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Effects of Host Plant Resistance and Fungicides on Severity of Cucumber Downy Mildew

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***Abstract*. Cucurbit downy mildew caused by the oomycete *Pseudoperonospora cubensis* (Berk. And Curt) Rostov is a major disease of cucumber (*Cucumis sativus* L.) (Palti and Cohen, 1980) globally. Chemical control of downy mildew is necessary to achieve high yields in the absence of adequate host plant resistance. Most of the currently grown cultivars have some resistance to downy mildew. Before the resurgence of the disease in 2004, host plant resistance was sufficient to control the disease without fungicide use, and downy mildew was only a minor problem on cucumber. There are currently no cultivars that show resistance at a level equal to that observed before 2004. However, differences in resistance exist among cultivars, ranging from moderately resistant to highly susceptible. In this study, we evaluated the disease severity and yield of four cucumber cultivars that differed in disease resistance and were treated with fungicide programs representing a range of efficacy levels. The experiment was a split plot design with six replications and four years. Disease was evaluated as chlorosis, necrosis, and reduction in plant size on a 0 to 9 scale. Cultigen had a large effect in all four years. Fungicide has a smaller effect on resistance component traits and a larger effect on yield traits. The effects of cultivar resistance and fungicides appear to be additive until a threshold where maximum yield is reached. Highly resistant cultigens such as PI 197088 required only the least effective fungicides to achieve highest yields, whereas moderately resistant cultigens required a more effective fungicide to reach a similar level of yield. Susceptible cultigens did not achieve high yield even with the most effective fungicide treatments. It is likely that, even as highly resistant cultivars are released, growers will need to continue a minimal fungicide program to achieve maximum yield.**

Cucurbit downy mildew caused by the oomycete *Pseudoperonospora cubensis* is economically the most important disease of cucumber (*Cucumis sativus* L.) (Palti and Cohen, 1980). Studies on the host range of *P. cubensis* indicate that ≈20 genera are hosts, including 50 species in the Cucurbitaceae and 19 host species genus *Cucumis* alone (Lebeda, 1992; Lebeda and Widrlechner, 2003; Palti and Cohen, 1980). In 2010, ≈35,840 ha of cucumbers for processing and fresh market were grown in the United States with a value of $378 million [U.S. Department of Agriculture (USDA), 2011]. Other economically important hosts of *P. cubensis* are melon (*Cucumis melo* L.), watermelon (*Citrullus lanatus*), and squash (*Cucurbita* spp.) (Whitaker and Davis, 1962). The pathogen infects when wind-blown sporangia are introduced onto cucurbit hosts under favorable environmental conditions.

P. cubensis is a biotroph and, with the exception of oospore production, survives only on living host tissue (Bains and Jhooty, 1976). Previously, oospore production was thought to be rare, but Cohen et al. (2011) reported recently on oospore formation in the laboratory from crosses between different pathotypes, resulting in the production of viable F1 recombinants. In warm production regions such as southern Florida, overwintering occurs on wild and cultivated cucurbits (Bains and Jhooty, 1976). The pathogen can also overwinter on cucumbers grown in greenhouses. Hausbeck (2007) reported the possibility of cucumbers in greenhouses in Ontario as a local source of *P. cubensis* inoculum for Canada and neighboring states in the Great Lakes region.

Environmental conditions affect overwintering capacity as well as disease development and intensity. Leaf moisture is required for germination of sporangia. Rain, dew, or irrigation can easily supply enough moisture for sporangia to germinate. Under optimum temperature, infection can occur with only 2 h of leaf wetness (Cohen, 1977). The level of occurrence for compatible reactions is a result of the combination of time, moisture, temperature, and inoculum concentration.

Symptoms of cucumber downy mildew occur almost exclusively on the foliage. Infection first appears as small, water-soaked lesions on the underside of leaves. Symptoms vary by cucurbit species but in cucumber lesions are angular, bounded by leaf veins, and turn chlorotic to varying degrees. Sporulation occurs on the undersides of the leaves. Chlorotic lesions eventually turn necrotic and the entire leaf may be affected by the pathogen as the leaf tissue dies. Symptoms vary depending on relative susceptibility of host plants. The most resistant will exhibit a hypersensitive response (HR) with small necrotic or chlorotic flecks and sparse sporulation, whereas leaves of the most susceptible will become completely necrotic within 2 to 3 weeks.

The HR type resistance was first described by Barnes and Epps (1954) in the accession PI 197087. Resistance from PI 197087 was used to develop resistant cultivars, and most current cultivars are thought to have some resistance derived from PI 197087. This resistance proved highly effective for many years until a resurgence of the disease in 2004. Since then, cultivars having resistance tracing to PI 197087 are only moderately resistant in the United States (Call et al., 2012a). However, differences among cultivars do exist, ranging from moderately resistant to highly susceptible. New sources of disease resistance have been reported (Call et al., 2012b) but this resistance has not yet been incorporated into cultivars.

