Inheritance of Increased Sensitivity to *N*-Phenylcarbamates in Benzimidazole-Resistant *Venturia nashicola*

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Contribution: Fruit Tree Res. Stn., A-220.
We are grateful to Professor Dr. J. Dekker, Department of Phytopathology, Agricultural University, Wageningen, The Netherlands, and to Professor Dr. S. G. Georgopoulos, Agricultural Department, Lapapharm Inc., Athens, Greece, for critical reading of this manuscript. We also wish to thank Dr. H. Yanase, Fruit Tree Research Station, and Miss T. Toyoshima for their assistance.
Accepted for publication 7 January 1988.

ABSTRACT

Ishii, H., and van Raak, M. 1988. Inheritance of increased sensitivity to *N*-phenylcarbamates in benzimidazole-resistant *Venturia nashicola*. Phytopathology 78:695-698.

Most of the highly resistant isolates of *Venturia nashicola* to the benzimidazole fungicide carbendazim showed increased sensitivity *in vitro* to the *N*-phenylcarbamate compounds MDPC and diethofencarb, but the intermediately and weakly carbendazim-resistant isolates did not. A segregation ratio of 1:1 was obtained from the cross between MDPC-sensitive and -resistant isolates. In an allelism test, no MDPC-resistant progeny appeared from the cross between two MDPC-sensitive isolates, and no MDPC-sensitive progeny resulted from the cross between two MDPC-resistant isolates. These results indicated that the increased sensitivity to MDPC is controlled by a single chromosomal gene. When a highly carbendazim-resistant isolate (MDPC-sensitive) was crossed with an intermediately carbendazim-resistant or a carbendazim-sensitive isolate (both resistant to MDPC), several progeny strains were doubly resistant, i.e., highly resistant to carbendazim and resistant to MDPC. Such strains also were detected among field isolates, and the double resistance was heritable.

Additional key words: benomyl, negatively correlated cross-resistance, pear scab, thiophanate-methyl.

During the last decade, benzimidazole resistance of phytopathogenic fungi has increased throughout the world, making it difficult to control many plant diseases with benzimidazole fungicides. In Japan, pear scab, caused by *Venturia nashicola* Tanaka et Yamamoto is one of the most serious diseases of Japanese pear (*Pyrus serotina* Rehder var. *culta* Rehder). Since 1975, benzimidazole-resistant strains of *V. nashicola* have been frequently isolated from pear orchards, where poor scab control with these fungicides has occurred. In orchards where the resistant strains predominate, use of benzimidazole fungicides was stopped or restricted, and these fungicides were replaced by conventional fungicides such as captafol, dithianon, and captan. At present, however, the benzimidazole-resistant strains remain widely distributed throughout Japan. Field experiments also suggest the persistence of benzimidazole resistance in *V. nashicola* after removal of the selection pressure by these fungicides (4).

To cope with fungicide resistance, the use of chemicals to which the resistant strains show a negatively correlated cross resistance may be a promising measure (2). Leroux and Gredt (12) reported that some isolates of *Botrytis cinerea* and *Penicillium expansum* resistant to benzimidazoles displayed increased sensitivity to the *N*-phenylcarbamate herbicides barban, chlorbufam, and chlorpropham. In addition, Kato et al (8,9) found that benzimidazole-resistant strains of *B. cinerea* and *V. nashicola* were more sensitive to the newly developed *N*-phenylcarbamate fungicides such as methyl *N*-(3,5-dichlorophenyl)carbamate (MDPC) and isopropyl *N*-(3,4-diethoxyphenyl)carbamate (diethofencarb, S-165) than were the benzimidazole-sensitive strains. In *V. nashicola*, however, the increased sensitivity to MDPC and diethofencarb was only observed in the highly benzimidazole-resistant isolates but not in the intermediately resistant and weakly resistant ones (5).

The authors (5) previously demonstrated inheritance of benzimidazole resistance in *V. nashicola* and suggested that the manifestation of three different levels of resistance could be attributed to three allelic mutations in a single gene. The purpose of the present paper is to demonstrate inheritance of the increased sensitivity of highly benzimidazole-resistant isolates to MDPC and to examine the mode of inheritance. The increased sensitivity is later discussed with respect to the genetic background of benzimidazole resistance. Brief results of this work have already been reported (6).

