Research Note

The *I* Gene of Bean: A Dosage-Dependent Allele Conferring Extreme Resistance, Hypersensitive Resistance, or Spreading Vascular Necrosis in Response to the Potyvirus *Bean common mosaic virus*

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The resistance to the potyvirus *Bean common mosaic virus* (BCMV) conferred by the *I* allele in cultivars of *Phaseolus vulgaris* has been characterized as dominant, and it has been associated with both immunity and a systemic vascular necrosis in infected bean plants under field, as well as controlled, conditions. In our attempts to understand more fully the nature of the interaction between bean with the *I* resistance allele and the pathogen BCMV, we carefully varied both *I* allele dosage and temperature and observed the resulting, varying resistance responses. We report here that the *I* allele in the bean cultivars we studied is not dominant, but rather incompletely dominant, and that the system can be manipulated to show in plants a continuum of response to BCMV that ranges from immunity or extreme resistance, to hypersensitive resistance, to systemic phloem necrosis (and subsequent plant death). We propose that the particular phenotypic outcome in bean results from a quantitative interaction between viral pathogen and plant host that can be altered to favor one or the other by manipulating *I* allele dosage, temperature, viral pathogen, or plant cultivar.

Additional keywords: plant virus resistance, temperature-sensitive resistance.

The precise role of the hypersensitive response (HR) in resistance responses by plants to their pathogens has been a question of long-standing interest (Ponz and Bruening 1986; Culver et al. 1991; Matthews 1991; Goodman and Novacky 1994; Hammond-Kosack and Jones 1996; Dangl et al. 1996; Greenberg 1997; Yu et al. 1998). In typical hypersensitive resistance against plant viruses, necrotic local lesions appear on the inoculated leaf concomitant with the inhibition of the virus in the vicinity of the lesions; the virus is limited to the inoculated leaf and does not spread systemically. However,

what mediates that inhibition and exactly what role the HR plays are not yet fully understood. In plant–pathogen interactions that normally involve hypersensitive resistance, there are some clearly documented situations in which there is effective resistance without accompanying hypersensitive cell death (Takusari and Takahashi 1979; Yu et al. 1998). Conversely, there are examples of viruses or viral mutants that are not confined to the inoculated leaf even though the HR is induced (Matthews 1991; Culver et al. 1991). By constructing different mutants of *Cucumber mosaic virus* (CMV), Kim and Palukaitis (1997) have shown that the resistance to CMV in cowpea involves both a local HR and a viral inhibition response (IR) and that these two responses can be separated using different viral mutants. This separation of two different responses is supported by the phenotype of the *Arabidopsis* mutant *dnd1*, in which there is loss of the HR but not resistance to the bacterial pathogen *Pseudomonas syringae* (Yu et al. 1998).

It is not surprising then that attempts to label plant viral resistance genes as hypersensitive, or not so, have encountered difficulties. Clearly there are some forms of resistance to viral pathogens that do not involve the HR. The term immunity refers to an “absolute state of exemption from infection” with a virus (Cooper and Jones 1983); there is no detectable replication and the virus cannot be recovered from the inoculated leaf. A classic example of this type of resistance is that against *Cowpea mosaic virus* due to interference with processing of viral polyproteins by a protease inhibitor in the cowpea cv. Arlington (Ponz et al. 1988). While immunity and typical hypersensitive resistance are readily distinguishable, attempts to delineate other categories of resistance responses between these two extreme classes have been problematic.

In potato, resistance and resistance genes were classified into two main types: necrotic or hypersensitive resistance, conferred by *N* genes; and extreme resistance, showing very little or no visible effect, conferred by *R* genes (Delhey 1975). However, the difficulties in reliably differentiating these two

