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Short Notes

Pea seed-borne mosaic virus pathotypes isolated from Australian pea (*Pisum sativum*) seed

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Summary. Pea seed lots (144) from Australian farms and research trials were assessed for pea seed-borne mosaic virus (PSbMV) seed transmission rates. High infection rates (up to 40%) were detected, particularly in the widely grown and highly PSbMV susceptible variety 'Kaspa', with only 12 out of 54 seed lots found to be free of the virus. PSbMV strains were isolated from 15 infected seed lots, and were pathotyped on a set of homogeneous *Pisum sativum* PSbMV differentials. Of the four pathotypes identified, P1 and P4 (eight and 20 isolates, respectively) were earlier reported in Australia. Pathotype P3, as yet unreported in Australia, was the most frequently identified pathotype (42 isolates). One PSbMV isolate was identified as the P2 pathotype, which has previously been isolated only from lentil seed. None of the isolated pathotypes could overcome the PSbMV resistance gene *sbm1*. The relevance of these findings for field pea breeding programmes and genomic studies of PSbMV are discussed.

Keywords. PSbMV, BYMV, virus resistance, pathogenicity test.

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INTRODUCTION

Pea seed-borne mosaic virus (PSbMV, *Potyvirus pisumsemenportati*) is present in field pea (*Pisum sativum*) in many countries, probably because the virus is seed transmitted at high rates. Distribution of this virus, seed transmission, host and vector range, and symptomatology have been reviewed extensively (Khetarpal and Maury, 1987; Congdon, 2017). Depending on the pea genotype, PSbMV symptoms can be difficult to identify in the field, and serological or molecular tests are required to determine its presence in host plants.

Cultivation of field pea, and of winter pulses in general, is recent in Australia (Siddique and Sykes, 1997), and it was not until 1991 that PSbMV was reported in commercial pea crops (Ligat *et al.*, 1991). Later surveys showed that the virus is widespread in Australia's pea crops, both in the western (Latham and Jones, 2001a, Congdon *et al.*, 2016) and eastern (Freeman *et al.*, 2013) regions of the country.

PSbMV causes considerable yield losses in Australian field peas, despite the generally mild symptoms it causes (Coutts *et al*., 2009), and the level of PSbMV infection in a pea crop is closely related to the level of seed-transmission in the seed stock used (Coutts *et al*., 2009; Freeman *et al*., 2013). Use of virus-free seed would therefore provide adequate control of PSbMV-induced yield losses. However, the virus can be introduced from neighbouring infested fields, and Australian pea growers generally save their own seed and rarely test the seed stocks for virus presence. Host resistance will provide a lasting control option, and the Australian field pea breeding programme has made the incorporation of PSbMV resistance in new varieties a priority (Rosewarne, 2016).

Breeding peas for PSbMV resistance is facilitated by the availability of single recessive genes in the host that provide immunity, and these can be identified in seedling tests using mechanical inoculations. Provvidenti and Alconero (1988) proposed four independent recessive resistance genes, *sbm1*, *sbm2*, *sbm3* and *sbm4*, to explain the differential reactions to the three PSbMV pathotypes, P1, P4 and P2, reported at the time (Alconero *et al*., 1986). The gene *sbm1* conferred resistance to P1, *sbm2* and *sbm3* conferred resistance to P2, and *sbm4* gave resistance to P4. These authors also reported strong linkages between gene *sbm2* and the *mo* resistance gene for bean yellow mosaic virus (BYMV) and between *sbm1*, *sbm3* and *sbm4* resistance genes. Johansen *et al*. (2001) showed that the differences between the three PSbMV pathotypes could be explained by the properties of two viral cistrons, and predicted the existence of a fourth pathotype, P3. The P3 pathotype was subsequently isolated from seed of a faba bean gene-bank accession originating from Nepal (Hjulsager *et al*., 2002). Gao *et al*. (2004) showed that only two recessive resistance genes were operating in the pea/PSbMV pathosystem. The *sbm1* gene (present in a range of germplasm of Indian and Ethiopian origin), confers resistance to all four PSbMV pathotypes (P1, P2, P3, P4), while a different allele of the *sbm1* gene, $sbm1^1$ (present in the germplasm accessions PI 269774 and PI 269818), gives resistance only to the P1 and P2 pathotypes, and the *sbm2* gene (in 'Dark Skin Perfection' and a large number of commercial pea lines) only provides resistance to pathotypes P2 and P3. The use of two differentials, one with the $sbm1^1$ gene and one with the *sbm2* gene, allows the classification of PSbMV to one of the four pathotypes (Table 1).

