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Relative tolerance of nine olive cultivars to *Pseudomonas savastanoi* causing bacterial knot disease

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Summary. Reactions of nine olive cultivars inoculated with olive or oleander strains of *Pseudomonas savastanoi* were evaluated under glasshouse conditions. Two quantitative indices of pathogenicity were used: 1. gall size from standardised inoculations, measured after 2 months; 2. rate of change of gall size with increasing inoculum concentration. These two indices were combined in a single plot that appears to give the best resolution of all data. Responses of olive cultivars to olive strains of *P. savastanoi* suggest that cultivars Carolea, Koroneiki, Leccino and Pendolino are the most tolerant, and that Barnea, Manzanillo, Picholine, Picual and a South Australian selection of Verdale are the least tolerant. Olive and oleander strains of *P. savastanoi* differed in virulence, and between-plant variability in reaction to inoculation was noted. Hypersensitive reactions do not appear to be part of mechanisms of tolerance. Olive strains of *P. savastanoi* affected all cultivars more severely than did equivalent inoculations with oleander strains.

Introduction

Pseudomonas syringae pv. *savastanoi* was previously regarded as the single pathogen responsible for bacterial knot disease in olive, oleander, ash and other hosts (Bradbury, 1986). Olive knot and oleander knot are now considered to be diseases caused by distinct pathogenic populations in *P. savastanoi* (Janse, 1982; Caponero *et al*., 1995; Young *et al*., 1996), referred to here as olive strains or oleander strains of *P. savastanoi*.

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Olive trees have been grown in New Zealand for more than a century, mostly as specimen ornamentals, with some amateur production of fruit for processing. Commercial olive groves for oil and for fruit have been a recent initiative in New Zealand. When the oleander knot pathogen was recorded in New Zealand (Dye, 1956) it was believed that this pathogen also caused olive knot disease. The specific olive knot pathogen was first detected in New Zealand in 1997 (Braithwaite *et al*., 1999). Subsequent disease outbreaks occurring in olives with origins that could not be linked to the first recorded outbreak suggested the possibility of more than one introduction of this pathogen (Braithwaite *et al*., 1999).

In the Northern Hemisphere, strategies to

minimize the severity of disease depend on attention to programmes for pruning, irrigation and fertilizer application. There is no cost-effective way to control knot in olive groves using currently available bactericidal sprays (Young, 2004). Sisto and Iacobellis (1999) noted the need for identified tolerant varieties of olive as a way to minimise the effects of the disease, but at present no commercial cultivars have been identified that are usefully tolerant (Iacobellis, 2001). Few quantitative studies of susceptibility of olive cultivars to bacterial knot pathogens have been reported (Varvaro and Surico 1978a; Sisto *et al.*, 2001). Because of the potential significance of bacterial knot to the olive industry, the relative tolerance of those cultivars currently favoured for the development of the industry in New Zealand was investigated.

Materials and methods

Bacterial strains and pathogenicity tests

Strains of *Pseudomonas savastanoi* used in this study were from the International Collection of Micro-organisms from Plants (ICMP), Landcare Research, Auckland, New Zealand (www.landcareresearch.co.nz/research/biodiversity/fungiprog/icmp.asp). Olive strains ICMP 13519 and 13813 were chosen on the basis of their proved pathogenicity to olive cv. Barnea but lack of pathogenicity to oleander (pink ornamental cultivar), and oleander strains ICMP 791 and 13815 were chosen on the basis of their proved pathogenicity to both these cultivars (unpublished data).

Olive cultivars included in the study were chosen in consultation with New Zealand growers: Barnea, Carolea, Koroneiki, Leccino, Manzanillo, Pendolino, Picholine, Picual and a South Australian selection of Verdale ('Verdale'). Plants used were single-stemmed, nursery stock (60–80 cm high), grown from rooted cuttings.

Bacterial inoculum was harvested from slope cultures incubated at 25°C for 2 days on yeast-extract phosphate salts agar (NH_4Cl, 0.5 g; KCl, 0.2 g; $MgSO_4 \cdot 7H_20$, 0.2 g; K_2HPO_4, 1.0 g; yeast extract [Difco Laboratories, Detroit, MI, USA], 3.0 g; agar [Davis, Germantown (NZ) Co., Marukan, NZ] 12 g; de-ionised water, 1 l) and suspended in sterile de-ionised water. Inoculum turbidity was standardized using a nephelometer and calibrated by plate counts of decimal dilution series.