MATERIALS AND METHODS

Isolates. Pear leaves with sporulating *V. nashicola* were collected from orchards in Ibaraki, Chiba, Kanagawa, Shizuoka, Toyama, Tottori, Okayama, and Oita Prefectures, and conidia from the lesions were directly suspended in sterile distilled water. Drops of conidial suspensions were placed on water agar plates and incubated at 15 C for 3–5 days. Agar blocks containing germinated conidia were individually isolated with a steel needle after microscopic observation and transferred onto potato-dextrose agar (PDA) slants. After incubation at 20 C for about 2 mo, the cultures were stored at 10 C. Twenty-three isolates in total were tested. An isolate that acquired high resistance to the benzimidazole fungicide carbendazim after repeated subculture of an intermediately resistant isolate on carbendazim-amended culture medium described earlier (5) was also used.

Sensitivity to carbendazim and to *N*-phenylcarbamates. Each isolate was previously cultured on PDA plates at 20 C for 45 days. Mycelial disks, 4 mm in diameter, were cut from the margins of the colonies and transferred onto PDA plates containing carbendazim, MDPC, or diethofencarb. Stock solutions of these compounds were obtained by dissolving technical grades in acetone. Addition of the proper amount of stock solution to molten PDA after autoclaving gave a series of concentrations of each fungicide. The final acetone concentration in PDA was 2%. After incubation at 20 C for 3 wk, mycelial growth of the isolates on PDA plates was observed, and the minimum inhibitory concentrations (MIC) of each fungicide necessary for the complete inhibition of mycelial growth of the isolates were determined. Based on the difference in sensitivity to carbendazim, isolates were divided into four groups as follows: highly carbendazim resistant,

MIC > 100 μg/ml; intermediately carbendazim resistant, 100 μg/ml ≥ MIC > 10 μg/ml; weakly carbendazim resistant, 10 μg/ml ≥ MIC > 1 μg/ml; carbendazim sensitive, 1 μg/ml ≥ MIC. In addition, MDPC-sensitive and -resistant isolates were distinguished as follows: sensitive, growth at 1 μg/ml, but not at 10 μg/ml; resistant, growth at 10 μg/ml. Carbendazim was kindly supplied by Du Pont Japan Ltd., Tokyo. MDPC and diethofencarb were synthesized by the methods of Kato et al (10) and Noguchi et al (14).

Crosses. Fifteen crosses in total were analyzed. Parent isolates were separately precultured on Czapek agar plates at 20 C in the dark for 45 days, then the mycelial disks from cultures were aseptically homogenized in distilled water. In petri dishes, equal volumes of the mycelial suspensions of the two isolates to be crossed were mixed and poured on plates of molten malt extract agar (malt, 0.5%; agar, 2.5%) containing pear leaf decoction (dry material 25 g/L of the medium). The petri dishes were kept at 5 C in the dark for 5–6 mo until pseudothecia and ascospores matured. After microscopic observation, pseudothecia were removed from the agar medium and crushed in sterile distilled water using a stainless steel spatula. Drops of the ascospore suspensions were transferred on water agar plates and 100 ascospores from each cross were individually isolated at random by the same methods as in the monoconidial isolation mentioned above. Sensitivity of progeny isolates thus obtained to carbendazim and MDPC was evaluated as described above.

RESULTS

Sensitivity to carbendazim and to *N*-phenylcarbamates. Sensitivity of 23 isolates tested to carbendazim, MDPC, and diethofencarb *in vitro* is listed in Table 1. Out of 11 highly carbendazim-resistant isolates, seven isolates showed a large increase of sensitivity to MDPC and/or diethofencarb as compared with six carbendazim-sensitive isolates. Four other highly carbendazim-resistant isolates, viz., field isolates JS-11, JS-168, JS-232, and a laboratory mutant, JS-40M, did not show such an increase of sensitivity to the *N*-phenylcarbamates. Furthermore, increased MDPC sensitivity was not observed in three intermediately carbendazim-resistant isolates nor in three weakly resistant isolates.