Within Australia, limited attention has been given to pathotyping of PSbMV strains. Ligat and Randles (1993) pathotyped three Australian isolates as P1 (one isolate) or P4 (two isolates), using a differential set of six pea genotypes. Torok and Randles (2007) pathotyped 14 Australian PSbMV isolates, and classified ten as P4 and four as the P1 pathotype.

Table 1. Differentiation of four PSbMV pathotypes by two pea genotypes with specific resistances (modified from Johansen *et al*., 2001, and Gao *et al*., 2004).

		sbm2 differential	
		Resistant	Susceptible
$sbm1^1$ differential	Resistant	P2	P1
	Susceptible	P3	P4

For pea breeding programmes to successfully incorporate PSbMV resistance, knowledge is required of the composition of the local PSbMV strain population. The present study isolated PSbMV strains from pea seed originating from geographically distinct locations in Australia, and pathotyped the isolates using a differential set of homogeneous pea genotypes.

MATERIAL AND METHODS

Tissue blot immunoassays (TBIA) were used for all virus diagnostics, as these provide a reliable, rapid, and cost-efficient methodology for processing large numbers of plant samples (Freeman *et al*., 2013; van Leur *et al*., 2013a). Samples were each blotted onto nitrocellulose membranes (Schleiger & Schuell Protran, 0.45 µm pore size), and were processed using a polyclonal PSbMV antibody (DSMZ, AS-0129), following the procedures described by Kumari *et al.* (2022).

PSbMV seed-to-plant transmission (PSbMV-SPT) rates were determined from 144 seed lots (92 harvested from farmer fields and 52 from trial fields), submitted to the Tamworth laboratory for PSbMV seed testing during 2006-2010. 'Kaspa' was the predominant cultivar in this evaluation, with 54 seed lots, followed by 'Excell' (18 seed lots), 'Morgan' (12), and 'Parafield' (nine).

Seeds were incubated on wet filter paper at 22°C in the dark for 7-10 d, after which plumules or radicles of the germinated seeds were tested for PSbMV presence by TBIA. Seed lots were first tested in batches of 30 seeds. A sequential sampling approach was later taken, with further tests made on seed lots that had low infection levels. For most seed lots, more than 90 seeds were tested. The PSbMV-SPT rate was calculated as:

100 × (total number of PSbMV positive germlings / total number of germinated seeds).

To isolate PSbMV strains, 20 seed lots were selected. This selection was based on PSbMV-SPT rates, but also included a range of varieties and seed sources, from three Australian states New South Wales (NSW), Western Australia (WA) and Victoria. For each selected seed lot, 20 small pots were sown with three seeds/pot, using a commercial potting mix, and the pots were then held in an aphid-proof, temperature-controlled (18–24°C) greenhouse. Two to 3 weeks after sowing, all emerged plants were tested by TBIA, and PSbMV positives were selected. The PSbMV strains were isolated by inoculating 2- to 3-week-old plants of the faba bean variety 'Fiesta' with individual PSbMV positive seedlings, using the inoculation methods described below. PSbMV presence in the faba bean plants was confirmed with TBIA prior to pathogenicity tests.

Three PSbMV pea lines with reported single resistance genes (Hjulsager *et al*., 2002; Gao *et al*., 2004), including PI 269774 (*sbm1*[1] gene), Dark Skin Perfection (*sbm2*) and PI 193835 (*sbm1*) were obtained from the Australian Grains Genebank (Horsham, Australia). To ensure host plant homogeneity, single plant selections were made from these lines prior to testing. For pathogenicity tests, four to five plants each of the three differentials, and a universally susceptible pea line ('Kaspa') were inoculated 10 to 14 d after sowing, by dusting first and second leaves of each plant with carborundum powder (silicon carbide # 400), and rubbing into the leaves a virus suspension from young virus-infected faba bean leaves. The suspension was prepared by homogenisation of the faba bean leaves in a cold 0.1 M sodium phosphate buffer (pH = 7.0). Each inoculation was repeated after one week on the third leaves of the plants. Two weeks after the second inoculation, the plants were TBIA-tested for virus presence. Virus isolates that gave variable results, or failed to infect the susceptible 'Kaspa' plants, were retested.