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classes are readily apparent from the very derivation of the term extreme resistance, used by H. Ross when localized necrotic lesions were observed on *Potato virus X* (PVX)–inoculated leaves of some plants previously called immune (Cockerham 1970). Even though potato breeders reported difficulties in scoring plants “accounted for by the difficulty in differentiating between [extremely] resistant individuals showing exceptional localised necrosis....and hypersensitive individuals showing little or no necrosis through physiological effects operating at the time of symptom production” (Cockerham 1970), the categories have persisted (Valkonen 1994) though they have not served the field particularly well. Thus, it was not surprising to find that a “*hypersensitive response-like mechanism*” [our italics] is involved in the *extreme resistance* in potato conferred by the *Ry_{sto}* gene against the potyviruses *Potato virus Y* (PVY) and *Tobacco etch virus* (Hinrichs et al. 1998). Even the very strong extreme resistance to PVX in potato conferred by the *Rx* loci is associated with hypersensitivity in some cultivars and under some environmental conditions (Bagnall 1961; Bendahmane et al. 1999; Benson and Hooker 1960; Delhey 1974;). One of the conditions affecting hypersensitivity in plants with the *Rx* gene is temperature (Benson and Hooker 1960; Delhey 1974). The same is true for hypersensitivity exhibited in several other systems, e.g., the temperature sensitivity of the resistance response in *N* gene tobacco inoculated with *Tobacco mosaic virus* (Ponz and Bruening 1986).

What emerges then, for many resistance genes, is a continuum of possible response in the reaction between virus and resistant plant that may include an effective hypersensitive resistance under some, but not all, conditions. Nowhere is this more clearly seen than in the interaction between the potyvirus *Bean common mosaic virus* (BCMV) and cultivars of *Phaseolus vulgaris* carrying the *I* allele (Ali 1950), which is widely used in bean breeding as a source of resistance to BCMV. Plant breeders who first characterized this resistance reported apparent immunity in cv. Corbett Refugee to the virus (Pierce 1934) and agreed that the resistance was conferred by a single, dominant gene (Pierce 1935; Ali 1950). However, reports soon followed that an occasional plant developed systemic necrosis and died (Grogan and Walker 1948), and eventually it became clear (Ali 1950) that BCMV is also the causal agent for the “black root syndrome” previously described by Jenkins (1940, 1941). “Black root” symptoms include spreading, usually lethal, phloem necrosis seen under certain conditions in plants carrying the ‘Corbett Refuge’ type

of resistance but never in susceptible plants, which exhibit mosaic symptoms, or in those with a different type of resistance (Grogan and Walker 1948). In our attempts to more fully understand the nature of the interaction between the *I* resistance locus and the pathogen BCMV, we carefully varied both *I* allele dosage and temperature and observed the resulting, varying resistance responses. We report here that the *I* allele in the bean cultivars we studied is not dominant, but rather incompletely dominant, and that the system can be manipulated to show a clear continuum of response in these plants to BCMV that ranges from immunity or extreme resistance, to hypersensitive resistance, to phloem necrosis that spreads systemically and results in plant death.

These three types of response can be elicited reliably by mechanically inoculating three different *P. vulgaris* ‘Black Turtle’ genotypes (Fisher and Kyle 1994) with BCMV NY15 (isolate NY 68-95) at two different temperatures. When ‘Black Turtle’ plants homozygous for *I*, heterozygous (*I/i*), or fully susceptible (*i/i*) are inoculated with BCMV, a continuum of qualitatively different classes of plant response to the virus are observed (Table 1). Inoculating homozygous resistant plants (BT-1, *I/I*) with BCMV at 23°C elicits the extreme resistance against BCMV originally described for the *I* locus; neither inoculated primary leaves nor uninoculated upper trifoliates show detectable symptoms, and no coat protein can be detected in those leaves using direct double-antibody sandwich–enzyme-linked immunosorbent assay (Clark and Adams 1977). In this case, there is an effective viral IR but no visible HR. However, by changing either the plant gene dosage (to *I/i*, at 23°C) or the temperature (to 34°C, with *I/I* plants), a typical HR can be elicited in at least some of the plants. In all of the *I/i* plants held at 23°C, there are pinpoint necrotic lesions (pnl) on the inoculated leaf (although still no detectable viral coat protein), but there are neither symptoms nor detectable coat protein in upper, uninoculated leaves. In these cases, the virus is confined to the inoculated leaf, where there is an effective IR and an HR. Finally, by altering conditions still more (e.g., *I/i* plants at 34°C), the third response type can be elicited. The IR in the inoculated leaf is lost or significantly reduced, resulting in detectable viral coat protein as well as local lesions. In addition, virus spreads to, and replicates in, upper leaves, causing a systemic vascular necrosis (vn) in the phloem tissue (an HR) that eventually kills the plant. Note that at the intermediate conditions between those eliciting the second and third response types (i.e., plants with double *I* gene dose [*I/I*] at high temperature [34°C]), half of the plants show each of the