Inoculations were made in the first week of December (the beginning of the Southern hemisphere summer). Each strain of the olive pathogen and of the oleander pathogen was inoculated at four different bacterial concentrations (two plants per concentration) into each olive cultivar (a total of 32 plants per cultivar). For each cultivar, two additional control plants were inoculated with sterile de-ionised water. Plants were inoculated by pricking stems at three sites in every internode of each plant using hypodermic needles charged with suspension. Four, thirty-three fold, dilutions, calibrated at 10^8, 3.3×10^6, 10^5 and 3.3×10^3 cfu ml^{-1}, were inoculated.

Young (1991) showed how the volume of suspension introduced into plants by artificial inoculation could be determined, and hence that the absolute numbers of bacteria present at inoculated sites could be estimated. Using this approach, estimated volumes of 2.5×10^{-4}ml were injected in each inoculation, translating to dilutions as 2.5×10^4, 800, 25, and 1 cfu per inoculation site.

After inoculation, plants were randomised by cultivar and held in a glasshouse with each replication held in a separate unit.

Gall size recording

Glasshouse temperature was recorded at 2–3 day intervals. Plants were maintained with a suitable water and fertilizer regime. Gall size was measured after two months. After this recording, all plants were held in the glasshouse for a further eight months, with watering but without regular temperature recording. Ten months after inoculation, gall diameter on plants inoculated with ICMP 13519, calibrated at 3.3×10^6 cfu ml^{-1}, was measured. Only one series of cv. Manzanillo was counted because the other had been sacrificed for other studies (unpublished data).

Mean daily temperatures for the initial 2-month period were 21.5±1.7°C, with a maximum temperature of 26.9±2.2°C, and a minimum temperature of 16.1±1.9°C. The temperature range over the following 8-month period was 6–27°C.

Analysis of results

Three approaches to the assessment of cultivar responses were considered:

1. The mean diameters of galls developing on the different cultivars inoculated with olive strains standardised at 3.3×10^6 cfu ml^{-1} were compared. The *lme* function of Splus (2002) was used to fit separate variances for between-plant and within-plant variability, with within-plant variance being proportional to the cultivar mean.

2. Cultivar response was also measured using a variation of that reported by Hu *et al*. (1997). The effect of inoculation was assessed as the increase in mean lesion size between inoculated and control treatments, to correct for the differing reactions between cultivars to mechanical wounding. A mixed model was fitted to the data using REML in the statistical package Genstat 6.1 (Genstat Committee, 2002), regressing gall diameter against the log of inoculum concentration, with separate regressions for each plant randomly varying about a mean value for each strain. A correction for the effect of mechanical wounding was applied by subtracting the mean gall diameters of the control plants (data not shown) from the mean value of inoculations for each dilution, as an offset.

3. Data from these two approaches were combined by plotting fitted gall diameters of olive strains inoculated at 3.3×10^6 cfu ml^{-1} against the gradient of increase in gall size / 10-fold increase in inoculum.

Results

Data recorded two months after inoculation

Correlations based on the diameter, area (as diameter^2), or volume (as diameter^3) of galls, versus bacterial inoculum dose, were compared. Gall diameter versus logarithm of bacteria inoculated gave the best linear correlation when each cultivar was considered independently. Gall diameter was therefore chosen as the basic parameter to define disease severity.

1. Separate variances for between-plant and within-plant variability, with within-plant variance were shown to be proportional to the cultivar mean. The variability of cultivars increased with their means, and the fitted model assumed that the variance was proportional to the mean.

Mean gall diameters in all cultivars inoculated with olive and oleander strains at 3.3×10^6 cfu ml^{-1} with their standard errors are shown (Table 1). Mean lesion size varied between strains for most cultivars. Those cultivars where the *P*-value for a test of difference in fitted gall diameters between strains is <0.05 are shown as shaded. For each cultivar, galls induced by olive strains were larger than those induced by oleander strains (*P*-value < 0.001).