RESULTS AND DISCUSSION

PSbMV seed infections were common in the 144 submitted seed lots assessed, with 52 seed lots PSbMV-free and 47 having PSbMV-SPT rates greater than 5% (Table 2). 'Kaspa' and 'Excell' were found to be particularly susceptible to PSbMV seed infection, with only 12 (22%) 'Kaspa' seed lots PSbMV-free and 25 (46%) with infection levels greater than 5%. For 'Excell', five (27%) seed lots were PSbMV-free and six (33%) had infection levels greater than 5%. In contrast, ten out of 12 seed lots of 'Morgan' were PSbMV-free, although this variety does not contain any of the *sbm* genes for PSbMV resistance (van Leur *et al*., 2013a). The greatest PSbMV-SPT rate, 40%, was found in an 'Excell' seed lot from a trial plot at the Wagga Wagga Agricultural Institute, NSW. The greatest 'Kaspa' infection, 25%, was from a trial plot at the Plant Breeding Institute at Narrabri, NSW. Greater infection levels were found in seed originating from research stations as compared to farmer fields; out of nine 'Kaspa' seed lots harvested from trial fields, four (44%) had PSbMV-SPT levels >10%, while nine out of 45 'Kaspa' seed lots (20%) harvested in farmer fields had similar infection levels. Similarly, for all seed lots combined, 37 of 52 (71%) seed lots harvested from trial fields, and ten of 92 (11%) from farmer fields, had PSbMV-SPT levels >10%. These differences are probably a result of research stations growing a wide range of germplasm, including highly susceptible genotypes that can be inoculum reservoirs for viruses and virus vectors.

Table 2. Pea seed-borne mosaic virus seed-to-plant transmission (PSbMV-SPT) rates for 144 pea seed lots, tested during 2006 to 2010.

Variety	Harvested from farmer fields					Harvested from trial fields					Grand total
	Number of seed lots / PSbMV-SPT category				Total seed lots	Number of seed lots / PSbMV-SPT category				Total seed lots	
	0%	>0% ≤5%	>5% ≤10%	>10%		0%	>0% ≤5%	>5% ≤10%	>10%		
Kaspa	9	15	12	9	45	3	2	0	4	9	54
Excell	4	5	5	1	15	1	1	0	1	3	18
Morgan	9	1	1	0	11	0	1	0	0	1	12
Parafield	4	3	0	0	7	0	1	0	1	2	9
Other varieties[a]	8	5	1	0	14	14	11	8	4	37	51
Total	34	29	19	10	92	18	16	8	10	52	144

[a] Other varieties: 'Alezan', 'Alma', 'Bluey', 'Bonzer', 'Bundi', 'Collegian', 'Celine', 'Cooke', 'Cressy Blue', 'Derrimut', 'Dun', 'Dundale, 'Dunwa', 'Early Dunn', 'Glenroy', 'Helena', 'Jupiter', 'Laura', 'Moonlight', 'Mukta', 'Santi', 'Snowpeak', 'Soupa', 'Sturt', 'Wirrega'.

Table 3. Origins and pathotypes of the PSbMV isolates detected in this study.

Lot No.	Pea variety	Origin, State	Harvest year	% PSbMV-SPT	Isolates pathotyped	Isolates per pathotype			
						P1	P2	P3	P4
1	Kaspa	Farmer field, WA	2008	16	6			4	2
2	Kaspa	Farmer field, WA	2008	39	10			7	3
3	Kaspa	Farmer field, southern NSW	2010	3	2				2
5	Bluey	Trial field, Wagga Wagga, NSW	2005	13	4			1	3
7	Moonlight[a]	Trial field, Wagga Wagga, NSW	2007	13	4	3			1
8	Alezan	Trial field, Narrabri, NSW	2008	12	5	2		2	1
9	Parafield	Trial field, Horsham, Victoria	2006	14	4			3	1
10	Excell	Trial field, Wagga Wagga, NSW	2006	52	10	1		5	4
11	Kaspa	Trial field, Narrabri, NSW	2008	25	8	1		6	1
12	Kaspa	Farmer field, southern NSW	2008	15	7			6	1
13	Dundale	Trial field, Wagga Wagga, NSW	2005	19	4		1	2	1
14	Kaspa	Farmer field, Scaddan, WA	2006	20	2			2	
15	Parafield	Farmer field, Esperance, WA	2006	9	1			1	
16	Excell	Farmer field, southern NSW	2008	9	3			3	
18	Soupa[a]	Trial field, Wagga Wagga, NSW	2006	8	1	1			
Total number of isolates tested/pathotyped					71	8	1	42	20

[a] Indicates varieties resistant to bean yellow mosaic virus (van Leur *et al*., 2013a).

Out of the 20 selected seed lots, five did not yield PSbMV. From the remaining 15 seed lots, 88 PSbMV strains were isolated of which 71 were pathotyped (Table 3). None of the isolates infected PI 193835, confirming the effectiveness of the *sbm1* gene against all known PSbMV pathotypes. Both the P1 pathotype (with eight isolates identified), and the P4 pathotype (20 isolates identified), were previously reported in Australia (Ligat and Randles, 1993; Torok and Randles, 2007). The most frequently found pathotype was P3, with 42 isolates able to infect PI 269774 (containing *sbm1*^{1}), but not 'Dark Skin Perfection' (containing *sbm2*). The pathotype P3 has not been recorded in Australia previously, but previous studies (Ligat and Randles, 1993; Torok and Randles, 2007) may have missed this pathotype because they did not use a reliable *sbm2* differential. Ligat *et al.* (1991) compared symptom development of four Australian PSbMV isolates with an American isolate on a range of pea varieties, and noted that the Australian isolates did not infect the BYMV resistant pea variety 'Greenfeast'. This could indicate that the Australian isolates were P3 pathotypes.