Table 1. Symptoms and accumulation of *Bean common mosaic virus* (BCMV) NY-15 coat protein in inoculated primary leaves and uninoculated trifoliates of *Phaseolus vulgaris* BT-1, BT-2, and (BT-2 × BT-1) F₁ plants^a

Temperature	BT-1 (genotype <i>I/I</i>)			(BT-1 × BT-2) F ₁ (genotype <i>I/i</i>)			BT-2 (genotype <i>i/i</i>)		
	Sym ^b	PL ^c	UT ^d	Sym	PL	UT	Sym	PL	UT
23°C	--/--	0.045 ± 0.007 ^e	0.022 ± 0.015 (4) ^e	pnl/--	0.029 ± 0.004	0.066 ± 0.017 (4)	cl/m	1.543 ± 0.11*	1.823 ± 0.177* (2)
34°C	pnl/--	0.049 ± 0.007	0.061 ± 0.014 (6)	pnl/vn	0.190 ± 0.096*	0.557 ± 0.125* (11)	cl/m	1.082 ± 0.018*	1.511 ± 0.051* (2)
	pnl/vn	0.050 ± 0.009	0.305 ± 0.168* (5)						

^a All plants sampled 6 days postinoculation.

^b Sym = Symptoms, local/systemic: -- = no symptoms, cl = chlorotic spots, m = mosaic, pnl = pinpoint necrotic lesions, and vn = systemic vascular necrosis.

^c PL = Primary inoculated leaves.

^d UT = Uninoculated top trifoliolate.

^e Values represent the mean absorbance (405 nm) ± standard deviation determined using enzyme-linked immunosorbent assay. Numbers in parentheses indicate the number of plants sampled. The asterisk indicates that the mean absorbance value is positive for the presence of BCMV antigen, defined as any value above the mean absorbance value for mock-inoculated control plants plus three standard deviations.

two response types (6 pnl/-- and 5 pnl/vn). Finally, it should be pointed out that varying the particular potyvirus (Fisher and Kyle 1994) or the bean cultivar (Fisher 1995; M. Jahn, *unpublished data*) of the virus–host interaction can also result in a shift in the type of resistance response observed.

In addition to showing a continuum of responses in the resistant (*I/I* and *I/i*) plants under different conditions, the data in Table 1 make a preliminary case for the characterization of the resistance conferred by the *I* allele as incomplete rather than complete dominance. At both temperatures tested, there are differences in the phenotypes of the *I/I* versus the *I/i* plants. At 23°C, the inoculated primary leaves of the *I/i* plants have small, necrotic lesions (pnl), while those of *I/I* plants are always symptomless. At 34°C, in those plants in which lesions are seen on inoculated leaves and virus spreads to upper, uninoculated leaves (the pnl/vn plants), it is only in *I/i* plants, and not in *I/I* plants, that viral coat protein is detectable in the inoculated leaves. This is consistent with the observation that the virus does spread systemically in 100% of the *I/i* plants but in only 45% (5/11) of the *I/I* plants at that higher temperature. Thus, at both temperatures, the resistance against the virus conferred by the *I* allele is less effective in the heterozygous than in the homozygous plants. Because the number of plants in this experiment was small, we did additional experiments to demonstrate the clear difference in response of *I/I* and *I/i* plants. The experiments detailed in Tables 2 and 3 show a definitive difference in the number of necrotic lesions visible on inoculated primary leaves of *I/i* versus *I/I* plants. In the experiment shown in Table 2, a 20 to 1 difference in the average number of lesions per leaf was apparent by 3 to 4 days post-inoculation on *I/i* versus *I/I* plants held at 34°C (20 and 21 plants tested, respectively). Equally striking is the difference in the number of plants of each genotype in which the virus spread to upper, uninoculated leaves and caused a systemic, lethal necrosis of the phloem (18/20 in the *I/i* plants versus 7/21 of the *I/I* plants by the fifth day, the end of the

