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# Resistance in Rhizoctonia solani to tolclofos-methyl

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**Abstract**

Isolates of *Rhizoctonia solani* were adapted in vitro to grow on a medium amended with tolclofos-methyl at a concentration 500 times that which initially almost completely inhibited growth.
Acquired resistance was retained after five transfers on a fungicide-free medium. Pathogenicity of resistant isolates was not reduced, but their growth rates on PDA were significantly lower than those of the original isolates. Recovery of the resistant isolates was not improved on a selective medium amended with tolclofos-methyl.

*Additional keywords:* fungicide resistance, Rizolex, stable resistance, fitness, pathogenicity.

## Introduction

The use of isolates with acquired resistance to organic fungicides has contributed greatly to our understanding of the ecology and population dynamics of soil-borne plant pathogens or biocontrol agents (Kloepper et al., 1980; Papavizas et al., 1982; Stack and Millar, 1982). Media amended with a given fungicide can be used to facilitate recovery of the resistant isolates from soil and enable distinguishing between fungicide-resistant propagules added to soil and those naturally present.

*Rhizoctonia solani* (Kuehn) has acquired resistance both to protectant organic fungicides, such as quintozene (PCNB) (Shatla and Sinclair, 1963; Meyer and Parmeter, 1968; Kataria and Grover, 1974), captan, dichlone, maneb and thiram (Elsaid and Sinclair, 1964; Abdalla, 1975) and chloroneb (Kataria and Grover, 1974) and to systemic fungicides such as the oxathiins oxycarboxin and carboxin (Grover and Chopra, 1970), benomyl and thiophanate-methyl (Kataria and Grover, 1974; Vanachter and Van Wambeke, 1977) and 2-(thiocyanomethylthio)-benzothiazole (TCMTB) and dichlozoline (Vanachter and Van Wambeke, 1977). In most cases the resistance apparently was temporary and possibly due to enzymatic adaptation (Elsaid and Sinclair, 1964; Grover and Chopra, 1970; Kataria and Grover, 1974). For quintozene and TCMTB, however, the resistance was stable (Shatla and Sinclair, 1963; Vanachter and Van Wambeke, 1977) and arose as a consequence of genetic changes, either due to heterokaryosis (Meyer and Parmeter, 1968) or to 'sectoral mutation' (Vanachter and Van Wambeke, 1977).

Tolclofos-methyl (Rizolex) is organic in nature, has curative and slightly systemic action, and is highly fungitoxic towards *R. solani* (Ohtsuki and Fujinami, 1982). Although its mode of action is not yet fully understood (Ohtsuki and Fujinami, 1982),

it apparently is metabolically specific and therefore likely to select resistant isolates (Dekker, 1976). We report here on our efforts: i) to obtain isolates of *R. solani* resistant to tolclofos-methyl for use in ecological studies, ii) on the stability of the acquired resistance; iii) on the pathogenicity of resistant and original isolates, and iv) on the ease of recovery of resistant and sensitive isolates on media with and without tolclofos-methyl.

## Materials and methods

*Fungal isolates.* Nine isolates of *R. solani* (isolated by J.J. Galindo and kindly supplied by G.S. Abawi of NYS Agric. Exp. Station, Geneva) were used. All isolates originated in New York State; isolates R12, 19, 28, 31, 39 and 40 were from bean soil, R76 and 81 from bean roots, and R2 from beet roots. According to Galindo (1979), anastomos groupings were as follows: AG1: R39; AG2: R40; AG4: R12, 19, 28, and 31; no anastomosis: R2; and not tested: R76 and 81.

*Fungicide.* Tolclofos-methyl (Rizolex) is an organophosphate, developed by Sumitomo Chemical Co., Ltd., Japan. A 50% wettable powder of this fungicide was kindly provided by AAgrunol BV, Groningen, the Netherlands.

*Growth rate on agar plates.* A preliminary test indicated that tolclofos-methyl added to the medium before or after autoclaving was equally effective in reducing mycelial growth of nine isolates of *R. solani* (paired T-test, $t = 1.70$). Therefore, the fungicide was added before autoclaving for subsequent tests. Four disks (4-mm diam.) were transferred with a sterile cork borer from PDA plates with *R. solani* to PDA amended with tolclofos-methyl.

All fungal isolates were exposed initially to 50 $\mu$g ml$^{-1}$ (active ingredient), and those isolates able to grow during 1 week of incubation at 27 °C were transferred to increasingly higher concentrations (100, 200, 400, 800, and 1600 $\mu$g ml$^{-1}$).