Inheritance of increased sensitivity to *N*-phenylcarbamates. First, of two highly carbendazim-resistant isolates that showed increased sensitivity to MDPC, one was crossed with an intermediately carbendazim-resistant and one with a carbendazim-sensitive isolate (both resistant to MDPC). As is evident from Table 2, the inheritance of increased sensitivity to MDPC was confirmed in the crosses. Regarding the sensitivity to MDPC, progeny segregation coincided with a 1 sensitive:1 resistant ratio. In both crosses, most progeny isolates reacted to the fungicides used in the same manner as one of their parent isolates. However, progenies that showed resistance to MDPC with high carbendazim resistance also appeared in a relatively high frequency of 10%. However, progeny isolates sensitive to both carbendazim and MDPC were not obtained. Next, the allelism test was carried out in nine crosses using three MDPC-sensitive isolates and 10 MDPC-resistant isolates. The results are summarized in Table 3. In all crosses, progeny isolates showed a same phenotype for fungicide sensitivity as one of their parent isolates. No MDPC-resistant progeny appeared from the cross between two MDPC-sensitive isolates and no MDPC-sensitive progeny resulted from the cross between two MDPC-resistant isolates. Finally, inheritance of the

TABLE 1. Sensitivity of *Venturia nashicola* isolates to carbendazim, methyl *N*-(3,5-dichlorophenyl)carbamate (MDPC), and diethofcarb

Isolate	Reaction to carbendazim[a]	Minimum inhibitory concentration (μg/ml) of		
		Carbendazim	MDPC	Diethofencarb
JS-11	HR	>800	25	>800
JS-40M	HR*	>800	25	>800
JS-111	HR	>800	1.56	0.78
JS-114	HR	>800	1.56	0.78
JS-134	HR	>800	3.12	0.78
JS-135	HR	>800	1.56	0.78
JS-137	HR	>800	3.12	0.39
JS-140	HR	>800	1.56	0.78
JS-168	HR	>800	25	NT[b]
JS-206	HR	>800	1.56	NT
JS-232	HR	>800	25	NT
JS-40	IR	25	25	>800
JS-41	IR	25	25	>800
CS-22	IR	50	25	>800
JS-27	WR	1.56	25	>800
JS-77	WR	1.56	25	>800
JS-132	WR	1.56	25	>800
JS-2	S	<0.19	25	>800
JS-4	S	<0.19	25	>800
JS-18	S	<0.19	25	>800
JS-115	S	<0.19	25	>800
CS-1	S	<0.19	25	>800
CS-11	S	<0.19	25	>800

[a] Abbreviations: HR = highly resistant; HR* = highly resistant (laboratory mutant); IR = intermediately resistant; WR = weakly resistant; S = sensitive.
[b] NT = not tested.

TABLE 2. Inheritance of reaction to carbendazim and methyl *N*-(3,5-dichlorophenyl)carbamate (MDPC) in random ascospore progenies of *Venturia nashicola* crosses

Cross	Progenies (no.)				χ^2 for MDPC sensitivity (1:1)[e]
	HR,S[a]	HR,R[b]	IR,R[c]	S,R[d]	
HR,S × IR,R (JS-111) (CS-22)	47	10	43	0	0.36
HR,S × S,R (JS-135) (JS-115)	47	10	0	43	0.36

[a] HR,S: Highly resistant to carbendazim and sensitive to MDPC.
[b] HR,R: Highly resistant to carbendazim and resistant to MDPC.
[c] IR,R: Intermediately resistant to carbendazim and resistant to MDPC.
[d] S,R: Sensitive to carbendazim and resistant to MDPC.
[e] Expected value for a 1:1 ratio at $P = 0.05$ is 3.84.