Identification of a single P2 pathotype strain (isolate Ps11-13/19) from a 'Dundale' seed lot originating from the Wagga Wagga Agricultural Institute (NSW) field trial was unexpected. To date, P2 pathotypes have only been isolated from lentil seed (Hampton, 1982; Alconero *et al*., 1986; van Leur *et al*., 2013b). Wylie et al. (2011) reported a P2 pathotype (isolate W1) obtained from a pea plant grown at the Medina Research Station near Perth, WA (Latham and Jones, 2001a). Latham and Jones (2001b) noted severe PSbMV symptoms in germplasm plots at this station that could have been caused by exotic virus strains, and the W1 strain could have originated from imported lentil germplasm. Ashby *et al.* (1986) pathotyped a PSbMV strain isolated from peas in New Zealand using *sbm1*, *sbm1*^{1}, and *sbm2* differentials, and found that the strain reacted as a P2 pathotype. PSbMV is common in New Zealand lentil crops (Fletcher, 1993), and cross-infection of pea crops from lentil seed-borne PSbMV could have occurred. While isolate Ps11-13/19 originated from a pea seed, the seed was harvested at a research station where a wide range of legume germplasm, including lentils, is grown. The infection of the mother plant could have originated from a seed-infected lentil plant.

Identification of P3 and P2 pathotypes depends on their inability to infect *sbm2* differentials. Alconero *et al.* (1986) noted that their P4 strain had delayed and erratic infection in pea lines that had the *mo*/*sbm2* gene. The present study showed that infection of the 'Dark Skin Perfection' differential was occasionally not detected on all inoculated plants, despite the use of homogenous differentials derived from single seed progenies. However, the identities of the P2 and P3 pathotypes were confirmed with repeated inoculations of 'Dark Skin Perfection' and pea varieties such as 'Greenfeast' and 'Bundi', that showed BYMV resistance in previous trials (van Leur *et al*., 2013a).

Pathotyping of PSbMV strains is required for pea breeding programmes, but these biological tests are time consuming. Developments in the gathering and analyses of genetic information using molecular methods may enable rapid PSbMV pathotype identification. To date, phylogenetic analyses of PSbMV strains have been achieved for limited numbers of virus isolates. Safarova *et al.* (2008) examined eight pathotyped PSbMV isolates from the Czech Republic, and showed distinct grouping of P1 and P4 pathotypes. Wylie *et al.* (2011) analysed six pathotyped Australian PSbMV isolates; their P1 and P4 isolates clustered in two separate groups together with the Czech strains of the same pathotype, but their P2 isolate did not group with the published P2 pathotype strain (PSbMV-L1 isolate, GenBank accession code: AJ252242). Further development of pathotyping techniques would require analyses of large numbers of PSbMV strains for each pathotype, and the use of strains from diverse geographic backgrounds. Until that is achieved, pathotype identification should be based on biological indexing, and care must be taken not to propose pathotypes based solely on phylogenetic analyses.

The outcome of the present study is particularly relevant to pea breeding programmes. Firstly, the study confirmed the effectiveness of the *sbm1* gene against all four PSbMV pathotypes. This gene is widely used in the Australian pea breeding programme, and is present in currently grown varieties such as 'Yarrum' and 'PBA Wharton', although none of the commercial varieties are homogeneous for resistance (van Leur *et al.*, 2013a). Pea varieties that are heterogeneous for PSbMV resistance could provide enough protection for commercial purposes, but these varieties will require purification before being used as parents in breeding programmes. Secondly, given the prevalence of P3 pathotypes, varieties carrying the *mo/sbm2* gene may appear to be resistant to PSbMV. However, cultivation of these cultivars will result in selection for the P1 and P4 pathotypes, as is demonstrated by the absence of P3 pathotypes in the two tested seed lots harvested from the BYMV resistant varieties 'Moonlight' and 'Soupa'.

The present study results have shown how quickly PSbMV can spread. They also provide clear indications for differences in PSbMV seed transmission rates among host varieties that lack *sbm* resistance genes. While highly susceptible varieties (e.g. 'Kaspa' and 'Excell') are being replaced with new varieties, some carrying the *sbm1* gene, it remains important to monitor commercial seed lots for PSbMV transmission to identify varieties with high seed transmission rates for PSbMV.

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