Simultaneously, disks from cultures of the original isolates maintained on PDA were transferred to unamended PDA and to PDA plus 50 $\mu$g tolclofos-methyl ml$^{-1}$ to serve as checks. The diameters of the colonies were determined after incubation at 25 ° C for 30-60 h (depending on the growth rate of the cultures). Growth rates were calculated by dividing the radius by the incubation period.

Growth rates of the different isolates were compared with the mean growth rate of the corresponding original culture on unamended PDA (control), and a reduction in growth rate was expressed as percent of the control. Plotting of the percent reduction in growth rate versus the concentration of tolclofos-methyl resulted in sigmoid curves, which were straightened using probit and natural log transformations. Zero and 100% growth reduction were equated to 2.67 and 7.33 probits, corresponding to 1% and 99%, respectively.

*Stability of resistant isolates.* Three resistant isolates (R19, 40 and 76), that had grown on PDA plus 400 $\mu$g tolclofos-methyl ml$^{-1}$ were transferred five successive times to unamended PDA and then returned to PDA amended with 50, 100, 200 and 400 $\mu$g ml$^{-1}$ of the fungicide.

*Relative pathogenicity of original and resistant isolates.* Three isolates resistant to 400 μg tolclofos-methyl $ml^{-1}$ along with the original isolates from which they were obtained were tested for pathogenicity on dry bean cv. Redkloud. Optimal conditions for symptom expression had been determined previously for isolate R2. All isolates were transferred to 20 g autoclaved green beans in glass petri dishes and incubated at 25 °C for 11 days. The cultures were air-dried at 30 °C and then ground in a mortar to separate individual sclerotia. Twenty-five mg sclerotia of each strain were mixed in separate batches of 750 g natural field soil (Darien gravelly silt loam, pH = 7.1, sieved through a 7-mm mesh). The inoculum density was equivalent to about 500 sclerotia/liter soil. The infested soil was added to three Pyrex storage dishes. Three dishes with uninfested soil served as controls. Five untreated bean seeds were sown per dish, at a depth of 1 cm. Ten ml water was added to each dish containing 250 g soil, resulting in a soil moisture level of about 16%, equivalent to −170 kPa suction pressure. After incubation at 25 °C for 6 days, the bean seedlings were checked for hypocotyl and root lesions.

*Recovery from hypocotyls and soil.* Hypocotyls with lesions induced by three isolates resistant to 400 μg tolclofos-methyl $ml^{-1}$ (R19, 40 and 76) and by four sensitive isolates (R2, 19, 40 and 76) were immersed in 10% Clorox (5.25% NaOCl) for 1 min, rinsed with sterile distilled water and plated on Ko and Hora's medium (Ko and Hora, 1971), with or without 400 μg tolclofos-methyl $ml^{-1}$.

Samples of 60 ml of natural field soil to which sclerotia of sensitive and resistant isolates of *R. solani* had been added (see pathogenicity test) were wet-sieved using a 300-μm mesh sieve (Weinhold, 1977). The organic matter was plated out in 12-15 small heaps on Ko and Hora's medium with or without 400 μg tolclofos-methyl $ml^{-1}$. The plates were checked for growth of *R. solani* after 24 and 48 h of incubation at 25 °C. The numbers of colonies with hyphae typical for *R. solani* were recorded.

Table 1. Growth rates (mm/day) of *R. solani* on PDA and on PDA + 50 μg tolclofos-methyl $ml^{-1}$.

| Isolate | PDA (control) (mm/day) | PDA + tolclofos-methyl | |
|---|---|---|---|
| | | mm/day | % of control |
| R2 | 10.4 (1.6)[a] | 0.05 (0.14)[a] | 0.5 d[b] |
| R12 | 10.6 (2.2) | 0.20 (0.21) | 1.9 cd |
| R19 | 15.5 (1.0) | 1.00 (0.21) | 6.5 b |
| R28 | 13.7 (1.5) | 0.25 (0.21) | 1.8 cd |
| R31 | 14.8 (0.4) | 0.55 (0.30) | 3.7 bcd |
| R39 | 11.9 (1.2) | 0.25 (0.21) | 2.1 cd |
| R40 | 12.9 (0.5) | 0.40 (0.00) | 3.1 bcd |
| R76 | 14.3 (1.5) | 0.65 (0.21) | 4.5 bc |
| R81 | 11.4 (0.8) | 1.20 (1.03) | 10.5 a |

[a] Means of 8 replications and their standard deviations.