Gall diameter values resulting from inoculation with the olive strains and with the oleander strains were separately combined (Table 2). For olive strains, there was a range of gall sizes, ordered

Table 1. Mean gall diameter (mm) in nine olive cultivars inoculated with *P. savastanoi* olive knot strains ICMP 13519 and 13813 and *P. savastanoi* oleander knot strains ICMP 791 and 13815 at concentrations of 3×10^6 cfu ml^{-1}, recorded after 2 months. Mean diameters with their standard errors are indicated. Values in bold indicate significantly different reactions between strains in each host.

Cultivar	Olive				Oleander			
	ICMP 13519		ICMP 13813		ICMP 791		ICMP 13815	
	Diameter	S.E.	Diameter	S.E.	Diameter	S.E.	Diameter	S.E.
Manzanillo	**2.10**	**0.073**	**1.53**	**0.059**	0.87	0.058	0.82	0.059
Picholine	**2.07**	**0.073**	**1.64**	**0.073**	**0.48**	**0.047**	**0.65**	**0.067**
Verdale	**1.69**	**0.060**	**1.42**	**0.086**	**0.19**	**0.032**	**0.12**	**0.028**
Barnea	1.68	0.087	1.69	0.076	**1.40**	**0.083**	**1.16**	**0.061**
Picual	**1.57**	**0.041**	**1.27**	**0.044**	**0.33**	**0.032**	**0.50**	**0.038**
Koroneiki	**0.93**	**0.053**	**0.62**	**0.044**	**0.16**	**0.03**	**0.27**	**0.038**
Leccino	0.90	0.039	0.87	0.047	**0.45**	**0.034**	**0.57**	**0.043**
Carolea	0.82	0.032	0.46	0.035	**0.37**	**0.036**	**0.07**	**0.018**
Pendolino	**0.76**	**0.053**	**1.05**	**0.056**	0.24	0.037	0.24	0.041

from largest to smallest, in the cultivars Picholine, Manzanillo, Barnea, Verdale, Picual, Pendolino, Leccino, Koroneiki and Carolea. For oleander strains the order was Barnea, Manzanillo, Picholine, Leccino, Picual, Pendolino, Koroneiki, Carolea and Verdale.

2. Regressions were fitted for both the olive and oleander inoculation series and rates of change in gall diameter / ten-fold change in inoculum concentration were estimated (Table 3). For olive strains, the rates differed between strains only for cultivars Carolea and Koroneiki (*P*-values 0.034, 0.029 respectively). For every cultivar, rates did not differ between oleander strains but were greater for the olive strains than for the oleander strains in each case (*P*-value <0.001). For the cultivars Carolea, Picholine and Verdale, the rates for the oleander strains did not differ from 0. The gradient values for the olive strains and for the oleander strains were combined for each cultivar (Table 4). For olive strains, combined values indicate a range, ordered from greatest to least rate of change in cultivars Manzanillo, Verdale, Barnea, Picual, Picholine, Koroneiki, Leccino, Pendolino and Carolea. For oleander strains, values indicate a range, from greatest to least rate of change, in cultivars Barnea, Manzanillo, Leccino, Picual, Pendolino, Koroneiki, Carolea, Picholine and Verdale.

3. Fitted gall diameters of olive strains inoculated at 3.3×10^6 cfu ml^{-1} were plotted against the gradient of increase in gall size / 10-fold increase in inoculum for olive strains (Fig. 1a) and oleander strains (Fig. 1b). For olive strains, the data indicate two groups of cultivars. The first group represented cultivars that expressed steep gradients and large galls: Barnea, Manzanillo, Picholine, Picual and Verdale. The second group represented cultivars that expressed shallow gradients and small galls: Carolea, Koroneiki, Leccino and Pendolino.

Table 2. Combined gall diameters (mm) in nine olive cultivars inoculated with *P. savastanoi* olive knot strains ICMP 13519 and 13813 and *P. savastanoi* oleander knot strains ICMP 791 and 13815 at concentrations of 3×10^6 cfu ml^{-1}, recorded after 2 months. Mean diameters with their standard errors are indicated.