Chemical control of downy mildew is necessary to achieve high yields in the absence of high host plant resistance. The discovery of systemic fungicides was a major advance over protectant fungicides in control of downy mildew. Systemic fungicides, in the absence of resistant biotypes, can provide effective control. Cohen (1979) reported on the effectiveness of two systemic fungicides, prothiocarb and propamocarb (derivative of prothiocarb), against downy mildew. Both prothiocarb and propamacarb were reported to have very good activity against downy mildew. Briggs et al. (2006) reported fluopicolide as having a novel mode of action controlling a wide range of oomycete pathogens. Typically, a protectant fungicide and a systemic fungicide are tank-mixed and alternated weekly with a different tank mix consisting of fungicides with different modes

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of action. An example of such a program is propamocarb (Previcur Flex) and chlorothalonil (Bravo) alternating with famoxadone + cymoxanil (Tanos) + mancozeb (Manzate).

In this study, we evaluated fungicide programs with different levels of efficacy against downy mildew in combination with cultivars or breeding lines (hereafter collectively referred to as cultigens) having different levels of resistance for their effect on disease severity and yield.

Materials and Methods

Experiment design. Field tests were done during the summer from 2008 to 2011 at the Horticultural Crops Research Station in Clinton, NC. All cucumbers were grown using recommended horticultural practices as summarized by Schultheis (1990). Fertilizer was incorporated before planting at a rate of 90N–39P–74K kg·ha^{-1} with an additional 34 kg nitrogen/ha applied at the vine-tip-over stage (four to six true leaves). There were six (2008 to 2010) and eight (2011) plot rows surrounded by two border rows. End borders were used at the front and back of test plot rows. End borders were 1.6 m long separated from test plots by 1.6-m alleys. Plots were hand-seeded on raised, shaped beds with centers 1.5 m apart and plots 1.6 m (2009) or 3.2 m (2008 to 2011) long. Plots were separated at each end by 1.6-m alleys. Two plot lengths were used in 2009 (1.6 m and 3.2 m), each in individual blocks, with the 3.2-m plots planted 11 d after the shorter plots. Two plot lengths were used as a result of incorrect field layout on the first planting date. The study was planted on 10 July 2008, 18 June 2009 (1.6 m), 29 June 2009 (3.2 m), 15 July 2010, and 8 July 2011.

Plots and borders were planted when inoculum was present in adjacent cucumber fields (within 50 m). Plots were harvested twice, at 2-week intervals, by hand and graded into marketable and cull fruit when the largest fruit reached Size 4 (greater than 2 in) or oversized according to industry standards (6 to 8 weeks after planting). Cull fruit were crooked (curved) or constricted enough to seriously effect fruit appearance according to industry standards (USDA, 1958). Number of fruit and total weight were recorded for marketable and cull fruit for each plot. Percent early yield is the percent of yield from Harvest 1. Yield data for the 1.6-m plots were doubled in the analysis to estimate the yield on a 3.2-m plot basis and to compare with the rest of the study. There was likely a border effect on yield as shown by Wehner (1984) indicating the estimate obtained by doubling will be biased slightly upward. Because this is equal for all treatments, there will be no rank change, and because these were not yield trials, this bias is acceptable.

The experiment was a split plot design with six replications per year. There were three (2008) and four (2009 to 2011) fungicide programs used as the whole plots. Three (2008 to 2010) and four (2011) cultigens were used as the subplots. Data were analyzed using the General Linear Model, GLIMIX, Means, and Correlation procedures of SAS (SAS Institute, 2008).

Germplasm and fungicide treatments. Four cucumber cultigens differing in disease resistance were used to evaluate severity of disease based on previous studies at North Carolina State University (Call et al., 2012a, 2012b; Shetty et al., 2002; Wehner and Shetty, 1997) (Table 1): M 21 (North Carolina State Univ.) is moderately resistant, 'Sumter' (Clemson Univ.) is slightly resistant, 'Wisconsin SMR-18' (Wisconsin AES) is highly susceptible, and PI 197088 (USDA-ARS) is highly resistant (2011 only). The cultigens used are not elite cultivars that could be marketed directly but have fruit that can be described as unmarketable if environmental conditions affect development.

Fungicide programs were selected based on results from annual fungicide efficacy tests conducted in North Carolina (Colucci et al., 2008b, 2008c; Kanetis et al., 2009a, 2009b) (Table 1). Fungicide treatments were applied using a CO_2-pressurized backpack sprayer equipped with hollow cone nozzles (TXVS-26; TeeJet Inc., Springfield, IL) delivering 61 L·ha^{-1} at 310 kPa. Application rates were as per label instructions and are presented in Table 2. Fungicides were applied weekly for all treatments, beginning at the first true leaf stage, before the appearance of disease symptoms on test plots, with seven applications each year.

Field inoculation and disease ratings. No artificial inoculum was used and plots were exposed to natural epidemics during the course of the growing season. Susceptible cultivar Coolgreen (Asgrow) was used for side and end borders to monitor and increase inoculum in the field. Epidemics were encouraged using overhead irrigation at least 3 d per week.