[b] Percent of growth on unamended PDA. Numbers followed by different letters differ significantly from each other according to Duncan's multiple range test ($\alpha$ = 0.05).

## Results

*Growth rate on agar.* The effect of 50 $\mu$g tolclofos-methyl ml$^{-1}$ on linear growth rates of nine isolates of *R. solani* during their first exposure to the fungicide is given in Table 1. Tolclofos-mesthyl was highly effective in reducing the growth rates of all isolates, R2 being most sensitive and R81 least.

Transfer of colonies which were able to grow at 50 $\mu$g tolclofos-methyl ml$^{-1}$ to PDA with higher concentrations of the fungicide resulted in increased levels of resistance (Fig. 1).

Dose responses of representative isolates before exposure and after successive transfers on tolclofos-methyl are shown in Fig. 2 in the form of probit-lines. Several

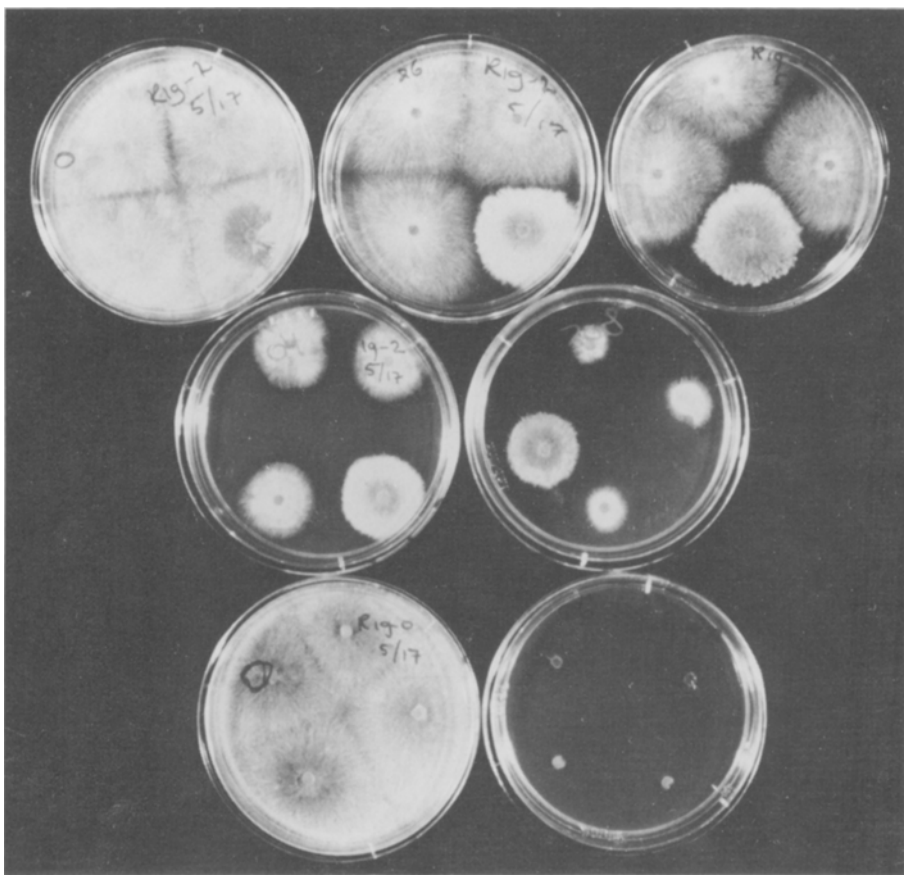

Fig. 1. Colonies of *R. solani,* isolate R19, after 4 days of growth on PDA with different concentrations of tolclofos-methyl (tm); upper row (from left to right): resistant isolate (after two previous exposures to 50 $\mu$g tm ml$^{-1}$) at 0, 50 and 100 $\mu$g tm ml$^{-1}$; middle row: the same isolate at 200 and 400 $\mu$g ml$^{-1}$; lower row: the original, sensitive isolate at 0 en 50 $\mu$g tm ml$^{-1}$. (Note sector in upper left petri dish, and variability in resistant isolate).