Cultivar	Olive strains		Oleander strains	
	Diameter	SE	Diameter	SE
Picholine	1.87	0.053	0.56	0.041
Manzanillo	1.82	0.050	0.85	0.041
Barnea	1.68	0.058	1.27	0.051
Verdale	1.57	0.052	0.16	0.021
Picual	1.44	0.030	0.42	0.025
Pendolino	0.90	0.039	0.24	0.027
Leccino	0.89	0.030	0.52	0.028
Koroneiki	0.77	0.036	0.22	0.024
Carolea	0.65	0.025	0.22	0.022

Table 3. Gradients representing reactions of nine olive cultivars inoculated with *P. savastanoi* olive knot strains ICMP 13519 and 13813 and oleander knot strains ICMP 791 and 13815 recorded after 2 months. Values are expressed as change in gall diameter / ten-fold change in inoculum concentration. Mean gradients with their standard errors are indicated.

Cultivar	Olive				Oleander			
	ICMP 13519		ICMP 13813		ICMP 791		ICMP 13815	
	Gradient	SE	Gradient	SE	Gradient	SE	Gradient	SE
Manzanillo	0.83	0.043	0.73	0.036	0.23	0.035	0.31	0.036
Verdale	0.69	0.035	0.70	0.052	0.00	0.019	0.00	0.016
Barnea	0.64	0.048	0.69	0.045	0.31	0.049	0.30	0.037
Picual	0.59	0.024	0.64	0.029	0.07	0.019	0.09	0.021
Picholine	0.58	0.044	0.68	0.043	-0.02	0.028	0.04	0.038
Koroneiki	0.43	0.032	0.34	0.026	0.07	0.018	0.07	0.022
Leccino	0.37	0.023	0.33	0.028	0.22	0.021	0.25	0.026
Pendolino	0.36	0.030	0.44	0.033	0.10	0.022	0.07	0.025
Carolea	0.35	0.020	0.29	0.021	0.03	0.023	0.00	0.011

Table 4. Gradients of combined data representing reactions of nine olive cultivars inoculated with *P. savastanoi* olive knot strains ICMP 13519 and 13813 and oleander knot strains ICMP 791 and 13815 recorded after 2 months. Values are expressed as change in gall diameter / ten-fold change in inoculum concentration. Mean gradients with their standard errors are indicated. Values in bold indicate significantly different reactions between strains in each host.

Cultivar	Olive strains		Oleander strains	
	Gradient	SE	Gradient	SE
Manzanillo	0.78	0.030	0.27	0.025
Verdale	0.70	0.031	**0.00**	**0.013**
Barnea	0.66	0.033	0.30	0.031
Picholine	0.62	0.032	**0.01**	**0.024**
Picual	0.60	0.019	0.09	0.015
Pendolino	0.39	0.023	0.09	0.016
Koroneiki	0.38	0.021	0.07	0.014
Leccino	0.35	0.018	0.23	0.017
Carolea	0.31	0.015	**0.02**	**0.013**

Oleander strains caused reactions generally leading to small galls and low rates of increase with inoculum dose. Only in cultivars Barnea, Manzanillo and Leccino were reactions expressed to oleander strains that were comparable to those of tolerant olive cultivars to olive strains.

Fitted regressions of the dose response for olive and oleander, expressed as rates of change in gall diameter / ten-fold change in inoculum concentration (Table 3), were corrected to give absolute bacterial numbers (cfu) inoculated. The intersection of the slopes on the x-axis showed that cultivars in both groups inoculated with olive strains both expressed reactions to inoculation when bacterial numbers exceeded 3±0.2 cfu per inoculation site. The variability of reactions to oleander strains did not allow meaningful generalised dose-response curves to be derived, and no useful value for symptom initiation could be given.