Disease severity was evaluated weekly as chlorotic and necrotic lesions and degree of stunting. Chlorosis and necrosis were rated on a 0 to 9 scale based on percentage of symptomatic leaf area (0 = 0%, 1 = 1% to 3%, 2 = 3% to 6%, 3 = 6% to 12%, 4 = 12% to 25%, 5 = 25% to 50%, 6 = 50% to 75%, 7 = 75% to 87%, 8 = 87% to 99%, 9 = 100%) as described by Jenkins and Wehner (1983). Stunting was rated on the same 0 to 9 scale as reduction in plant size relative to observations on the same cultigens planted in fungicide-treated, non-inoculated trials planted in adjacent fields. Even without disease, different genotypes have different plant sizes. For instance, M 21 is a dwarf determinate type with a naturally smaller habit than the other cultigens used in this study. Because of this fact, this rating is used to compare the effect of fungicides within a cultigen but cannot be used to compare cultigens. It allows us to identify those fungicide treatments under which plants remain large under a disease epidemic.

Table 1. Fungicide and cultigen treatments used.

Fungicide treatments		Fungicide efficacy
1.	Control	None
2.	Mancozeb applied weekly	Slightly effective
3.	Famoxadone + cymoxanil + mancozeb alternating weekly with propamocarb-hydrochloride + chlorothalonil	Moderately effective
4.	Cyazofamid + mancozeb alternating weekly with fluopicolide + chlorothalonil (added in 2009)	Highly effective
Cultigens		**Cultigen resistance**
1.	Wisconsin SMR-18	Susceptible
2.	Sumter	Slightly resistant
3.	M 21	Moderately resistant
4.	PI 197088 (2011 only)	Highly resistant

Results and Discussion

Data were analyzed using means of all ratings for each trait. There was no plot size effect on disease ratings in 2009 so these data

Table 2. Trade names, active ingredients, fungicide group name, and supply company for fungicides used in 2008 and 2009.

Trade name	Active ingredient	Group name and FRAC code[y]	Application rate	Supply company
Manzate® Pro-Stick™	Mancozeb	Multisite inhibitors	0.37 kg·ha^{-1}	E.I. Dupont de Nemours and Co.
Bravo Weather-Stick®	Chlorothalonil	Multisite inhibitors	0.39 L·ha^{-1}	Syngenta Crop Protection, Inc.
Tanos®	Famoxadone + cymoxanil	Quinone outside inhibitors (C3) Cyanoacetamide-oximes	0.10 L·ha^{-1}	E.I. Dupont de Nemours and Co.
Previcur® Flex	Propamocarb-hydrochloride	Carbamates (F4)	0.23 L·ha^{-1}	Bayer CropScience
Presidio®[z]	Fluopicolide	Pyridinylmethyl-benzamides (B5)	0.04 L·ha^{-1}	Valent USA Corporation
Ranman®[z]	Cyazofamid	Quinone inside inhibitors (C4)	0.04 L·ha^{-1}	FMC Agricultural Products

[z]Only used in 2009.
[y]Fungicide Resistance Action Committee (http://frac.info).

were pooled (data not shown). Two basic data sets were analyzed to account for added treatments: 1) 2008 to 2011 with fungicide programs 1 to 4 (Table 1) and cultigens 1 to 3; and 2) 2011 with fungicide programs 1 to 4 and cultigens 1 to 4.

Four fungicide programs, three cultigens (2008 to 2011). PI 197088 is excluded from this data set. In 2009 a new fungicide treatment was added to the study to represent a higher efficacy fungicide program (fluopicolide + chlorothalonil alternated with cyazofamid + mancozeb). The analysis of variance results (Table 3) indicate that differences in both cultigen and fungicide treatments contribute significantly ($P < 0.001$) to differences in disease severity as well as total and percent marketable yield. A larger cultigen effect was observed for chlorosis and necrosis compared with the fungicide effect on the same traits. For stunting, cultigen had a smaller effect than fungicide.

All correlations were calculated using the Pearson product-moment and Spearman's rank methods (data not shown). Yield data were generally highly correlated among environments ($P < 0.01$). Chlorosis and necrosis were significantly highly correlated (R^2 = 0.90) for both the Pearson and the Spearman tests, indicating they are likely the same trait. Stunting was less correlated with chlorosis and necrosis (Pearson $R^2 = 0.26$, $P < 0.001$).

Disease and yield data for 2008 to 2011 with all fungicide treatments and cultigens M 21, 'Sumter', and 'Wisconsin SMR-18' are summarized in Table 4 as least squares means. The resistant cultigen M 21 on average had higher yields and less disease than the moderately resistant 'Sumter' and susceptible 'Wisconsin SMR-18' in all yield traits and all disease traits with the exception of stunting and fruit size. M 21 is a determinate type and has a naturally smaller habit than indeterminate types. Differences can be seen in both yield and disease traits among cultigen and fungicide treatments. The effects of moving to a cultivar with more resistance appears additive for chlorosis, necrosis, yield, and percent marketable yield. Surprisingly, chlorosis and necrosis severity are reduced only slightly as one moves to higher efficacy fungicide programs. A larger effect is seen on plant stunting. Reduced stunting is equivalent to increased leaf area and more photosynthetic potential. The yield increase observed with more effective fungicide treatments could be partially the result of the reduction in stunting.

The highest total and percent marketable yield along with the lowest disease ratings within each cultigen were observed achieved with the high efficacy fungicide treatment fluopicolide + chlorothalonil alt. cyazofamid + mancozeb (Table 3). The combination of the highly susceptible 'Wisconsin SMR-18' with no fungicide treatment resulted in the highest disease ratings and lowest yield. In general, the disease and yield traits measured improve by moving to a more resistant cultivar or more effective fungicide program.