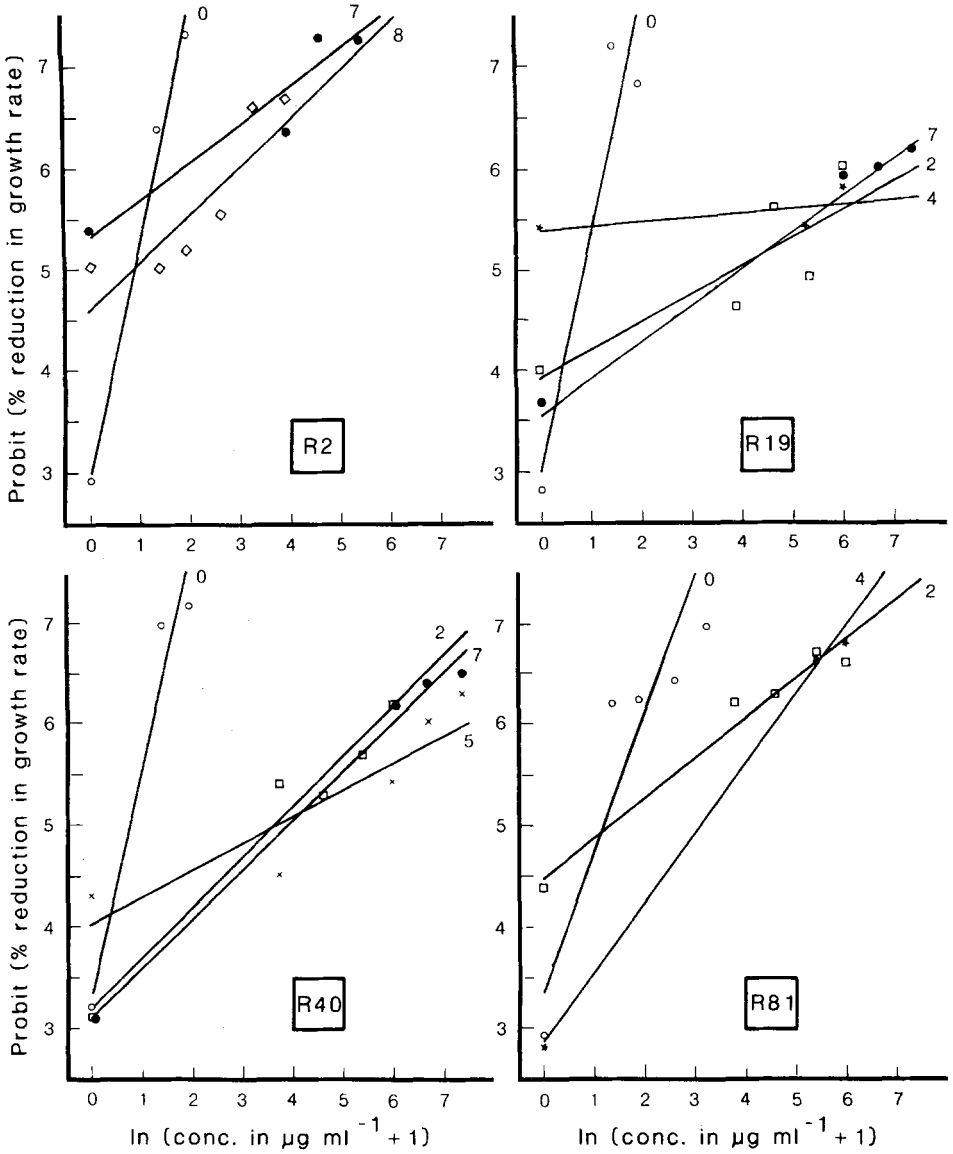


Fig. 2 Percent reduction in growth rate of *R. solani* isolates, expressed as probits, plotted against the natural log of the concentration of tolclofos-methyl in PDA; the predicted values of linear regression of the probit on ln (conc. in $\mu g\ ml^{-1} + 1$) are represented by lines, and the mean observed values after 0, 2, 4, 5, 7 or 8 prior exposures to tm are indicated by circles, squares, starts, crosses, dots or diamonds, respectively.

transfers of R2 on tolcolofos-methyl-amended PDA were needed before a moderate level of resistance was obtained. Comparison of 95% confidence intervals for slopes and intercepts revealed that the slopes of the lines for cultures exposed to tolclofos-methyl were significantly less than the slope of the original culture. The intercepts were