observation period). A second experiment (Table 3) comparing viral-induced symptoms on inoculated plants of the two genotypes incubated at a slightly lower temperature (31°C) confirmed the clear difference in number of lesions on the inoculated leaves. In addition, the size of the lesions on the inoculated leaves was significantly greater on the heterozygous (*I/i*) versus the homozygous (*I/I*) plants by 4 days postinoculation. Clearly, *I* allele dosage affects the resistance response at three different temperatures (Tables 1, 2, and 3), leading to the conclusion that the *I* allele mediates an incomplete dominance against BCMV in this cultivar, as well as in the cv. Corbett Refugee (Fisher 1995) of *P. vulgaris*.

Because these data seemed to indicate that increasing the temperature tipped the balance in the virus–plant interaction in favor of the virus, we looked at the effect of temperature on viral coat protein accumulation over time in both inoculated and upper, uninoculated leaves of the susceptible, near isoline BT-2 (*i/i*). The data presented in Figure 1 clearly show that increasing the temperature from 24 to 34°C results in more rapid accumulation and higher levels of viral coat protein in both inoculated and systemically infected leaves, presumably indicating that the virus replicates, and possibly moves, faster at higher temperature in the susceptible BT-2 genotype. Assuming that a similar effect occurs in inoculated plants that are homozygous (BT-1) or heterozygous (*F*₁ plants) for the *I* allele, what emerges from the combined data of Tables 1 to 3 and Figure 1 is the notion of a quantitative battle between virus and plant, with the combatant that musters the largest force first—the number of viral molecules or the number of plant molecules—gaining the upper hand. Clearly doubling the *I* allele dosage in the resistant plant offers the possibility of more defensive molecules, and data from Figure 1 suggest that raising the temperature allows the virus to replicate and produce more viral molecules faster, possibly disproportionately faster, than the plant's ability to produce more plant molecules.

Table 2. Comparison of symptoms in *I/I* and *I/i* genotypes inoculated with *Bean common mosaic virus* (BCMV) and held at 34°C

Plant genotype	Days postinoculation									
	1		2		3		4		5	
	PL ^a	SN ^b	PL	SN	PL	SN	PL	SN	PL	SN
BT-1 (<i>I/I</i>)	0.0	0/21	0.1 ± 0.3	0/21	0.4 ± 0.8	1/21	1.2 ± 1.6	3/21	1.6 ± 1.8	7/21
(BT-2 × BT-1) <i>F</i> ₁ (<i>I/i</i>)	0.0	0/20	11.7 ± 7.7	0/20	20.2 ± 11.7	6/20	21.1 ± 12.9	13/20	21.5 ± 12.5	18/20

^a PL = Inoculated primary leaves, average number of necrotic lesions per leaf on inoculated primary leaves ± standard deviation. Number of primary leaves is twice the number of plants.

^b SN = Systemic necrotic symptoms. Number of plants with systemic symptoms/total number of plants.

Table 3. Comparison of symptoms in *I/I* and *I/i* genotypes inoculated with *Bean common mosaic virus* (BCMV) and held at 31°C

Plant genotype	Days postinoculation									
	1		2		3		4		5	
	PLP ^a	Size ^b	PLP	Size	PLP	Size	PLP	Size	PLP	Size
BT-1 (<i>I/I</i>)	0	--	0 ^c	0.05	0 ^c	0.05	0 ^c	0.05 ^d	0 ^c	0.05 ^d
(BT-2 × BT-1) <i>F</i> ₁ (<i>I/i</i>)	0	--	6 ^c	0.05	9.5 ^c	0.05	9 ^c	0.10 ^d	10 ^c	0.20 ^d

^a PLP = Inoculated primary leaves, median number of necrotic lesions per plant on inoculated primary leaves (two primary leaves per plant). Twenty-four plants were observed for each genotype.

^b Size = Median diameter of necrotic lesions in millimeters. Pinpoint necrotic lesions were assigned a diameter of 0.5 mm.

^c Indicates a significant difference at the 0.1% level of probability between median lesion number on *I/I* versus *I/i* plants on a particular day using the Wilcoxon Rank Sum Test (used because the data were not normally distributed).