Disease severity ratings for the new high efficacy fungicide treatment (fluopicolide + chlorothalonil alt. cyazofamid + mancozeb) were not significantly lower than the previous high-efficacy (propamocarb + chlorothalonil alternating with famoxadone + cymoxanil + mancozeb) treatment with the exception of necrosis ratings in cultigen M 21; however, the means were lower for all tests. Fungicide had a much larger effect on yield traits with the largest yield difference among fungicide treatments observed in the less resistant cultigens. The mean total yield of 'Sumter' under the fluopicolide + chlorothalonil alternated with cyazofamid + mancozeb treatment was 22.2 $Mg \cdot ha^{-1}$, an increase of 20.0 $Mg \cdot ha^{-1}$ over the non-treated 'Sumter' plots. Similar results were observed for other fungicide treatments. In general, disease ratings for the propamocarb + chlorothalonil alternating with famoxadone + cymoxanil + mancozeb treatment were lower but not significantly different from mancozeb alone for chlorosis, necrosis, and stunting. Although these plots appeared similar as far as disease appearance, the difference in yield was significant between the treatments.

In a study by Kanetis et al. (2009a) in 2008, conducted in Clayton, NC, propamocarb showed significantly less disease [area under the disease progress curve (AUDPC)] than both famoxadone + cymoxanil and mancozeb, which were not significantly different. In 2009 a study from the same group (Adams and Ojiambo, 2010) at the same location, showed mancozeb and propamocarb (not significantly different) had a significantly lower AUDPC than famoxadone + cymoxanil. In studies conducted in 2009 in Faison, NC, the mancozeb treatment had a significantly lower AUDPC than both propamocarb and famoxadone + cymoxanil (Adams et al., 2010). These results may indicate some loss in effectiveness by famoxadone + cymoxanil and propamocarb, although more studies should be done to evaluate this observation. Yield data were not reported for these studies, which would have been helpful in determining treatment efficacy.

Our data show that although apparent disease may not be reduced greatly with improved fungicide programs, the effect on yield was large. In our study, the propamocarb + chlorothalonil alternating with famoxadone + cymoxanil + mancozeb treatment yielded significantly higher (least significant difference 5% = 1.8) than mancozeb in all cultigens. The newly added program, fluopicolide + chlorothalonil alternating with cyazofamid + mancozeb, generally outperformed all other treatments for both disease and yield.

Table 3. Analysis of variance of downy mildew resistance component and yield trait means for data collected in Clinton, NC, in 2008 to 2011, excluding PI 197088 (top) and for data collected in 2011 for only the cultigen PI 197088 (bottom).[z]

2008–11 NO PI 197088		Resistance components[y]			Yield components[x]			
Source	df	Chlorosis	Necrosis[x]	Stunting	Total yield ($Mg \cdot ha^{-1}$)	Percent market yield[w]	Percent early yield[v]	Fruit size (kg/fruit)
Year	3	22.34***	3.54**	23.92***	849.85***	4084**	7492**	0.1666**
Replications (year)	20	0.66*	0.66	1.55***	61.23**	588**	1432***	0.0256
Fungicide	3	30.78***	33.72***	78.38***	3161.78***	18995***	666	0.0653
Fungicide*year	8	3.05***	3.82***	5.00***	115.82***	682	374	0.0352
Replications (fungicide*year)	55	0.31	0.38	0.65	14.72	347	443	0.0418
Cultigen	2	445.94***	224.55***	9.08***	1926.96***	16419***	7230***	0.0106
Cultigen*year	6	3.36***	8.50***	17.89***	115.28**	726*	401	0.0642
Cultigen*fungicide	6	0.86*	1.38*	0.70	99.03**	2313***	450	0.0229
Year*cultigen*fungicide	16	0.75*	0.62	1.10*	16.19	294	301	0.0440
Error	222	0.41	0.63	0.65	31.94	287	437	0.0341
2011—PI 197088 only								
Fungicide	3	0.04	0.24	2.82**	67.98	40**	43	0.0002
Replication	5	0.05	0.15	0.56	89.27	22*	250**	0.0052**
Error	15	0.03	0.13	0.50	80.44	7	44	0.0012

[z]Data are means of six (2008, 2010, 2011) and 12 (2009) replications.
[y]Data are means or all ratings.
[x]Data are from two harvests.
[w]Percent marketable yield is percent non-cull fruit.
[v]Percent early yield is yield from Harvests 1 of 2.
*, **, ***Significant at 0.05, 0.01, and 0.001, respectively.