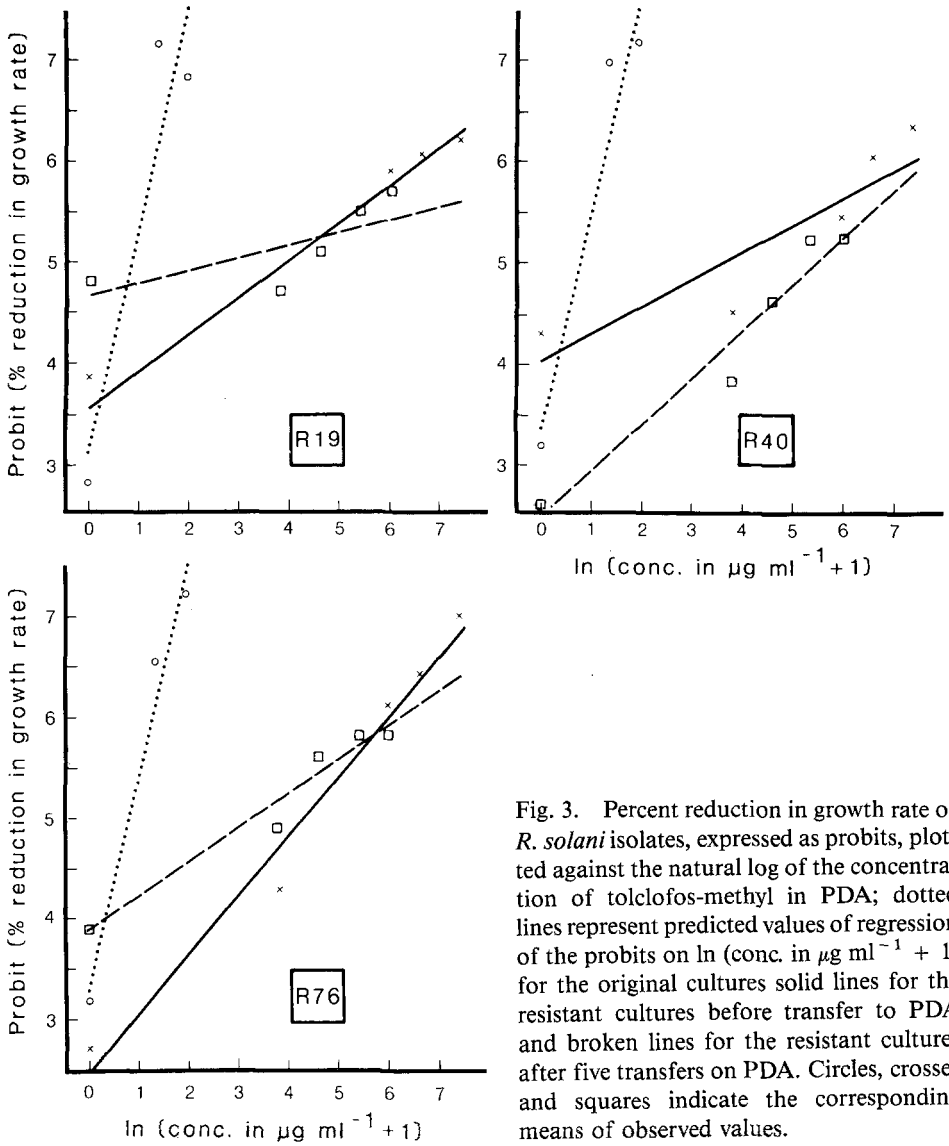


Fig. 3. Percent reduction in growth rate of *R. solani* isolates, expressed as probits, plotted against the natural log of the concentration of tolclofos-methyl in PDA; dotted lines represent predicted values of regression of the probits on ln (conc. in $\mu g\ ml^{-1} + 1$) for the original cultures solid lines for the resistant cultures before transfer to PDA and broken lines for the resistant cultures after five transfers on PDA. Circles, crosses and squares indicate the corresponding means of observed values.

significantly increased after exposure to the fungicide. Isolates R19 and R40 showed a similar pattern in their reaction to higher levels of tolclofos-methyl, but the growth on unamended PDA was more reduced for R19 than for R40. The slopes and intercepts of all transfers not given in Fig. 2 were similar to those of the second and seventh transfer. Only those of the fourth or fifth transfer differed significantly from those of the second an seventh. Isolate R81 was slightly resistant before exposure to tolclofos-methyl (having a less steep slope than the other isolates before exposure), but gained only little in resistance after being exposed to the fungicide.

Four kinds of reactions to exposure to the fungicide were distinguished: i) only a small or no increase in resistance after the first exposure to 50 $\mu$g tolclofos-methyl $ml^{-1}$ (isolates R2, R31, and R39); ii) a considerable increase in resistance to tolclofos-methyl after the first exposure to it, and a decreased growth on unamended PDA (R12, R19, R28 and R76), iii), an increase in resistance after the first exposure, but less growth reduction on PDA than that of type 2 (R40); iv) lower sensitivity without pre-exposure to tolclofos-methyl and some increase in resistance after exposure (R81).

