.3	a
	PDV	9.6	c	12.5	b
	PDV + PNRSV	18.0	b	16.5	ab
Sam/Inmil	virus free	23.4	a	36.4	a
	PNRSV	21.2	ab	39.5	a
	PDV	17.2	b	22.6	c
	PDV + PNRSV	18.1	ab	29.2	b

Means separation within columns for each cultivar/rootstock combination by Duncan's multiple range test ($P = 0.05$)

infected pollen spreads these viruses from tree to tree. ELISA testing in the summer in 1993 of initially virus free trees indicated that 6% were infected with PDV and 35% with PNRSV. Viruses were again tested in spring 1995 when 10% were infected with PDV and 48% with PNRSV. These viruses spread faster in sweet cherries than in sour cherries (ANDERSONE et al. 2002).

DISCUSSION

The vegetative growth of sweet cherry trees was inhibited by the virus infections. PDV had the most negative effect and inhibited growth in all scion/rootstock combinations. Only when inoculated early (in the nursery) the combination PDV + PNRSV had a more inhibitory effect. PNRSV on its own only inhibited growth of the weak growing Lapins/Inmil combination. In the more vigorous Sam/Inmil and Lapins/Damil combinations PNRSV had little inhibitory effect. The extent of growth reduction of sweet cherries by PDV and PDV + PNRSV was dependent on the scion/rootstock combination. The least vigorous Lapins/Inmil combination was most severely affected and the most vigorous Lapins/Damil combination the least. In these trials the observed growth reductions were not compensated for by increased generative tendencies.

Our findings contrast those of LANG et al. (1998) who tested the sensitivity of Bing on different rootstocks in the testing programme in Washington State, USA. They found Bing on Inmil to be tolerant to PDV + PNRSV infection, and Bing on Damil to be sensitive. Differences in sensitivity to virus infection are known to vary with *Prunus* species (LANG et al. 1998; UPHOFF et al. 1988). Rootstocks from the cross breeding of species can have tolerance. For example, the rootstock Gisela 5 has tolerance to PDV and PNRSV, while other rootstocks from the same crossing of *P. cerasus* × *P. canescens* are sensitive to both viruses. This makes the growth reaction of cherry cultivars on different rootstocks difficult to predict, especially in the presence of potential virus infection.

These findings and those reporting our work on sour cherry (*P. cerasus* L.) confirm that while the virus infection of virus free trees by pollen transfer was more rapid sweet cherry than sour cherry, these viruses had less detrimental effects on vegetative and reproductive growth of sweet cherry trees than sour cherry trees (DESVIGNES 1990; ANDERSONE et al. 2002). PNRSV presence generally did not inhibit the growth of sweet cherries. Sweet cherries are sensitive to PDV, but the growth reduction was mostly less than observed in our sour cherry trials.

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Vliv kontaminace viry na vegetativní a generativní růst třešní na podnožích Damil a Inmil

ABSTRAKT: U třešní (*Prunus avium* L.) byl zkoumán vliv infekce viry *Prunus necrotic ringspot virus* (PNRSV) a *Prune dwarf virus* (PDF) na vegetativní a generativní růst stromů. Infikované stromy byly menší a intenzita jejich růstu byla snížena – silněji u odrůdy Lapins než u odrůdy Sam, když byly stromy infikovány ve školce (v raném stadiu) oproti infekci o dva roky později v sadu a když byly stromy naštěpovány na zakrslé podnoži Inmil při porovnání se vzrůstnější podnoží Damil. Úhyn stromů po vysazení do sadu nebyl problémem, protože během pětiletého sledovaného období uhynul pouze jeden strom nakažený virem. Redukovaná intenzita růstu stromů byla doprovázena zvýšeným vyholováním větví. I přes vyšší hustotu plodných plodonošů měly celkově infikované stromy menší počet plodonošů a v důsledku toho i nižší výnos než kontrolní bezvirózní stromy. Navíc byla u infikovaných stromů redukována klíčivost pylu. Obecně infekce PNRSV neměla žádný nebo jen statisticky nevýznamný vliv na vegetativní a generativní projevy stromů, kdežto infekce PDV obě tyto charakteristiky významně redukovala. Kombinovaná infekce oběma viry měla výraznější vliv, zvláště pokud byla použita zakrslá podnož Inmil a citlivá odrůda Lapins. Po čtyřech letech růstu v sadu bylo infikováno 10 % původně bezvirózních stromů PDV a 48 % stromů PNSRV.

Klíčová slova: třešně; viry; *Prunus avium* L.; *Prune dwarf virus*; *Prunus necrotic ringspot virus*; růst stromů; výnosy

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