Table 4. Disease resistance component ratings and yield components for combinations of cultigens and fungicide programs for the control of downy mildew in cucumber.[z]

		Resistance components			Yield components			
		Chlorosis mean	Necrosis mean	Stunting mean	Total yield (Mg·ha^{-1})	Percent market yield[v]	Percent early yield[u]	Fruit size (kg/fruit)[t]
Cultigen	Fungicide program[x]	EST ± SEM	EST ± SEM	EST ± SEM	EST ± SEM	EST ± SEM	EST ± SEM	EST ± SEM
M 21	Fluopicolide + chlorothalonil alt. cyazofamid + mancozeb[w]	2.00 ± 0.42 ^{As}	2.83 ± 0.37 ^{A}	3.40 ± 0.53 ^{ABC}	20.92 ± 2.51 ^{A}	68.80 ± 8.61 ^{A}	34.7 ± 7.5 ^{BCD}	0.19 ± 0.05 ^{A}
	Famoxadone + cymoxanil + mancozeb alt. propamacarm + chlorothalonil	2.52 ± 0.39 ^{AB}	3.58 ± 0.34 ^{B}	3.69 ± 0.50 ^{ABC}	15.99 ± 2.35 ^{B}	69.47 ± 8.26 ^{A}	36.6 ± 7.2 ^{BC}	0.18 ± 0.05 ^{A}
	Mancozeb	2.74 ± 0.39 ^{AB}	3.95 ± 0.34 ^{BC}	4.37 ± 0.50 ^{CDE}	11.39 ± 2.35 ^{CD}	60.90 ± 8.26 ^{AB}	41.0 ± 7.2 ^{AB}	0.15 ± 0.05 ^{A}
	None	3.65 ± 0.39 ^{CD}	4.64 ± 0.34 ^{CD}	5.27 ± 0.50 ^{E}	08.06 ± 2.35 ^{DE}	55.74 ± 8.26 ^{BC}	48.2 ± 7.2 ^{A}	0.13 ± 0.05 ^{A}
Sumter	Fluopicolide + chlorothalonil alt. cyazofamid + mancozeb	3.07 ± 0.42 ^{BC}	4.07 ± 0.37 ^{BC}	2.27 ± 0.53 ^{A}	22.22 ± 2.51 ^{A}	66.19 ± 8.63 ^{AB}	23.5 ± 7.5 ^{CDE}	0.23 ± 0.05 ^{A}
	Famoxadone + cymoxanil + mancozeb alt. propamacarm + chlorothalonil	3.86 ± 0.39 ^{C}	4.94 ± 0.34 ^{CD}	3.15 ± 0.50 ^{ABC}	10.90 ± 2.35 ^{CD}	42.67 ± 8.28 ^{C}	26.6 ± 7.2 ^{CD}	0.17 ± 0.05 ^{A}
	Mancozeb	4.29 ± 0.39 ^{DE}	5.28 ± 0.34 ^{DE}	4.00 ± 0.50 ^{BCDE}	05.91 ± 2.35 ^{EF}	28.17 ± 8.26 ^{D}	21.6 ± 7.2 ^{DE}	0.15 ± 0.05 ^{A}
	None	4.75 ± 0.39 ^{E}	6.06 ± 0.34 ^{FG}	4.92 ± 0.50 ^{DE}	02.19 ± 2.35 ^{FG}	11.68 ± 8.58 ^{E}	27.9 ± 7.5 ^{BCD}	0.14 ± 0.06 ^{A}
Wisconsin SMR-18	Fluopicolide + chlorothalonil alt. cyazofamid + mancozeb	6.14 ± 0.42 ^{F}	6.05 ± 0.37 ^{EF}	2.86 ± 0.53 ^{AB}	14.60 ± 2.51 ^{BC}	64.23 ± 8.63 ^{AB}	23.4 ± 7.5 ^{CDE}	0.23 ± 0.05 ^{A}
	Famoxadone + cymoxanil + mancozeb alt. propamacarm + chlorothalonil	6.72 ± 0.39 ^{FG}	6.80 ± 0.34 ^{GH}	3.58 ± 0.50 ^{ABCD}	04.69 ± 2.35 ^{EFG}	44.79 ± 8.28 ^{C}	30.0 ± 7.2 ^{BCD}	0.27 ± 0.05 ^{A}
	Mancozeb	7.10 ± 0.39 ^{GH}	6.89 ± 0.34 ^{GH}	4.30 ± 0.50 ^{CDE}	01.96 ± 2.35 ^{FG}	26.01 ± 8.36 ^{DE}	13.5 ± 7.3 ^{E}	0.16 ± 0.06 ^{A}
	None	7.40 ± 0.39 ^{H}	7.17 ± 0.34 ^{H}	5.36 ± 0.50 ^{E}	00.51 ± 2.35 ^{G}	–2.50 ± 8.79 F	20.8 ± 7.9 ^{DE}	0.16 ± 0.14 ^{A}
M 21		2.73 ± 0.34 ^{A}	3.75 ± 0.28 ^{A}	4.19 ± 0.43 ^{A}	14.09 ± 2.02 ^{A}	63.73 ± 7.56 ^{A}	40.1 ± 6.4 ^{A}	0.16 ± 0.04 ^{A}
Sumter		3.99 ± 0.34 ^{B}	5.09 ± 0.28 ^{B}	3.58 ± 0.43 ^{A}	10.31 ± 2.02 ^{B}	37.18 ± 7.60 ^{B}	24.9 ± 6.5 ^{B}	0.17 ± 0.04 ^{A}
Wisconsin SMR-18		6.84 ± 0.34 ^{C}	6.72 ± 0.28 ^{C}	4.02 ± 0.43 ^{A}	05.44 ± 2.02 ^{C}	33.13 ± 7.62 ^{B}	21.9 ± 6.5 ^{B}	0.20 ± 0.05 ^{A}
	Fluopicolide + chlorothalonil alt. cyazofamid + mancozeb	3.74 ± 0.36 ^{A}	4.32 ± 0.27 ^{A}	2.84 ± 0.39 ^{A}	19.25 ± 2.20 ^{A}	66.40 ± 7.90 ^{A}	27.2 ± 6.5 ^{A}	0.22 ± 0.04 ^{A}
	Famoxadone + cymoxanil + mancozeb alt. propamacarm + chlorothalonil	4.37 ± 0.34 ^{AB}	5.11 ± 0.24 ^{B}	3.47 ± 0.36 ^{AB}	10.53 ± 2.06 ^{B}	52.31 ± 7.65 ^{B}	31.1 ± 6.3 ^{A}	0.21 ± 0.04 ^{A}
	Mancozeb	4.71 ± 0.34 ^{BC}	5.38 ± 0.24 ^{BC}	4.22 ± 0.36 ^{B}	06.42 ± 2.06 ^{C}	38.36 ± 7.66 ^{C}	25.4 ± 6.3 ^{A}	0.15 ± 0.04 ^{A}
	None	5.27 ± 0.34 ^{C}	5.96 ± 0.24 ^{C}	5.18 ± 0.36 ^{C}	03.59 ± 2.06 ^{C}	21.64 ± 7.78 ^{D}	32.3 ± 6.5 ^{A}	0.14 ± 0.06 ^{A}