TABLE 3. Inheritance of reaction to carbendazim and methyl *N*-(3,5-dichlorophenyl)carbamate (MDPC) (test for allelism) in random ascospore progenies of *Venturia nashicola* crosses

Cross	Progenies (no.)				χ^2 for MDPC sensitivity (1:1)[e]
	HR,S[a]	IR,R[b]	WR,R[c]	S,R[d]	
HR,S × HR,S (JS-111) (JS-135)	100	0	0	0	NS[f]
HR,S × HR,S (JS-111) (JS-140)	100	0	0	0	NS
IR,R × WR,R (JS-41) (JS-77)	0	55	44	0	NS
IR,R × S,R (JS-40) (JS-2)	0	43	0	57	NS
IR,R × S,R (JS-41) (JS-4)	0	50	0	50	NS
WR,R × WR,R (JS-132) (JS-27)	0	0	100	0	NS
WR,R × S,R (JS-27) (CS-1)	0	0	53	47	NS
S,R × S,R (JS-115) (CS-1)	0	0	0	100	NS
S,R × S,R (CS-1) (CS-11)	0	0	0	100	NS

[a] HR,S: Highly resistant to carbendazim and sensitive to MDPC.
[b] IR,R: Intermediately resistant to carbendazim and resistant to MDPC.
[c] WR,R: Weakly resistant to carbendazim and resistant to MDPC.
[d] S,R: Sensitive to carbendazim and resistant to MDPC.
[e] Expected value for a 1:1 ratio at $P = 0.05$ is 3.84.
[f] NS: no segregation.

double resistance, i.e., high carbendazim resistance and MDPC resistance, was checked. As shown in Table 4, the double resistance was inherited in all the crosses as follows: in three of the crosses highly carbendazim-resistant isolates JS-11, JS-168, and JS-232 isolated from the field were used, and in the other cross JS-40M, an isolate, which had changed from intermediate carbendazim-resistance to high resistance in the laboratory (5), was used. In a cross of JS-168 (highly carbendazim resistant and resistant to MDPC) with JS-206 (highly carbendazim resistant and sensitive to MDPC), nonparental type of progenies, viz., weakly carbendazim-resistant ones, appeared in a frequency of 50%.

DISCUSSION

Currently, benzimidazole-resistant strains of phytopathogenic fungi are distributed throughout the world. Strategies such as alternating or mixing unrelated fungicides are usually recommended to avoid the occurrence of a practical problem due to fungicide resistance. In many cases, however, monitoring data in the field demonstrated the stability of benzimidazole resistance (4). Therefore, the development and introduction of chemicals to which the benzimidazole-resistant strains show an increased sensitivity may become one of the measures to cope with such resistance.

Leroux and Gredt (11,12) first reported negative correlations for cross resistance between benzimidazoles and *N*-phenylcarbamates. Subsequently, Kato et al (8,9) developed several *N*-phenylcarbamate fungicides such as MDPC and diethofencarb and reported their higher activity against benzimidazole-resistant strains in comparison with that against the sensitive strains. Leroux et al (13) further reported that diethofencarb (S 32165 in their article) applications alone or a mixture with carbendazim controlled benzimidazole-resistant strains of *B. cinerea* in vineyards where such strains had predominated. As a result, diethofencarb has already started to be used practically in France in 1987. However, for *V. nashicola*, Ishii et al reported that increased sensitivity to MDPC and diethofencarb *in vitro* was observed only in highly carbendazim-resistant isolates but not in isolates with intermediate or weak resistance (5,6). Moreover, highly carbendazim-resistant isolates with resistance to MDPC and diethofencarb were found in the laboratory and in the field (Table 1). For *Cercospora beticola*, Demakopoulou and Georgopoulos (1) reported that negative correlation between benomyl and MDPC was observed only in isolates with very high resistance to benomyl. Similar results have been observed for *V. pirina* (16) and in *Pseudocercosporella herpotrichoides* (eyespot fungus of cereals). Although negatively correlated cross resistance to diethofencarb was observed in benzimidazole-resistant isolates, field isolates that were resistant to both carbendazim and diethofencarb have been obtained (Hollomon, *unpublished data*).

TABLE 4. Inheritance of reaction to carbendazim and methyl *N*-(3,5-dichlorophenyl)carbamate (MDPC) in random ascospore progenies of *Venturia nashicola* crosses

Cross	Progenies (no.)				χ^2 for MDPC sensitivity (1:1)[e]
	HR,S[a]	HR,R[b]	WR,R[c]	S,R[d]	
HR,R × S,R (JS-40M) (JS-2)	0	47	0	53	NS[f]
HR,R × S,R (JS-232) (JS-115)	0	47	0	53	NS
HR,R × WR,R (JS-11) (JS-77)	0	43	57	0	NS
HR,R × HR,S (JS-168) (JS-206)	32	18	50	0	12.96

[a] HR,S: Highly resistant to carbendazim and sensitive to MDPC.
[b] HR,R: Highly resistant to carbendazim and resistant to MDPC.
[c] WR,R: Weakly resistant to carbendazim and resistant to MDPC.
[d] S,R: Sensitive to carbendazim and resistant to MDPC.
[e] Expected value for a 1:1 ratio at $P = 0.05$ is 3.84.
[f] NS: no segregation.