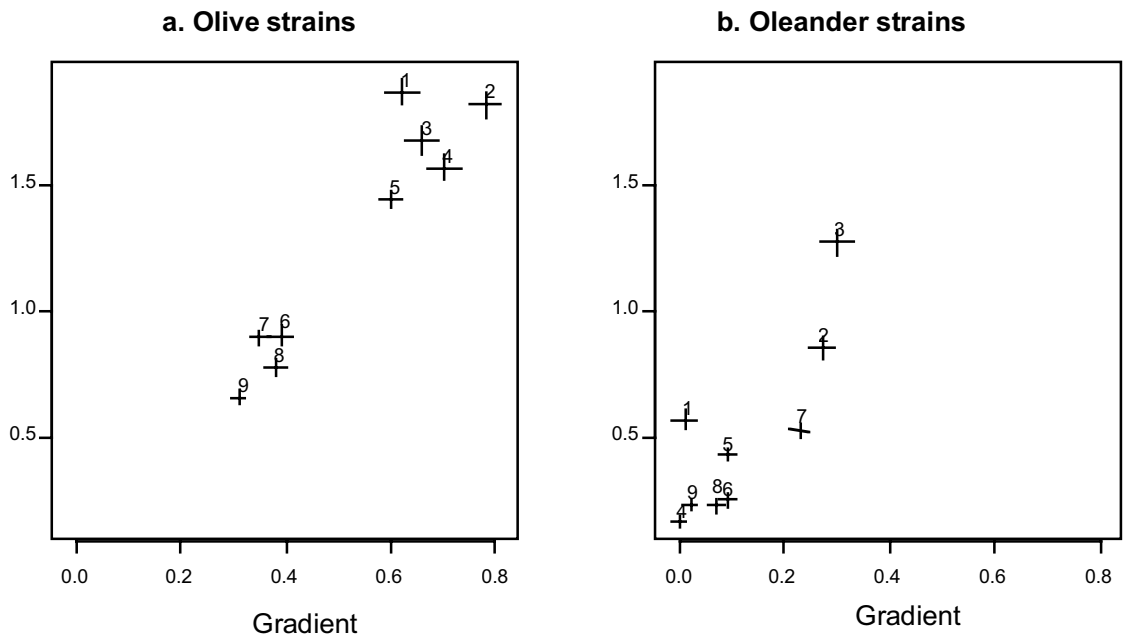

Fig. 1. Mean gall diameters (mm) in nine olive cultivars inoculated with *P. savastanoi* olive knot strains ICMP 13519 and 13813 (a), and oleander strains ICMP 791 and 13815 (b), at 3×10^6 cfu ml^{-1}, plotted versus gradient expressed as change in gall diameter / ten-fold change in inoculum concentration. Cultivar: 1, Picholine; 2, Manzanillo; 3, Barnea; 4, Verdale; 5, Picual; 6, Pendolino; 7, Leccino; 8, Koroneiki; 9, Carolea. Each bar represents ± standard error of estimated values of the gradient and gall diameter. If the bars for the gradients of two cultivars do not overlap, it is concluded that they are significantly different ($P<0.05$).

Table 5. Mean gall diameter (mm) in 9 olive cultivars inoculated with *P. savastanoi* olive knot strain ICMP 13519 at 3×10^6 cfu ml^{-1}, recorded after 10 months. Mean diameters with their standard errors are indicated.

Cultivar	Diameter	SE
Picholine	4.4	0.40
Verdale	4.2	0.40
Manzanillo	3.7	0.55
Picual	2.2	0.34
Leccino	1.8	0.33
Pendolino	0.5	0.28
Carolea	0.5	0.28
Koroneiki	0.2	0.27
Barnea	0.0	–
Control	0.0	–

Data recorded 10 months after inoculation

Analysis based on the mean diameter of galls developing from inoculations with ICMP 13519, standardised at 3.3×10^6 cfu ml^{-1}, is shown in Table 5. These data can be interpreted as indicating two cultivar groups:
first group (large galls): Leccino, Manzanillo, Picholine, Picual and Verdale,
second group (small galls): Barnea, Carolea, Koroneiki and Pendolino.

The *lme* function of Splus 6 (2002) was used to fit separate variances for between-plant and within-plant variability, with within-plant variance being proportional to the cultivar mean. The variability of reactions by cultivars increased with their mean.