In general, the probits of the observed values on PDA plus tolclofos-methyl after any number of exposures seemed to fit one common line; differences in slopes and intercepts were significant, but were mainly due to the erratic behavior on unamended PDa after prior exposure to the fungicide.

The resistances obtained in isolate R19, R40 and R76 were checked for stability after five transfers on unamended PDA. Dosage response curves for the original cultures, the resistant cultures before being transferred to unamended PDA, and the resistant cultures after five transfers on PDA are given in Fig. 3. Although the intercepts and/or slopes of the resistant cultures after transfers on PDA differed significantly from those before transfers on PDA, the tolerant strains had not reverted to the sensitive type during those transfers.

*Pathogenicity test.* The percentages of plants with hypocotyl and root lesions are given in Table 2. There were significant differences between isolates in their capacity

Table 2. Percentages of plants with hypocotyl and root lesions after inoculation with *R. solani* isolates senstive or resistant to 400 $\mu$g tolclofos-methyl $ml^{-1}$

| Isolate | Resistance to tolclofos-methyl | Hypocotyl | | Roots | |
|---|---|---|---|---|---|
| | | mean[a] | s.d.[b] | mean[a] | s.d.[b] |
| R2 | – | 66.7 | 30.6 | 100.0 | 0.0 |
| R19 | – | 6.7 | 11.5 | 0.0 | 0.0 |
| R19 | + | 17.7 | 16.6 | 0.0 | 0.0 |
| R40 | – | 66.7 | 30.6 | 0.0 | 0.0 |
| R40 | + | 66.7 | 38.2 | 6.7 | 11.5 |
| R76 | – | 56.7 | 40.4 | 8.3 | 14.4 |
| R76 | + | 86.7 | 11.5 | 11.0 | 19.1 |
| none | | 0.0 | 0.0 | 0.0 | 0.0 |

| Contrast comparions | t | sign.[c] |
|---|---|---|
| R19 vs. all other isolates on hypocotyls | 5.0 | ** |
| R2 vs. all other isolates on roots | 15.4 | ** |
| Resistant vs. sensitive isolates on hypocotyls | 1.0 | n.s. |
| Resistant vs. senstive isolates on roots | 0.7 | n.s. |

[a] Means of three replications.
[b] s.d. = standard deviation.
[c] ** Significant at 1% level; n.s. not significant.

to infect hypocotyls ($F^6_{14} = 3.25$); isolate R19 infected significantly fewer hypocotyls than did the other isolates. The differences between isolates in there ability to induce root lesions were highly significant ($F^6_{14} = 39.6$); this result was due to R2 being considerably more pathogenic on roots than all other strains. Resistance to tolclofos-methyl did not affect pathogenicity of any of the isolates, neither on hypocotyls nor on roots.

*Isolation from hypocotyls and soil.* An attempt was made to reisolate *R. solani* from infected hypocotyls and to check the isolates for their sensitivity to tolclofos-methyl. Isolations were made on Ko and Hora's medium from 10 lesions per treatment, except in the case of R19 which induced only two lesions. When the medium was amended with tolclofos-methyl, resistant isolates were not recovered more frequently than their sensitive counterparts ($\chi^2 = 3.0$) (Table 3). Recovery of senstitive isolates was more frequent than that of resistant isolates on unamended medium ($\chi^2 = 10.0$), but recovery of sensitive isolates was significantly reduced on amended medium ($\chi^2 = 15.9$).

Table 3. Numbers of isolations from hypocotyl lesions which did or did not yield *R. solani* (R.s.) on Ko and Hora's medium without or with 400 $\mu$g tolclofos-methyl (tm) $ml^{-1}$.

| Isolate | Resistance to tm | − tm | | + tm | |
|---|---|---|---|---|---|
| | | R.s. | no R.s. | R.s. | no R.s. |
| R2 | − | 6 | 4 | 0 | 10 |
| R19 | − | 0 | 2 | 0 | 2 |
| R19 | + | 0 | 2 | 1 | 1 |
| R40 | − | 5 | 5 | 1 | 9 |
| R40 | + | 2 | 8 | 1 | 9 |
| R76 | − | 7 | 3 | 2 | 8 |
| R76 | + | 1 | 9 | 0 | 10 |

| Comparisons | $\chi^2$ | Sign.[a] |
|---|---|---|
| Interaction between resistance and tm amendment | 14.9 | ** |
| Effect of resistance using amended medium | 3.0 | n.s. |
| Effect of resistance using unamended medium | 10.0 | ** |
| Effect of amendment on isolation of resistant isolates | 1.3 | n.s. |
| Effect of amendment on isolation of senstive isolates | 15.9 | ** |

[a] ** significant at 1% level; n.s. not significant.