Before the registration of a new fungicide, it would be desirable to assess the genetics of the fungal sensitivity and the biochemical mechanism(s) of action of the fungicide. In *V. nashicola*, benzimidazole resistance was heritable, and it was suggested that the occurrence of three different levels of resistance is due to three allelic mutations in a single gene and that each level is controlled by one of the multiple alleles (5). Moreover, the binding of ^{14}C-carbendazim to tubulinlike protein of benzimidazole-resistant isolates was lower than to the protein from a sensitive isolate, suggesting that decreased affinity of the binding sites to carbendazim might be involved in benzimidazole resistance (3). A gene conferring benzimidazole resistance may encode tubulin production in this fungus. Recently, Shabi et al (15) also reported a negatively correlated cross resistance to MDPC and diethofencarb in some benomyl-resistant isolates of *V. inaequalis* and *V. pirina*. It was shown that the negatively correlated cross resistance was heritable and controlled by a single Mendelian gene. They also mentioned that isolates very highly resistant and highly resistant to benomyl are caused by the different allelic mutations and can be distinguished by their difference of sensitivity to diethofencarb, the former sensitive and the latter not sensitive to diethofencarb. The present study indicates that the increased sensitivity to MDPC in highly carbendazim-resistant isolates was inherited (Table 2). The segregation ratio of 1 sensitive:1 resistant from the cross between a MDPC-sensitive isolate and a resistant one indicated the sensitivity to MDPC is under control of a major gene. Results of the allelism test suggested the increased sensitivity to MDPC is due to the allelic mutation in a single gene (Table 3). However, nonparental doubly resistant progenies, i.e., highly carbendazim-resistant and MDPC-resistant progenies, appeared from two crosses between a MDPC-sensitive isolate and a MDPC-resistant one. In this point, the relation between benzimidazoles and *N*-phenylcarbamates in *V. nashicola* seemed to be different from that between organophosphorus thiolate (PTL) fungicides (IBP and EDDP) and phosphoramidate (PA) compounds in *Pyricularia oryzae*. In the latter case, no doubly resistant mutants were obtained through the selection experiments with PTL or PA (7), suggesting the involvement of a single genetic factor in the negatively correlated cross resistance.

Inheritance of the double resistance was also demonstrated experimentally (Table 4). As previously reported (4), strains of *V. nashicola* intermediately or weakly resistant to carbendazim are distributed as widely as the highly resistant or the sensitive strains. In addition, ascospore production of this fungus naturally occurs in Japan. These facts suggest the possibility of the occurrence of such a doubly resistant strain in nature. Field isolates JS-11, JS-168, and JS-232 carry high benzimidazole resistance with *N*-phenylcarbamate resistance. Although the reason for appearance of the doubly resistant strains is not clear and the other nonparental types (IR,S and S,S in Table 2) were not recovered from the crosses, possibly some genetic mechanism(s) may cause these phenomena as follows: 1) same phenotypes of high carbendazim resistance are governed by different genetic factors: one increasing and one not affecting *N*-phenylcarbamate sensitivity, and/or 2) high carbendazim resistance and increased sensitivity to MDPC or diethofencarb do not result from a pleiotropism of the same gene. As shown in Table 4, nonparental type of progenies, i.e., weakly carbendazim-resistant and MDPC-resistant progenies were found from the cross between JS-168 (highly carbendazim resistant and resistant to MDPC) and JS-206 (highly carbendazim resistant and sensitive to MDPC). This result might support the possibility of the genetic mechanism mentioned above in *V. nashicola*. Further research needs to be done to elucidate the mechanism(s) of increased sensitivity and double resistance in more details.

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