[z]Data are means of 12 (2009) and six (2008, 2010, 2011) replications and two harvests. Ratings were 0 to 9 (0 = 0%, 1 = 1% to 3%, 2 = 3% to 6%, 3 = 6% to 12%, 4 = 12% to 25%, 5 = 25% to 50%, 6 = 50% to 75%, 7 = 75% to 87%, 8 = 87% to 99%, 9 = 100%) for chlorosis, necrosis, and stunting.
[y]Fungicide treatments were applied weekly.
[x]Treatment not tested in 2008.
[w]Percent marketable yield is total yield that is non-culled fruit.
[v]Percent early yield was the percentage of total yield obtained in Harvest 1 (out of 2).
[u]Fruit size was calculated from marketable fruit.
[t]Results with the same letter in the same column are not significantly different ($P < 0.05$).

Table 5. Disease resistance component ratings and yield components for the combinations of highly resistant PI 197088 and fungicide programs for the control of downy mildew in cucumber.[z]

		Resistance components			Yield components			
		Chlorosis mean	Necrosis mean	Stunting mean	Total yield ($Mg \cdot ha^{-1}$)	Percent market yield[v]	Percent early yield[u]	Fruit size (kg/fruit)[t]
Cultigen	Fungicide program[x]	EST ± SEM	EST ± SEM	EST ± SEM	EST ± SEM	EST ± SEM	EST ± SEM	EST ± SEM
PI 197088	Fluopicolide + chlorothalonil alt. cyazofamid + mancozeb[w]	1.00 ± 0.23 ^{As}	1.83 ± 0.25 ^{A}	1.58 ± 0.31 $_{A}$	29.22 ± 2.62 ^{AB}	89.87 ± 5.34 ^{AB}	4.33 ± 10.19 ^{E}	0.32 ± 0.02 ^{A}
	Famoxadone + cymoxanil + mancozeb alt. propamacarm + chlorothalonil	1.11 ± 0.23 ^{A}	1.56 ± 0.25 ^{A}	1.58 ± 0.31 $_{A}$	28.76 ± 2.62 ^{AB}	92.93 ± 5.34 ^{A}	3.96 ± 10.19 ^{E}	0.31 ± 0.02 ^{A}
	Mancozeb	1.17 ± 0.23 ^{A}	1.89 ± 0.25 ^{A}	1.75 ± 0.31 $_{A}$	31.14 ± 2.62 ^{A}	95.15 ± 5.34 ^{A}	7.60 ± 10.19 ^{E}	0.31 ± 0.02 ^{A}
	None	1.17 ± 0.23 ^{A}	2.00 ± 0.25 ^{A}	3.00 ± 0.31 $_{B}$	23.30 ± 2.62 ^{BC}	95.49 ± 5.34 ^{A}	1.06 ± 10.19 ^{E}	0.32 ± 0.02 ^{A}
M 21	Fluopicolide + chlorothalonil alt. cyazofamid + mancozeb	2.56 ± 0.23 ^{B}	3.22 ± 0.25 ^{BC}	3.00 ± 0.31 $_{B}$	27.14 ± 2.62 ^{AB}	78.62 ± 5.34 ^{BC}	57.41 ± 10.19 ^{AB}	0.20 ± 0.02 ^{CDE}
	Famoxadone + cymoxanil + mancozeb alt. propamacarm + chlorothalonil	2.39 ± 0.23 ^{B}	2.94 ± 0.25 ^{B}	3.25 ± 0.31 $_{BC}$	26.91 ± 2.62 ^{AB}	77.20 ± 5.34 ^{BC}	51.06 ± 10.19 ^{ABC}	0.21 ± 0.02 ^{BCD}
	Mancozeb	2.72 ± 0.23 ^{B}	3.50 ± 0.25 ^{BC}	4.42 ± 0.31 $_{D}$	14.98 ± 2.62 ^{DE}	65.64 ± 5.34 ^{C}	45.54 ± 10.19 ^{ABC}	0.16 ± 0.02 ^{DEF}
	None	4.56 ± 0.23 ^{DE}	4.78 ± 0.25 ^{DE}	4.58 ± 0.31 $_{D}$	9.73 ± 2.62 ^{EF}	66.28 ± 5.34 ^{C}	63.56 ± 10.19 ^{A}	0.12 ± 0.02 ^{FG}