Discussion

In the field the pathogen of olive knot, *Pseudomonas savastanoi*, causes galls that can develop to greater than 3 cm in diameter, growing over several months. In pathogenicity tests, however, time constraints usually demand shorter incubation periods with resultant smaller galls. In this study, primary recording was done after 2 months, following Janse (1982). Relative sizes of galls were taken as reflecting relative cultivar tolerance, large galls indicating low tolerance and small galls indicating high tolerance. Gradients expressed as change in gall diameter / ten-fold change in inoculum concentration were taken as reflecting cultivar tolerance, steep gradients indicating low tolerance and shallow gradients indicating high tolerance.

Mean gall size varied between strains of the olive and oleander pathogens (Table 1), indicating differences in virulence, as observed in other reports of host-pathogen interactions (Young, 1987; Crosse and Garrett, 1996; Hu *et al.,* 1997). Preliminary studies (unpublished data) showed that strains differed markedly in pathogenicity, from avirulence to severe knot production. In a study of this kind, the choice of strains used obviously could influence results. Ideally, many strains should be included. In practice, the number is usually small (Sisto *et al.*, 2001). An alternative could be to include many strains in an inoculum *mélange*. This option seemed to invite a number of uncertainties in interpretation until the reliability of the model could be proved. In this study, only two proved pathogenic strains were used and then treated as representative of a larger population of strains; that is to say, standard statistical methods were applied, although the selection of strains was non-random. For each olive strain, mean gall size varied between plants, indicating between-plant variation in tolerance to the pathogen (Table 1) but because the stock source of plants from which cuttings were derived was restricted, the sample is too small to draw any conclusions.

Two forms of analysis were considered. Gall size measured after a fixed period of inoculation has been used for the quantitative measurement of relative tolerance/susceptibility of olive (Sisto *et al*. 2001). This method was compared with the model developed by Hu *et al*. (1997). In their study of *Acidovorax avenae* in *Avena sativa* and *Zea mays*, fitted gradients, expressed as change in lesion diameter per inoculum concentration, were used to determine strain virulence. This model was here used as a measure of relative olive cultivar tolerance to *P. savastanoi*. For olive strains, fitted gall sizes were expressed as a range, from largest to smallest galls, in the cultivars Picholine, Manzanillo, Barnea, Verdale, Picual, Pendolino, Leccino, Koroneiki and Carolea (Table 2). This was essentially similar to the range expressed as the gradients of cultivar responses to the pathogen, expressed in order from steepest to shallowest, comprising Manzanillo, Verdale, Barnea, Picual, Picholine, Koroneiki, Leccino, Pendolino and Carolea (Table 4).

Measurement of gall size (by mass) and rates of change of gall size as indices of tolerance to olive knot have been reported elsewhere (Varvaro and Surico, 1978a). They also found a correspondence

between gall size and rate of increase in gall size with time. In the present study an additional step was taken in that these data were combined in a single comparative plot of gall size versus gradient (Fig. 1). This analysis showed that cultivars could be placed in two groups. The first group: Barnea, Manzanillo, Picholine, Picual and Verdale, consisted of cultivars that expressed steep gradients and large galls that indicated susceptibility. The second group: Carolea, Koroneiki, Leccino and Pendolino, consists of cultivars that expressed shallow gradients and small galls that indicates tolerance or resistance. This approach appears to offer a robust method of assessing cultivar performance.

Gall diameters resulting from inoculation at concentrations of 3×10^6 cfu ml^{-1} recorded after 10 months largely confirmed this pattern for relative tolerance to olive strains, with cultivars Manzanillo, Picholine, Verdale, Picual and Leccino being least tolerant, and Barnea, Carolea, Koroneiki and Pendolino being most tolerant.

Results recorded here for cv. Barnea show it to be less tolerant to olive strains after 2 months and then more tolerant at 10 months. The reaction of this cultivar to inoculation did not result in typically raised galls. Instead, after 2 months, broad, flat lesions, slightly raised above the surrounding epidermis were apparent, and these were scored according to their diameter. After 10 months, associated with the expansion of the stems and the epidermis, these lesions became less obvious and were generally not recordable as erumpent galls.