^d Indicates a significant difference at the 0.1% level of probability between median lesion size on *I/I* versus *I/i* plants on a particular day using the Wilcoxon Rank Sum Test.

The observations reported here contribute to a growing appreciation of the complexity of the interaction between a potentially resistant plant and a potential viral pathogen. While a predictable plant–pathogen response, whether that be extreme resistance or hypersensitive resistance, can be elicited when plant cultivar, resistance allele dosage, viral strain, and environmental conditions (including temperature, light intensity, and humidity) are carefully controlled, the variability possible when they are not can extend from one end of the resistance continuum (extreme resistance) to the other (systemic vascular necrosis and plant death). Our model for the interaction between BCMV and bean plants carrying the *I* allele is a battle between plant and pathogen that is one of quantity and relative timing, in which the outcome depends on the relative accumulation across time of inhibitory molecules produced by the plant versus viral molecules produced by the pathogen. Our model is compatible with that proposed by Bendahmane et al. (1999) to explain the separate virus resistance and cell death responses mediated by the *Rx* gene of potato in interaction with PVX. In the interaction between BCMV and the *I* allele, rapid activation of resistance in an *I/I* plant at 23°C favors the plant and leads to extreme resistance; slower activation in an *I/i* plant at higher temperature favors the pathogen, allowing the virus to escape the IR and to move just ahead of a spreading hypersensitivity (HR), resulting in systemic vascular necrosis and eventual plant death.

The fact that the balance between plant and pathogen can so easily shift suggests caution in classifying types of resistance (e.g., extreme resistance versus hypersensitive resistance) as well as in predicting the cellular responses supposedly associated with those labels. For instance, it was once widely accepted that resistance is expressed in protoplasts derived from plants with extreme resistance, but that it is not expressed in those derived from plants showing hypersensitive resistance

(Otsuki et al. 1972; Barker and Harrison 1984; Adams et al. 1985, 1986). From the emerging understanding that these are not two distinct and immutable resistance types, but instead represent two different points along a continuum of possible responses, it follows that cellular responses are unlikely to be so black and white. For instance, while resistance to PVX is expressed in protoplasts carrying the extreme resistance gene *Rx*, that resistance is not expressed until after at least an 8-h lag (Köhm et al. 1993). Second, the difficulty in labeling the response to PVY of potato protoplasts carrying the “extreme resistance” gene *Ry_{sto}* is apparent in the different conclusions of two groups, with Barker and Harrison (1984) concluding that resistance is, and Hinrichs et al. (1998) concluding that resistance is not, expressed there. (While in both cases it is apparent that viral replication occurred in protoplasts of resistant plants, it was much more limited in comparison to the level of replication in a susceptible cultivar.) Finally, how, as well as when, one examines the reaction in question is obviously critical, as pointed out by Kim and Palukaitis (1997) in discussion of apparently discrepant data showing a clear IR against CMV after a 15-h lag in protoplasts isolated from hypersensitive resistant cowpeas. Whereas Gonda and Symons (1979), also studying CMV in protoplasts from hypersensitive resistant cowpeas, had earlier observed no inhibition of viral coat protein accumulation for 50 h postinoculation, those investigators had observed a severe drop in the rate of coat protein synthesis after 15 h, possibly the same IR as observed by Kim and Palukaitis (1997).

What is now apparent is that many factors, including resistance gene dosage, temperature (and other environmental conditions), virus isolate, plant cultivar, and simultaneous infection with other viruses (Jayasinghe et al. 1989), can influence both the ultimate outcome of a particular virus–host interaction as well as the host phenotypes observed as that interac-

Fig. 1. Accumulation postinoculation (PI) of *Bean common mosaic virus* (BCMV) NY-15 coat protein in **A**, inoculated primary leaves and **B**, uppermost top trifoliate of *Phaseolus vulgaris* BT-2 plants at two different temperatures (lo = 24°C; hi = 34°C). Coat protein accumulation was measured using direct double-antibody sandwich–enzyme-linked immunosorbent assay. Values represent the mean absorbance (at 405 nm) $\pm$ standard deviation of 10 BCMV NY15-inoculated plants (virus) and five buffer-inoculated plants (mock) per day per temperature.

tion unfolds across time. Such complexity dictates care in how, as well as when, one analyzes the interaction.

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