Sumter	Fluopicolide + chlorothalonil alt. cyazofamid + mancozeb	3.61 ± 0.23 ^{C}	3.83 ± 0.25 ^{C}	2.67 ± 0.31 $_{B}$	26.01 ± 2.62 ^{AB}	77.18 ± 5.34 ^{BC}	39.69 ± 10.19 ^{BCD}	0.25 ± 0.02 ^{B}
	Famoxadone + cymoxanil + mancozeb alt. propamacarm + chlorothalonil	4.00 ± 0.23 ^{CD}	4.50 ± 0.25 ^{D}	2.75 ± 0.31 $_{B}$	18.48 ± 2.62 ^{CD}	65.37 ± 5.34 ^{C}	45.36 ± 10.19 ^{ABC}	0.21 ± 0.02 ^{BCD}
	Mancozeb	4.78 ± 0.23 ^{E}	5.28 ± 0.25 ^{E}	4.42 ± 0.31 $_{D}$	9.44 ± 2.62 ^{EF}	40.07 ± 5.34 ^{D}	38.64 ± 10.19 ^{BCD}	0.16 ± 0.02 ^{CDEF}
	None	6.33 ± 0.23 ^{F}	6.72 ± 0.25 ^{F}	5.58 ± 0.31 $_{E}$	3.27 ± 2.62 ^{FG}	25.43 ± 5.34 ^{EF}	46.75 ± 10.19 ^{ABC}	0.10 ± 0.02 ^{G}
Wisconsin SMR-18	Fluopicolide + chlorothalonil alt. cyazofamid + mancozeb[w]	7.33 ± 0.23 ^{G}	7.11 ± 0.25 ^{FG}	4.00 ± 0.31 $_{CD}$	13.86 ± 2.62 ^{DE}	76.76 ± 5.34 ^{BC}	32.36 ± 10.19 ^{CD}	0.22 ± 0.02 ^{BC}
	Famoxadone + cymoxanil + mancozeb alt. propamacarm + chlorothalonil	7.44 ± 0.23 ^{G}	7.67 ± 0.25 ^{GH}	4.42 ± 0.31 $_{D}$	6.00 ± 2.62 ^{FG}	67.56 ± 5.34 ^{C}	41.53 ± 10.19 ^{ABC}	0.20 ± 0.02 ^{CD}
	Mancozeb	8.28 ± 0.23 ^{H}	8.17 ± 0.25 ^{H}	6.33 ± 0.31 $_{EF}$	2.38 ± 2.62 ^{G}	36.76 ± 5.34 ^{DE}	17.47 ± 10.19 ^{DE}	0.14 ± 0.02 ^{EFG}
	None	8.28 ± 0.23 ^{H}	8.28 ± 0.25 ^{H}	6.58 ± 0.31 $_{F}$	0.89 ± 2.62 ^{G}	11.80 ± 6.27 ^{F}	55.25 ± 11.58 ^{ABC}	0.12 ± 0.03 ^{FG}
PI 197088		1.11 ± 0.13 ^{A}	1.82 ± 0.15 ^{A}	1.98 ± 0.18 $_{A}$	28.10 ± 1.66 ^{A}	93.36 ± 3.68 ^{A}	4.24 ± 7.86 ^{C}	0.32 ± 0.01 ^{A}
M 21		3.06 ± 0.13 ^{B}	3.61 ± 0.15 ^{B}	3.81 ± 0.18 $_{B}$	19.69 ± 1.66 ^{B}	71.94 ± 3.68 ^{B}	54.39 ± 7.86 ^{A}	0.17 ± 0.01 ^{B}
Sumter		4.68 ± 0.13 ^{C}	5.08 ± 0.15 ^{C}	3.85 ± 0.18 $_{B}$	14.30 ± 1.66 ^{C}	52.01 ± 3.68 ^{C}	42.61 ± 7.86 ^{AB}	0.18 ± 0.01 ^{B}
Wisconsin SMR-18		7.83 ± 0.13 ^{D}	7.81 ± 0.15 ^{D}	5.33 ± 0.18 $_{C}$	5.78 ± 1.66 ^{D}	48.22 ± 3.77 ^{C}	36.65 ± 7.98 ^{B}	0.17 ± 0.02 ^{B}
	Fluopicolide + chlorothalonil alt. cyazofamid + mancozeb	3.63 ± 0.15 ^{A}	4.00 ± 0.14 ^{A}	2.81 ± 0.20 $_{A}$	24.06 ± 1.66 ^{A}	80.61 ± 3.51 ^{A}	33.45 ± 7.79 ^{A}	0.25 ± 0.01 ^{A}
	Famoxadone + cymoxanil + mancozeb alt. propamacarm + chlorothalonil	3.74 ± 0.15 ^{B}	4.17 ± 0.14 ^{B}	3.00 ± 0.20 $_{B}$	20.04 ± 1.66 ^{B}	75.76 ± 3.51 ^{A}	35.48 ± 7.79 ^{A}	0.23 ± 0.01 ^{A}
	Mancozeb	4.24 ± 0.15 ^{C}	4.71 ± 0.14 ^{C}	4.23 ± 0.20 $_{C}$	14.48 ± 1.66 ^{C}	59.41 ± 3.51 ^{B}	27.31 ± 7.79 ^{A}	0.20 ± 0.01 ^{B}
	None	5.08 ± 0.15 ^{C}	5.44 ± 0.14 ^{C}	4.94 ± 0.20 $_{C}$	9.30 ± 1.66 ^{D}	49.75 ± 3.60 ^{C}	41.66 ± 7.91 ^{A}	0.16 ± 0.01 ^{C}