Isolation of *R. solani* from soil infested with three resistant and three sensitive isolates was attempted three times, using Ko and Hora's medium with or without tolclofos-methyl. The three isolation attempts were considered as blocks, and the data were analysed with a two-way analysis of variance. Both the block and treatment (six strains on amended and unamended medium) effects were significant with F values of 4.6 and 2.9, respectively. The mean numbers of colonies isolated and contrast comparisons are presented in Table 4.

Table 4. Numbers of *R. Solani* colonies growing from organic matter (from 60 ml soil) onto Ko and Hora's medium without or with 400 $\mu$g tolclofos-methyl (tm) $ml^{-1}$.

| Strain | Tolerance | − tm | | + tm | |
|---|---|---|---|---|---|
| | | mean | s.d. | mean | s.d. |
| R19 | − | 1.0 | 1.0 | 0.0 | 0.0 |
| R19 | + | 4.3 | 2.1 | 1.0 | 1.0 |
| R40 | − | 1.7 | 2.1 | 0.0 | 0.0 |
| R40 | + | 2.0 | 2.7 | 2.0 | 2.7 |
| R76 | − | 1.0 | 1.0 | 0.0 | 0.0 |
| R76 | + | 0.7 | 0.6 | 0.0 | 0.0 |

| Contrast comparisons | t | sign.[a] |
|---|---|---|
| interaction resistance x tm amendment | 0.1 | n.s. |
| the effect of resistance using unamended medium | 1.81 | * |
| the effect of resistance using amended medium | 1.65 | n.s. |
| the effect of amendment on recovery of resistant isolates | 2.20 | * |
| the effect of amendment on recovery of sensitive isolates | 2.03 | * |

[a] * Significant at $\alpha = 0.05$ (one-sided test); n.s. not significant.

The resistant isolates were recovered significantly more frequently than the sensitive isolates on unamended medium. On amended medium the difference between sensitive and resistant strains was just not significant, despite the fact that R40 and R19 were isolated on amended medium. Both sensitive and resistant isolates were recovered significantly more often on unamended than on amended medium. Tolclofos-methyl prevented recovery of sensitive isolates altogether.

## Discussion

*R. solani* is a multinucleate organism, and field isolates are generally assumed to be heterokaryotic (Meyer and Parmeter, 1968). There is an enormous potential for variation in *R. solani*, as evidenced by the large numbers of isolates differing in cultural, ecological and pathogenic characteristics, and this may be the reason why resistance has been reported to so many protectant and systemic fungicides. However, most of these forms of resistance proved to be unstable in the absence to the fungicide. In those cases the resistance could be due to non-genetic adaptive changes (Dekker, 1973).

In one instance conclusive evidence was given of resistance to a fungicide as a result of heterokaryosis, namely to quintozene (Meyer and Parmeter, 1968). Apparently, this form of resistance could develop under field conditions, since Shatla and Sinclair (1963) demonstrated increased resistance levels in populations of *R. solani* isolated from cotton fields treated with quintozene.

In this paper we demonstrate development of resistance to tolclofos-methyl in most of the isolates tested. The resistance initially increased after subsequent transfers, and was variable there after, but remained substantially higher than the original isolates

which had not been exposed to the fungicide. Differences in resistance of subsequent transfers were mainly due to variable growth rates on unamended PDA. Three isolates with high levels of resistance maintained this trait after five transfers on fungicide-free medium. This does not mean that this resistance would be stable forever or that it would hold up under field conditions. In most isolates increased resistance was accompanied by a reduction in growth rate and by morphological changes on PDA (increased sclerotial formation at low concentrations of tolclofos-methyl and lack of sclerotia at high concentrations). These are common phenomena in isolates selected for resistance (Elsaid and Sinclair, 1964; Grover and Chopra, 1970; Dekker, 1977) and may result in decreased ecological fitness. A reduction in fitness of tolcolofos-methyl resistant of *R. solani* may not be surprising, because isolates of fungi with resistance to other organophosphates generally appeared to be less fit than their sensitive counterparts (Dekker, 1982).