Ercolani (1973) proposed that host responses to pathogenic strains in susceptible (homologous) reactions involved a mechanism of independent action, in which a single bacterial cell could be responsible for symptom induction, while strains in hypersensitive (heterologous) reactions with their host involved a mechanism of cooperative action by several cells in order to induce symptoms. The dose response in homologous reactions is linear, while that in heterologous reactions is non-linear. The observation here that low bacterial numbers (>3 cfu) were associated with symptom induction at each inoculation site, and the linear dose response curve in cultivars independent of their reaction suggests that hypersensitive reactions regulated by *avr* genes (Gabriel, 1999) do not play a significant role in differentiating olive cultivar tolerance to *P. savastanoi*.

Pathogenicity tests show that strains from olive are not virulent to oleander while oleander strains are virulent to oleander and at least some cultivars of olive (Surico *et al.*, 1985; Janse, 1992; Caponero *et al.*, 1995). By contrast, field isolations indicate that only olive strains are isolated from olive, and only oleander strains are isolated from oleander (Caponero *et al.*, 1995, G. Surico, personal communication), suggesting that each strain is specific to its respective host in the field. The data here (Tables 1, 2, 3 and 4) and our unpublished data showed the oleander strains as being less virulent to olive than the olive strains. A consideration of the plot (Fig. 1) indicates that the oleander strains produce little reaction in olive. They form galls consistently smaller than do the olive strains and, apart from their reactions in cultivars Barnea, Leccino and Manzanillo, show little tendency to induce larger galls with increasing inoculum. However, Caponero *et al.* (1995) and Surico *et al.* (1985) report that, at high inoculum doses, some strains isolated from oleander were equally or more virulent to olive than strains isolated from olive. These different reactions reported from pathogenicity tests and reports of field specificity of olive and oleander strains need to be reconciled.

Sisto and Iacobellis (1999) noted the importance of identifying varieties of olive tolerant to olive knot in order to improve both the quantity and quality of fruit and oil yields. Qualitative studies of knot tolerance by olive cultivars to bacterial knot have been reported (Varvaro and Surico, 1978b; Osman *et al.*, 1980; Benjama *et al.*, 1987; Marcelo *et al.*, 1999). No olive cultivars are known to be completely resistant to knot disease (Iacobellis, 2001).

In this study, recording reactions after 2 months follows earlier studies (Janse, 1982; Sisto *et al.*, 2001). The experimental data give an indication of the relative tolerance of olive cultivars to the olive knot pathogen and is worth taking account of in choosing cultivars if tolerance to bacterial knot is shown to be important in some parts of the country. It cannot yet be determined how climatic conditions prevailing in different regions in New Zealand will affect the relative resistance of olive cultivars to olive knot disease, compared with the same cultivars growing in a Mediterranean environment. As information on the responses of olives to knot is gathered by observation in future years, it may be that susceptibility to the disease will become an important determinant in cultivar selection.

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Literature cited

Benjama A.H., L. Walali, L. Jananti and A. Moukhli, 1987. Field reaction of different varieties of olive (*Olea europaea* L.) to olive knot disease caused by *Pseudomonas syringae* pv. *savastanoi* (E.F. Smith) Stevens. In: *Proceedings, 3rd International Working Group on Pseudomonas Syringae Pathovars*, September 1–4, 1987, Lisbon, Portugal, 41–52.

Bradbury J.F., 1986. *Guide to Plant Pathogenic Bacteria*. CAB International Mycological Institute, Kew, UK, 332 pp.

Braithwaite M., B.J.R. Alexander, J.M. Young and S. Ganev, 1999. Olive knot disease in New Zealand. In: *Abstracts, 12th Australasian Plant Pathology Society Conference*, September 27–30, 1999, Canberra, Australia, 157 (abstract).

Caponero A., A.M. Contesini and N.S. Iacobellis, 1995. Population diversity of *Pseudomonas syringae* subsp. *savastanoi* on olive and oleander. *Plant Pathology* 44, 848–855.

Crosse J.E. and C.M.E. Garrett, 1966. Bacterial canker of stone-fruits. VII. Infection experiments with *Pseudomonas mors-prunorum* and *P. syringae*. *Annals of Applied Biology* 58, 31–41.