[z]Data are means of six replications and two harvests. Ratings were 0 to 9 (0 = 0%, 1 = 1% to 3%, 2 = 3% to 6%, 3 = 6% to 12%, 4 = 12% to 25%, 5 = 25% to 50%, 6 = 50% to 75%, 7 = 75% to 87%, 8 = 87% to 99%, 9 = 100%) for chlorosis, necrosis, and stunting.
[y]Fungicide treatments were applied weekly.
[x]Treatment not tested in 2008.
[w]Percent marketable yield is total yield that is non-culled fruit.
[v]Percent early yield was the percentage of total yield obtained in Harvest 1 (out of 2).
[u]Fruit size was calculated from marketable fruit.
[t]Results with the same letter in the same column are not significantly different ($P < 0.05$).

Cultigen resistance seems to be more important for overall disease reduction than fungicides, but each contributed similarly to yield. It is likely that the benefits of fungicide application would be greater than data indicate in terms of severity of disease in a grower field because in our tests, neighboring plots that were not treated with fungicide may have increased spore density in the field, causing more disease. All borders were untreated and planted with a highly susceptible control as well. A field that is treated with complete fungicide coverage should be less affected, assuming neighboring fields are being controlled as well.

In our study, plots and borders were planted when inoculum was present in adjacent cucumber fields. This means that infection likely occurred before fungicides being applied (post-infection, before disease symptom appearance), which negatively impacts fungicide efficacy (Colucci et al., 2008a). Although fungicides are much more effective if applications are started before the arrival of inoculum, a typical grower will begin spraying only when symptoms appear or if disease has been reported in their area to minimize the costs of fungicide application. Growers who initiate a spray program before the appearance of disease symptoms would achieve superior results.

Four fungicide programs, four cultigens (2011). In 2011 the cultigen PI 197088 was added to the test as the representative for high resistance. PI 197088 shows few lesions when challenged with *P. cubensis*, is large, and has high, but late, yield. A summary of disease severity ratings and yield results for PI 197088 compared with the other cultigens in 2011 only is shown in Table 5. In this study, PI 197088 was highly resistant and significantly better for all rated disease and yield traits (except percent early) than other cultigens tested. The total yield of PI 197088 was not significantly different for the four fungicide programs, but treated plot means were similar and higher than the control. It appears that the highly resistant PI 197088 receives a small boost to yield from a fungicide treatment, indicating that even if this high resistance is incorporated into marketable cultivars, some fungicide application will be beneficial.

Conclusions

Both host resistance and fungicide treatment together contribute to plant performance in terms of reducing disease severity and increasing yields. This study examined how fungicide efficacy and host resistance interact to affect these traits. The cultigens used in this study are not isolines differing only in resistance; thus, both resistance and genetic background contribute to treatment differences. The cultigens were chosen to represent different levels of resistance so disease components can be directly compared. Any background genetic effects on cultigen resistance would by definition be part of the overall cultigen resistance. Differences in yield among cultigens are the result of both differences in resistance and background genetic potential.

The effects of cultigen resistance and fungicides seem to act additively. In general, a change to a more resistant cultigen or more effective fungicide treatment reduces apparent disease and increases yield. Cultigen treatments affected apparent disease to a far greater extent than fungicide treatments. Fungicides alone are not enough to achieve high yield in susceptible cultigens but are effective in combination with a moderately resistant cultigen. It is interesting that the overall effect of fungicides on apparent disease severity is low, whereas the effect on yield and marketable yield is large. More effective fungicide treatments resulted in slightly less apparent disease, higher yield, and a higher percent marketable yield.

In most cases, growers are already using cultivars with resistance comparable to M 21. The most effective fungicide treatment in our study was fluopicolide + chlorothalonil alternating with mancozeb + cyazofamid. For growers using cultivars with moderate resistance, higher yield would likely be achieved with this treatment compared with the previously recommended program (propamocarb + chlorothalonil alternating with famoxadone + cymoxanil + mancozeb).

It is clear that cultivars with resistance coupled with an effective fungicide program are required to achieve high yield at this time. To achieve high yield without fungicide, more resistance is needed. The highly resistant PI 197088 shows little disease but still appeared to benefit from fungicides, although it seems a low-level protectant fungicide (mancozeb) provides enough protection to achieve the highest yields. In other words, with the highest host resistance available to breeders, there was no yield benefit by moving from a low-level protectant fungicide program to a program of alternating systemic and protectant tank mixes. Nevertheless, even with recently identified high resistance incorporated into commercial cultivars, some fungicide applications are likely to be required to achieve the highest yield.

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