In a pathogenicity test the virulence of resistant isolates was not affected. However, reisolation of *R. solani* from hypocotyls on a selective medium plus tolclofos-methyl did not demonstrate unequivocably that the isolates recovered were still resistant or had reverted and regained some resistance, since some lesions of sensitive isolates also resulted in isolation of *R. solani* on amended medium. Of the resistant isolates which had been incorporated into soil only R19 and R40 were reisolated on a selective medium plus tolclofos-methyl. The resistant isolate of R40 was recovered equally well on amended as on unamended medium, but those of R19 and R76 were recovered less frequently on amended medium. This might be due to a slower growth rate at the concentration used.

For field use of tolclofos-methyl relatively high dosages are recommended, e.g. 1000 $\mu$g a.i. $ml^{-1}$ as a potato tuber spray (Ohtsuki and Fujinami, 1982). However, in vitro these dosages could be relatively easily overcome by a number of strains. This may imply that nuclei with genes for resistance are widespread, at least in the isolates used in these experiments. Despite the variability of *R. solani* and the ease with which resistance could be selected, this resistance may not be of practical importance because of a reduced fitness.

The original objective of this study was to obtain resistant mutants to be used in ecological studies. Those mutants have to meet several criteria to be of use: i) stability, ii) equal fitness and virulence as the wild-type and iii) improved isolation and recognition on media amended with the fungicide. The resistant isolate of R40 met these criteria most closely, but its growth rate was reduced on unamended medium. Since none of the resistant isolates meet all criteria, these isolates will not be used in further studies.

**Acknowledgment**

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## Samenvatting

*Resistentie tegen tolchlofos-methyl in Rhizoctonia solani*

Na overenting op een medium dat tolchlofos-methyl bevatte, raakten enkele isolaten van *Rhizoctonia solani* gewend aan 500 maal de dosis die oorspronkelijk bijna alle groei verhinderde.

Resistente isolaten van *R. solani* bleven minder gevoelig voor tolchlofos-methyl na vijf overentingen op een medium zonder het fungicide. De pathogeniteit van resistente isolaten was niet verminderd, maar hun groeisnelheid op PDA was significant vertraagd vergeleken bij die van de oorspronkelijke isolaten. Isolatie van de resistente stammen werd niet verbeterd op een selectief medium waaraan tolchlofos-methyl was toegevoegd.

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# Book review

S. Nagarajan, 1983. Plant disease epidemiology. Published by Mohan Primlani for Oxford & IBH Publishing Co., New Delhi. XV+267 pp. Rs. 80.00.

The author won early recognition when he participated as a junior scientist at an epidemiology top conference in Wageningen, 1971. He has kept his promise and presently he is one of the outstanding epidemiologists from the developing countries. As he chose to spend his career in India, except for a post-doc position in the Federal Republic of Germany, he has become an authority on the application of epidemiology in developing countries. His long fostered desire to publish a book on plant disease epidemiology for an Indian public finally materialized.

The book is somewhat unconventional in design. After an introduction (Ch. 1) it discusses the pathogen with emphasis on population behaviour (Ch. 2) and variability (Ch. 3). An exposition of pathometry is delayed to Ch. 7. The host is discussed in Ch. 4, but the concomitant non-chemical control is postponed to Ch. 10. Weather effects are dealt with in Ch. 5. Mathematical aspects of epidemiology, in Vanderplank's way, are treated in Ch. 8. The more speculative chapters are Ch. 6 on space, time and chance, and a lengthy Ch. 9 on systems approach. Exercises, a subject index, and a – quite incomplete – list of errata are given.

The author writes most convincingly where he discusses aerobiology, the area in which he has attained international excellence. The book ends with an epilogue containing some peppered remarks on the Indian way of teaching phytopathology, which is said to overemphasize taxonomy and mycology at the neglect of disease control by cultural methods, resistance breeding, and so on. The author offers a well-considered curriculum proposal. Hopefully, his book will help to change the criticized emphasis of phytopathology teaching in India. The western reader will be interested in the author's selection of topics, from simple logarithms to sophisticated remote sensing.

The book contains some 220 references, 24 per cent of which refer to India or Indian authors. This is not a low figure, considering that the body of epidemiological theory is of western origin. Though more examples might have been taken from Indian sources, the book gives the western reader a fair impression of the state of the art in India. The prospects for usefull applications of epidemiological insight, as – for example – gene deployment, seem to be very good indeed.

J.C. Zadoks