Dye D.W., 1956. Oleander knot (*Pseudomonas savastanoi* (Erw. Smith) Stevens). *New Zealand Journal of Science and Technology* 38A, 406–411.

Ercolani G.L., 1973. Two hypotheses on the aetiology of response of plants to phytopathogenic bacteria. *Journal of General Microbiology* 75, 83–95.

Gabriel D.W., 1999. Why do pathogens carry avirulence genes? *Physiological and Molecular Plant Pathology* 55, 205–214.

Genstat Committee, 2002. *GenStat 6.1. 2002*. VSN International, Oxford, UK.

Hu F.-P., J.M. Young, C.M. Triggs and J.P. Wilkie, 1997. Pathogenic relationships of the subspecies of *Acidovorax avenae*. *Australasian Plant Pathology* 26, 227–238.

Iacobellis N.S., 2001. Olive knot. In: *Encyclopaedia of Plant Pathology* (O.C. Malloy, T.D. Murray, ed.) Vol. 2, John Wiley, London, UK, 713–715.

Janse J.D., 1982. *Pseudomonas syringae* subsp. *savastanoi* (ex Smith) subsp. nov., nom. rev., the bacterium causing excrescences on *Oleaceae* and *Nerium oleander* L. *International Journal of Systematic Bacteriology* 32, 166–169.

Marcelo A., M. Fernandes, M. Fatima Potes and J.F. Serrano, 1999. Reactions of some cultivars of *Olea europaea* L. to experimental inoculations with *Pseudomonas syringae* pv. s*avastanoi*. *Acta Horticulturae* 474, 581–584.

Osman W.A., A.M. Tarabeih and S.H. Michail, 1980. Studies on olive knot disease in Iraq with reference to response of different cultivars. *Mesopotamia Journal of Agriculture* 15, 245–261 (in Arabic, English abstract).

Sisto A. and N.S. Iacobellis, 1999. Olive knot disease: pathogenic and epidemiological aspects and defence strategies. *Olivo e Olio* 2, 32–38 (in Italian, English abstract).

Sisto A., P.L. Cantore and N.S. Iacobellis, 2001. Preliminary results on the response of olive cultivars to artificial inoculation with *Pseudomonas syringae* subsp. *savastanoi*. *Proceedings, 11th Congress of the Mediterranean Phytopathological Union and 3rd Congress of the Sociedade Portuguesa de Fitopatologia,* 17–20 September, 2001, Evora, Portugal, 240–242.

Splus, 2002. *Splus 6.1 for Windows, 2002*. Insightful Corporation, Seattle, Washington, USA.

Surico G., N.S. Iacobellis and A. Sisto, 1985. Studies on the role of indole-3-acetic acid and cytokinins in the formation of knots on olive and oleander plants by *Pseudomonas syringae* pv. *savastanoi*. *Physiological Plant Pathology* 26, 309–320.

Varvaro L. and G. Surico, 1978a. Behaviour of different olive (*Olea europea* L.) cultivars upon artificial inoculation with *Pseudomonas savastanoi* (E.F. Smith) Stevens. *Phytopathologia Mediterranea* 17, 174–177 (in Italian, English abstract).

Varvaro L. and G. Surico, 1978b. Multiplication of *Pseudomonas savastanoi* (E.F. Smith) Stevens in olive (*Olea europaea* L.) tissues. *Phytopathologia Mediterranea* 17, 179–186 (in Italian, English abstract).

Young J.M., 1987. New plant disease record in New Zealand: *Pseudomonas syringae* pv. *persicae* from nectarine, peach, and Japanese plum. *New Zealand Journal of Agricultural Research* 30, 235–247.

Young J.M. 1991. Pathogenicity and identification of the lilac pathogen, *Pseudomonas syringae* pv. *syringae* van Hall 1902. *Annals of Applied Biology* 118, 283–298.

Young J.M., 2004. Olive knot disease and its pathogens. *Australasian Plant Pathology* 33, 34–39.

Young J.M., G. Saddler, Y. Takikawa, S.H. De Boer, L. Vauterin, L. Gardan, R.I. Gvozdyak and D.E. Stead, 1996. Names of plant pathogenic bacteria 1864–1995. *Review of Plant Pathology* 75, 721–763.

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